

Ecological consequences of heritable intraspecific variation

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Abstract

Trait variation among individuals of a population is now considered to be an important part of biodiversity. Many empirical studies have quantified this variation and showed that it can change over time. These studies have also made it clear that intraspecific variation is important in determining a population's response to disturbances. Heritable changes in traits that determine how species interact with its biotic and abiotic environment lead to eco-evolutionary feedbacks. Mathematical models that integrate some aspects of evolutionary models with those of ecological models are required to study these feedbacks. In this dissertation, I build and extend a series of population dynamics models focusing on heritable intraspecific variation in continuously varying traits. My models capture trait-based ecological interactions as well as stabilizing natural selection. With this mathematical approach I study (i) two-species competition, exploiter-victim interaction and mutualism, and (ii) biotic-abiotic interactions with a conditionable or a consumable abiotic factor. I show that (i) weak stabilizing selection promotes stable coexistence in two-species interactions and (ii) population dynamics and trait evolution are significantly different when a species interacts with a conditionable abiotic factor and a consumable abiotic factor. In a special case of the biotic-abiotic interaction, I show that smaller body size is intrinsically beneficial in a competition for space. Overall, my results point that heritable intraspecific variation has important ecological consequences and could significantly change our expectations relative to those based on purely ecological models.

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1. *sm_mathematica.nb*

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Chapter 1

Introduction

Biodiversity encompasses variation in many dimensions which span over spatial, temporal and organizational scales. A recent theme has emerged in ecology where the importance of variation between individuals of a population (intraspecific variation) is emphasized (Albert *et al.*, 2011, Bolnick *et al.*, 2011, Violle *et al.*, 2012, Des Roches *et al.*, 2018, 2021). This is in contrast to the traditional emphasis on variation between species (interspecific variation). We now know that intraspecific phenotypic variation plays a key role in the ecology of a species and in determining the function of the species in its ecosystem (Breza *et al.*, 2012, Wright *et al.*, 2016). Intraspecific variation also affects population dynamics, strength of ecological interactions within and between species, community composition, and other ecological processes (Frankham, 1996, Lloyd-Smith *et al.*, 2005, Vellend, 2006, Des Roches *et al.*, 2018, Allen *et al.*, 2018, Hausch *et al.*, 2018, Austin and Dunlap, 2019, Start, 2019, Start and Gilbert, 2019). In my dissertation, I develop a general and powerful mathematical framework to study the ecological consequences of heritable intraspecific variation and apply it to a variety of specific biotic-biotic and biotic-abiotic interactions.

Theoretically, Bolnick *et al.* (2011) identified six mechanisms by which intraspecific phenotypic variation could affect ecological dynamics. I focus on two mechanisms among them: nonlinear dependence of ecological interactions on trait values and rapid evolution due to heritable trait variation. First, if ecological processes depend nonlinearly on trait values, then the mean trait value cannot solely determine ecological dynamics. Variance (Albert *et al.*, 2011, Violle *et al.*, 2012) and higher moments can also play a role in determining

ecological dynamics. More broadly, the entire trait distribution (*i.e.* the abundance of different traits) can be important in determining the strength of ecological interactions. Second, if traits are heritable then the ecological dynamics (specifically, the dynamics of population densities) can affect trait evolution on short timescales. Since ecological interactions depend on these traits, the trait evolution feeds back to affect ecological dynamics forming an eco-evolutionary feedback (Lion, 2018, Patel *et al.*, 2018, Govaert *et al.*, 2018, Ware *et al.*, 2019).

Theoretical work demonstrates how this eco-evolutionary feedback affects intraspecific variation in different ecological interactions (Kopp and Gavrillets, 2006, Yoder and Nuismer, 2010). These processes are closely related to those responsible for the maintenance of genetic variation in the face of stabilizing selection (Bulmer, 1985). Frequency dependent selection due to ecological interactions is one of such mechanisms (Clarke, 1979, Gavrillets, 2004). Biotic and abiotic changes in the environment lead to changes in genetic variation which have been observed in natural populations (Buckling and Rainey, 2002, Summers *et al.*, 2003, Nijhawan *et al.*, 2019). Although trait variation typically changes on evolutionary timescales, there are cases where traits could change on a faster timescale. Particularly, man-made events have led to drastic changes in genetic variation (Smith *et al.*, 1991, Keller and Largiadèr, 2003, Mitrovski *et al.*, 2008, Jacquemyn *et al.*, 2009). Understanding the consequences of such changes requires theoretical models of evolving genetic variation which could provide insights into the role of intraspecific variation in eco-evolutionary dynamics.

Another well-studied example of temporal changes in intraspecific variation is the phenomenon of character displacement. Character displacement is the divergence of mean phenotypes of two species in sympatry (Brown and Wilson, 1956, Schuller and McPhail, 1992). When two closely related species with similar phenotypes come into contact with each other, subsequent eco-evolutionary dynamics leads to character displacement. Early empirical and theoretical works did not find much evidence for the role of selection due to ecological interactions being the cause of phenotypic differences among species in sympatry (Slatkin, 1980, Simberloff and Boecklen, 1981). Despite this initial skepticism, there is now a substantial empirical evidence for ecological character displacement from several taxa (Brown and Wilson, 1956, Hutchinson, 1959, Schuller and McPhail, 1992, Losos, 2000,

Dayan and Simberloff, 2005). Similarly, mathematical models suggest ecological character displacement might be more common (Taper and Case, 1992, Doebeli, 1996a, Aguilée *et al.*, 2011). Taper and Case (1992) have demonstrated that models of character displacement are sensitive to assumptions on intraspecific variation. None of the existing theoretical approaches explore multimodality in phenotypic distribution as an alternative to the classical character displacement of mean shifts in a unimodal distribution. However, evolutionary theory offers several promising approaches to build a mathematical framework for studying eco-evolutionary dynamics where intraspecific variation is evolving.

First, population genetics techniques allow us to model quantitative traits controlled by few major loci with two or more alleles. Population genetics models have been used to study the dynamics of genetic variation under stabilizing selection (Gavrilets and Hastings, 1994a, 1995). An immediate problem with major loci models is that the large number of loci or alleles required to approximate continuous trait distributions increases the dimensionality of the corresponding mathematical model usually making it analytically intractable and computationally costly. Despite these challenges, some progress is possible. For example, using a four-locus model, Kopp and Gavrilets (2006) studied by simulations the relationships between the strength of selection, strength of interaction, and the amount of genetic variation maintained. Second, quantitative genetics offers a way to deal with continuous traits which arise from a large number of loci (Lande, 1976, Bulmer, 1985, Iwasa *et al.*, 1991). This tool works best when the traits are normally distributed, and the variance is maintained by some exogenous processes. However using models of two-species competition and predator-prey interactions, Doebeli (1996b) demonstrated that the normality assumption restricted the range of possible dynamic outcomes. Although there are techniques which allow us to study the effects of deviations from these restrictive assumptions, both population genetics and quantitative genetics models largely ignore density-dependent effects arising from population dynamics which must be considered to understand the ecological consequences of intraspecific variation.

Third, adaptive dynamics offers another alternative to study eco-evolutionary dynamics. Analyses based on the adaptive dynamics framework assume that populations are composed of discrete morphs and that mutations produce the necessary genetic variation for evolution

(Geritz *et al.*, 1998, Dieckmann, 2004). Unlike the population genetics and quantitative genetics models, adaptive dynamics comes with strong analytical advantage even while directly accounting for ecological interactions. This advantage can mainly be attributed to an assumption of timescale separation in which the ecological dynamics is assumed to be much faster than the evolutionary dynamics (Sasaki and Dieckmann, 2011, Lion, 2018). Adaptive dynamics has been highly successful in the study of several eco-evolutionary phenomena (Dieckmann and Doebeli, 1999, Ferrière *et al.*, 2002, Kylafis and Loreau, 2008, Troost *et al.*, 2009, Zu and Wang, 2013, Kremer and Klausmeier, 2017, Vitale and Best, 2019, ten Brink *et al.*, 2019). However, due to the reliance on timescale separation this approach cannot be used to simultaneously track population dynamics and trait evolution. Moreover, the dynamics of continuously varying traits can only be approximated using an oligomorphic approach (Sasaki and Dieckmann, 2011) which has numerical and analytical challenges similar to those of the multilocus population genetics approach.

A final approach is to use the ecological model which underlies the adaptive dynamics method. In adaptive dynamics, ecological models are used to calculate the invasion fitness. But these ecological models can also be used to study eco-evolutionary dynamics without approximations inherent in the adaptive dynamics (Roughgarden, 1972, Slatkin, 1980, Taper and Case, 1992). However, existing studies using this approach assume that the traits are distributed according to a normal distribution. This is a major limitation because of the prevalence of both multimodal and skewed trait distributions. An important endeavor in studies of adaptive dynamics is to identify conditions for evolutionary branching. These are evolutionary dynamics where a population undergoes phenotypic diversification and becomes bimorphic. In this dissertation I build ecological models to study the consequences of eco-evolutionary feedback and analyze them without the restriction of the Gaussian approximation.

Specifically, I investigate the role of heritable intraspecific variation in two different eco-evolutionary feedback: (i) biotic-biotic and (ii) biotic-abiotic interactions. In chapter 2 I study three different biotic-biotic interactions: (i) competition, (ii) exploiter-victim and (iii) mutualism and derive conditions for stable coexistence. In chapter 3 I classify eco-evolutionary feedback in biotic-abiotic interactions. The classification is based on whether

the abiotic environment is conditionable like the soil pH or consumable like inorganic nitrogen. In chapter 4 I study a specific case of biotic-abiotic interaction where the consumable abiotic factor is space, and the biotic trait is body size. This is unlike the other two chapters where I consider a general continuously varying quantitative trait which determines the strength of ecological interactions. In all these chapters I start with an ordinary differential equation model of population dynamics and extend it to an integro-differential equation model of eco-evolutionary dynamics. These models are then analyzed to contrast the ecological dynamics in the presence and absence of heritable intraspecific variation.

Chapter 2: Coevolutionary systems

Studies of biotic-biotic interactions are a staple of both ecology and evolution. In ecology, many classic studies on competition and predator-prey interactions are between two interacting species (Lotka, 1920, Gause and Witt, 1935, Kostitzin, 1939). In evolution, coevolution is of particular interest because of its ability to explain many evolutionary patterns (Thompson, 1994, Nuismer, 2017). However theoretical studies of coevolution have not offered any insights into the quintessential ecological problem of species coexistence. To address this issue, I extend standard population dynamics models of two-species competition, predator-prey interaction and mutualism to include heritable intraspecific variation. In these eco-evolutionary models, I model the interaction based on trait-matching of a single continuously varying trait in the two species. Trait-matching is the assumption that the interaction is strongest when the two trait values match and it gradual weakens as the difference in the trait values increases. I also assume that these traits are under interaction-independent stabilizing selection. Overall, I investigate the effect of the range of interaction, strength of stabilizing selection and optimal trait value on stable species coexistence.

Chapter 3: Biotic-abiotic interactions

Evolutionary and eco-evolutionary feedbacks involving biotic-abiotic interactions have received relatively less attention compared to biotic-biotic interactions. In recent times, the field of niche construction (Odling-Smee *et al.*, 2013) has focused on the evolutionary feedback involving the abiotic environment. Specifically, it introduces the concepts of environmental inheritance and evolutionary niche which provides a novel perspective into the dynamic role of the abiotic environment. We also need to study such biotic-abiotic interactions to build a complete understanding of the effects of eco-evolutionary feedback in the field (Ware *et al.*, 2019). Eco-evolutionary models with heritable intraspecific variation provides a natural framework to study biotic-abiotic interactions. There is a crucial difference between the types of abiotic factors which organisms interact with in nature. Specifically, I draw attention to consumable and conditionable abiotic factors which underlie exploitative and interference competition, respectively.

To study the differences in the eco-evolutionary dynamics in a biotic-abiotic interaction between a consumable and a conditionable factor, I first build population dynamics model with two species/clonal traits and a single abiotic state. I then extend it to a continuously varying trait which is under stabilizing natural selection. Overall, I use the qualitative differences in equilibrium states, and the effect of the feedback strength and strength of selection on population size and trait distribution to demonstrate the need to distinguish between the two abiotic factors.

Chapter 4: Body size and competition for space

Body size affects several facets of ecology (Peters, 1983, Schmidt-Nielsen, 1984). This, combined with the fact that body size is a universal phenotype across the tree of life has led to a vast number of studies on body sizes. An interesting dimension of this research concerns with the costs and benefits of having a large versus a small body size (Blanckenhorn, 2000). I focus on this particular dimension with regards to competition for space. First, I build a simple deterministic population dynamics model to have an analytical understanding of

space competition among sessile organisms. Second, I build a spatially explicit stochastic model to investigate the consequences of the simplifying assumptions in the deterministic model. I also use this model to study the effects of dispersal. Finally, I extend the population dynamics model to continuously varying body size in a similar fashion to the other chapters. With this model, I study the effects of stabilizing natural selection and competition for space on body size distributions.

Chapter 2

Coevolutionary systems

Publication statement

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Abstract

The patterns and outcomes of coevolution are expected to depend on intraspecific trait variation. Various evolutionary factors can change this variation in time. As a result, modeling coevolutionary processes solely in terms of mean trait values may not be sufficient; one may need to study the dynamics of the whole trait distribution. Here, we develop a theoretical framework for studying the effects of evolving intraspecific variation in two-species coevolutionary systems. In particular, we build and study mathematical models of competition, exploiter-victim interactions, and mutualism in which the strength of within- and between-species interactions depends on the difference in continuously varying traits between individuals reproducing asexually. We use analytical approximations based on the invasion analysis and supplement them with numerical results. We find that intraspecific variation can be maintained if stabilizing selection is weak in at least one species. When intraspecific variation is maintained under competition or mutualism, coexistence in a stable equilibrium is promoted when between-species interactions mostly happen between individuals similar in trait values. In contrast, in exploiter-victim systems coexistence typically requires strong interactions between dissimilar exploiters and victims. We show that trait distributions can become multimodal. Our approach and results contribute to the understanding of the ecological consequences of intraspecific variation in coevolutionary systems by exploring its effects on population densities and trait distributions.

2.1 Introduction

Coevolution, that is, reciprocal evolutionary changes in ecologically interacting species, is a major research area where ecology and evolution come together (Futuyma and Slatkin, 1983, Thompson, 1994, Nuismer, 2017). Although coevolution is difficult to demonstrate in natural systems (Janzen, 1980, Nuismer *et al.*, 2010), its importance in nature is well established (Davies and Brooke, 1988, Clayton *et al.*, 1999, Soler *et al.*, 2001, Brouat *et al.*, 2001, Toju and Sota, 2006). Coevolution explains a number of evolutionary phenomena including character displacement in competing species, evolutionary arms race between exploiters and victims,

and trait correlation between mutualist partners (Pfennig and Pfennig, 2009, Benkman *et al.*, 2003, Anderson and Johnson, 2008). The geographic mosaic theory of coevolution (GMTC) further highlights the relevance of coevolution by incorporating spatial variation in natural selection and strength of ecological interactions (Thompson, 2005). Through GMTC, coevolution can explain patterns of clinal variation, local adaptation, and stability of mutualisms in the presence of costs (Nuismer *et al.*, 2000, Gomulkiewicz *et al.*, 2000, Gavrillets and Michalakis, 2008, Gómez *et al.*, 2009).

Coevolution is not only a very important process but is also complex due to the intricate relationship between evolutionary and ecological processes. Because of its complexity, there is a strong need for a mathematical theory capturing this relationship. Correspondingly, various population genetics, quantitative genetics, and adaptive dynamics models and methods have been used to study eco-evolutionary dynamics in specific two-species systems: competition (Roughgarden, 1976, Slatkin, 1980, Taper and Case, 1992, Leimar *et al.*, 2013, Kremer and Klausmeier, 2017), mutualism (Ferrière *et al.*, 2002, 2007, Foster and Kokko, 2006, Akçay, 2016), and exploiter-victim interactions (Gavrillets, 1997, Doebeli, 1997, Abrams, 2000, Nuismer *et al.*, 2005, Gavrillets and Michalakis, 2008, Schreiber *et al.*, 2018, Fleischer *et al.*, 2018). Doebeli and Dieckmann (2000), Kopp and Gavrillets (2006), Yoder and Nuismer (2010) contrasted all three types of two-species interactions in their models. Specifically, using a weak selection approximation and numerical simulations, Kopp and Gavrillets (2006) studied the dynamics of allele frequencies, means, and variances of a trait controlled by several diploid diallelic loci. Yoder and Nuismer (2010) modeled how trait variation in a metapopulation changes due to coevolution using a quantitative genetics approach and an individual-based model. Doebeli and Dieckmann (2000) used the adaptive dynamics approach to study phenotypic diversification due to ecological interactions. They also verified the analytic predictions using individual-based models. These and other similar mathematical models explicated the effects of the eco-evolutionary feedback in coevolution.

Both biological intuition and mathematical models tell us that the time-scales, patterns, and outcomes of coevolution should depend on within-species genetic and phenotypic variation (Bolnick *et al.*, 2011, Violle *et al.*, 2012, Albert *et al.*, 2011). Genetic and phenotypic variation affects not only evolutionary but also ecological forces and factors

including population dynamics, interaction strengths, and community composition (Des Roches *et al.*, 2018, Hausch *et al.*, 2018, Austin and Dunlap, 2019, Start, 2019, Start and Gilbert, 2019, Allen *et al.*, 2018, Lloyd-Smith *et al.*, 2005, Vellend, 2006, Frankham, 1996). These forces can change in time as the level of genetic variation in natural populations is not constant but rather can change on a much faster ecological time scale (Summers *et al.*, 2003, Buckling and Rainey, 2002, Nijhawan *et al.*, 2019). In particular, man-made events have lead to drastic changes in genetic variation (Keller and Largiadèr, 2003, Mitrovski *et al.*, 2008, Jacquemyn *et al.*, 2009, Smith *et al.*, 1991). Intraspecific variation is also expected to change as a result of coevolutionary processes. Character displacement, the divergence of mean phenotypes of two species in sympatry (Brown and Wilson, 1956, Schuller and McPhail, 1992, Dayan and Simberloff, 2005), is a well-studied example of temporal changes in phenotypes which decreases interspecific competition between the two species. Alternately, interspecific competition can be decreased by phenotypic diversification of one of the species (Abrams and Matsuda, 1994, Dieckmann and Doebeli, 1999, Winkelmann *et al.*, 2014). Overall, the importance of intraspecific variation in eco-evolutionary dynamics is well established. Thus, the description of the coevolutionary processes solely in terms of mean trait values may not be sufficient - one also needs to consider variances and higher-order moments of trait distribution.

Evolutionary theory has long been concerned with the problem of the maintenance of genetic variation (Lande, 1975, Barton, 1986, Gavrilets and Hastings, 1994b, Clarke, 1979, Gavrilets, 2004, Walsh and Lynch, 2018). There is now a rich variety of models explaining the equilibrium maintenance of genetic variation by mutation-selection balance, frequency-dependent selection, spatial heterogeneity, *etc.* However significantly less efforts have focused on the dynamics of variances. In an early study of the dynamics of genetic variation, Bulmer (1971) used the infinitesimal model which assumes that a quantitative trait is controlled by infinitely many loci with infinitely small effects. In his model, selection builds linkage disequilibrium which changes genetic variation. Using a population genetics model with major loci, Gavrilets and Hastings (1994a, 1995) studied the dynamics of genetic variation under stabilizing selection. Finally, the adaptive dynamics approach has been used to study emergence of genetic polymorphism as a result of evolutionary branching (Geritz *et al.*, 1998,

Diekmann, 2004, Dieckmann and Doebeli, 1999, Zu and Wang, 2013). All these methods have to deal with a trade-off between mathematical complexity and biological realism; each of these methods has its own advantages but also shortcomings. For example, Bulmer (1971) and Gavrillets and Hastings (1994a, 1995) ignored population densities, and the assumptions underlying the adaptive dynamics methods prevent one from exploring the changes in genetic variation in detail (Geritz *et al.*, 1998, Waxman and Gavrillets, 2005).

Here we develop an alternative framework for studying the effects of phenotypic variation in coevolution. Our framework is based on earlier single-species theoretical studies capturing both population densities and trait distributions. In our models, trait-based interspecific and intraspecific interactions will be the strongest when traits match in value. We will assume stabilizing selection independent of species interactions. For simplicity, we will assume that individuals reproduce asexually with no mutation. This allows us to focus on the effects of stabilizing selection and species interactions, and equate genetic variation with phenotypic variation. In contrast to existing adaptive dynamics and quantitative genetics models of coevolution which focus exclusively on mean trait values, we will also allow for the shape and variance of the trait distribution to change through time.

Below we start by introducing our approach using a single-species logistic model as an example. For this model, we characterize the effects of intraspecific competition and stabilizing selection on the equilibrium trait distribution and population density. Then we use a similar structure for three models describing two-species coevolutionary interactions: (i) competition, (ii) exploiter-victim, and (iii) mutualism. For the cases of competition and exploiter-victim interactions, we study the conditions for coexistence, equilibrium trait distributions, and the relationship between the strength of interaction and phenotypic variance. In the case of mutualism, we study the conditions for coexistence in a stable equilibrium. Our framework relates stabilizing natural selection, trait-based interactions between coevolving species, phenotypic distributions, and population densities to understand the ecological consequences of intraspecific variation in two-species coevolutionary systems.

2.2 Modeling framework: Single-species model

To introduce our approach, we start with the standard logistic model for the dynamics of the population density $N(t)$ in time:

$$\frac{dN(t)}{dt} = rN(t) \left(1 - \frac{N(t)}{K}\right), \quad (2.1)$$

where r is the population growth rate at low densities and K is the carrying capacity. Here, the population density always approaches K asymptotically. This model implicitly assumes that all individuals are identical (Kot, 2001).

Roughgarden (1972) and Doebeli and Ispolatov (2010) extended this model for the case of heritable intraspecific trait variation. Following their method, we assume that individuals differ with respect to a single continuous trait x . Then the population density $\phi(x, t)$ of trait x at time t changes according to equation

$$\frac{\partial \phi(x, t)}{\partial t} = r\phi(x, t) \left(1 - \frac{\int C(x, y)\phi(y, t)dy}{K(x)}\right). \quad (2.2)$$

Here r is the growth rate at low densities (assumed to be independent of x), competition kernel $C(x, y)$ measures the effect of competition with individuals with trait y , and function $K(x)$ is the “carrying capacity” for individuals with trait value x . The population size (total population density) is given by the integral $N(t) = \int \phi(y, t)dy$. Equation (2.2) implies that individuals reproduce asexually without mutation.

It is standard and mathematically convenient to use Gaussian functions $C(x, y) = \exp[-(x - y)^2/2\sigma_c^2]$ and $K(x) = K_0 \exp(-(x - \theta)^2/2\sigma_s^2)$. The former function implies that competition decreases with increasing the difference in trait values. The latter function assumes that carrying capacity decreases with deviation from the optimum trait value θ . This represents stabilizing selection independent of competitive interactions. Alternately, in coevolutionary models of Gavrilets (1997), Nuismer *et al.* (2010), Yoder and Nuismer (2010) stabilizing selection has been modelled through a reduction of density-independent intrinsic growth. Parameter σ_c of the competition kernel measures a characteristic *range of competitive interference*: with small σ_c , competition mostly happens between very similar

organisms. Parameter σ_s of the carrying capacity measures a characteristic *range of optimal trait values*: with small σ_s , stabilizing selection is strong and only individuals with trait values close to the optimum can have high carrying capacity.

In this model, depending on parameter values the population evolves to one of two possible equilibrium states (Doebeli and Ispolatov, 2010). Specifically, if stabilizing selection is relatively strong, i.e. the range of optimal trait values is smaller than the range of competitive interference ($\sigma_s \leq \sigma_c$), the population becomes monomorphic with the optimum trait ($\bar{x} = \theta$) and the equilibrium population size $N^* = K_0$. This outcome is similar to that in the standard logistic model. However, if stabilizing selection is relatively weak (i.e., if $\sigma_s > \sigma_c$), then the equilibrium distribution is Gaussian (i.e.,

$$\phi^*(x) = \frac{\sigma_s}{\sigma_c} \frac{K_0}{\sqrt{2\pi(\sigma_s^2 - \sigma_c^2)}} \exp\left(-\frac{(x - \theta)^2}{2(\sigma_s^2 - \sigma_c^2)}\right)$$

) with the mean at the optimum ($\bar{x} = \theta$) and a positive variance $v = \sigma_s^2 - \sigma_c^2$ which increases with decreasing σ_c . That is, if competition between dissimilar individuals is weak, variation can be maintained. The corresponding equilibrium population size is $N^* = K_0\sigma_s/\sigma_c$ which is always larger than the population size K_0 at the monomorphic state. That is, weak stabilizing selection or a large range of competitive interference lead to the maintenance of phenotypic variation and an increase in the population size (Figure 2.1). Therefore stabilizing selection affects the population size which in turn affects phenotypic variance.

Below we will generalize this approach for three different two-species models which we will study using analytical approximations and numerical solutions (see the Supplementary Information for details on our numerical method).

2.3 Two-species competition

The standard Lotka-Volterra competition model describes the dynamics of two competing species with densities N_1 and N_2 :

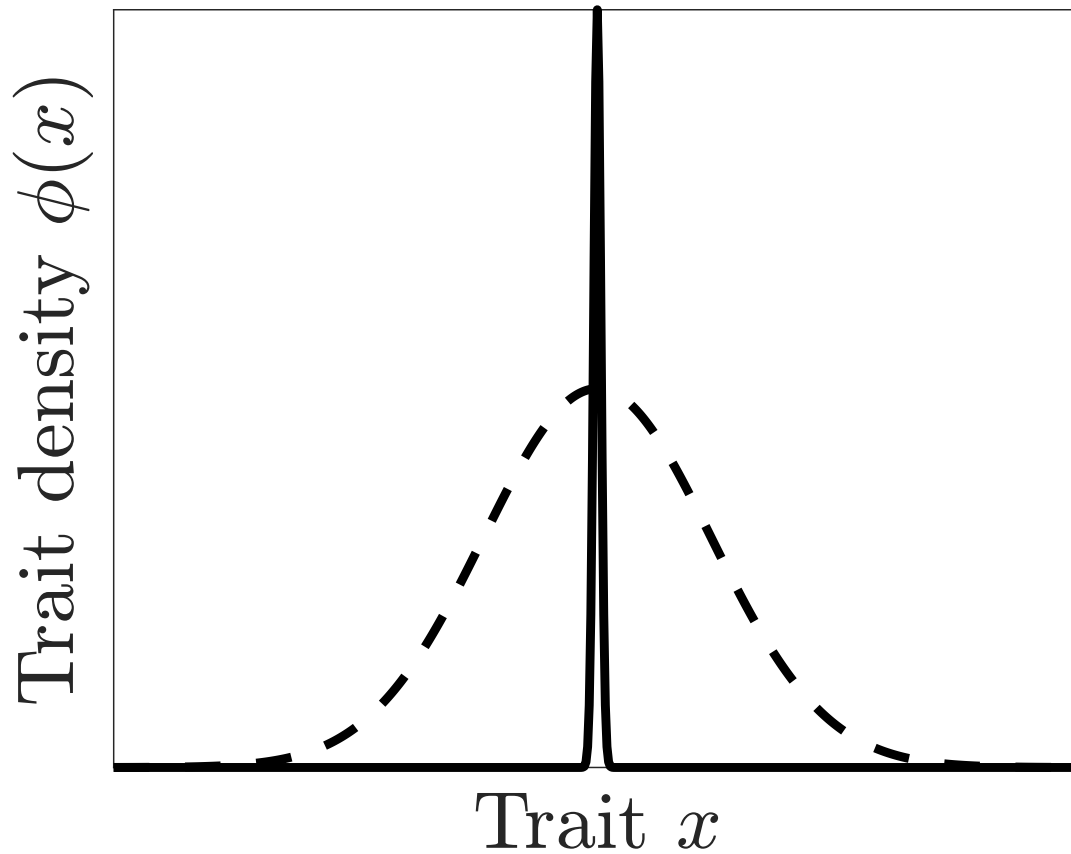


Figure 2.1: Equilibrium trait distributions in the single-species model. With stronger stabilizing selection ($\sigma_c^2 \geq \sigma_s^2$), the population becomes monomorphic at the optimum trait value $x = \theta$ (solid line). With stronger competition ($\sigma_c^2 < \sigma_s^2$), the equilibrium trait distribution is Gaussian with a positive variance $v = \sigma_s^2 - \sigma_c^2$ (dashed line).

$$\frac{dN_1}{dt} = r_1 N_1 \left(1 - \frac{N_1 + \alpha_{12} N_2}{K_1} \right), \quad (2.3a)$$

$$\frac{dN_2}{dt} = r_2 N_2 \left(1 - \frac{\alpha_{21} N_1 + N_2}{K_2} \right). \quad (2.3b)$$

Here, for species i ($i = 1, 2$), r_i is the growth rate at low densities, K_i is the carrying capacity in the absence of the competing species, and parameters α_{12} and α_{21} represent the strength of between-species competition relative to that within species. In this model, the necessary and sufficient condition for coexistence is that within-species competition is stronger than between species competition for both species: $\alpha_{12} K_2 / K_1 < 1$ and $\alpha_{21} K_1 / K_2 < 1$. If the condition is satisfied in one species (say $\alpha_{12} K_2 / K_1 < 1$) and not satisfied in the other species (so that $\alpha_{21} K_1 / K_2 \geq 1$) then the first species survives and the second species goes extinct. We then say that the first species 1 is a stronger competitor and species 2 is a weaker competitor. If $\alpha_{12} K_2 / K_1 \geq 1$ and $\alpha_{21} K_1 / K_2 \geq 1$, then one species survives and the other becomes extinct based on initial conditions.

We extend the Lotka-Volterra competition model by allowing individuals to differ in a continuous trait x in the first species and y in the second. We do this by adapting the single species approach described above:

$$\frac{\partial \phi_1(x, t)}{\partial t} = r_1 \phi_1(x, t) \left(1 - \frac{\int C_{11}(x, z) \phi_1(z, t) dz + \alpha_{12} \int C_{12}(x, z) \phi_2(z, t) dz}{\kappa_1(x)} \right), \quad (2.4a)$$

$$\frac{\partial \phi_2(y, t)}{\partial t} = r_2 \phi_2(y, t) \left(1 - \frac{\alpha_{21} \int C_{21}(y, z) \phi_1(z, t) dz + \int C_{22}(y, z) \phi_2(z, t) dz}{\kappa_2(y)} \right). \quad (2.4b)$$

Here ϕ_i, r_i and κ_i are the population density of the trait, the intrinsic growth rate, and carrying capacity for species i , and C_{ij} are the corresponding competition kernels. As above, we assume that carrying capacity, intraspecific competition, and interspecific competition kernel functions are Gaussian: $\kappa_i(z) = K_i \exp(-(z - \theta_i)^2 / 2\sigma_{si}^2)$, $C_{ii}(x, y) = \exp(-(x - y)^2 / 2\sigma_{ci}^2)$, and $C_{ij}(x, y) = \exp(-(x - y)^2 / 2\sigma_{cij}^2)$ respectively. Here θ_i, σ_{ci} , and σ_{si} are the optimum trait value, the range of within-species competitive interference, and the range of optimal trait values for species i , σ_{c12} and σ_{c21} measure the ranges of

between-species competitive interference, whereas α_{12} and α_{21} measure the strength of interspecific competition due to trait-independent between-species differences. Our choice of the competition kernel implies that competition is stronger among individuals with similar traits. Although this is a standard assumption in niche theory (MacArthur and Levins, 1967, Roughgarden, 1972), there are biological scenarios where the effects of competition are stronger when individuals are dissimilar. For example, exploitative competition mediated by a trait that allows for faster exploitation will result in stronger competition between individuals with more different traits values.

2.3.1 Results

To find sufficient conditions for coexistence we used mutual invasibility analysis (Armstrong and McGehee, 1980, Geritz *et al.*, 1998). The idea underlying this method is that the two species will coexist only if each of them can invade a resident population of the other species at equilibrium. For example, in the Lotka-Volterra competition model described above, species 2 can invade a resident population of species 1 at equilibrium ($N_1 = K_1$) from low population density ($N_2 \approx 0$) only when $dN_2/dt > 0$. This gives the invasion criterion for species 2: $\alpha_{21}K_1/K_2 < 1$. Invasion criteria are sufficient conditions for coexistence since they guarantee neither species can go extinct. Figure 2.2a illustrates the analytical results for our two-species competition model.

Assume first that the resident population is monomorphic which is the case if stabilizing selection is sufficiently strong ($\sigma_{sj} \leq \sigma_{cj}$, region A in Figure 2.2a). Then the invasion criterion for the invader species i is identical to that in the standard Lotka-Volterra competition model:

$$\alpha_{ij} \frac{K_j}{K_i} < 1 \quad (2.5a)$$

(see Appendix A.1 for details). Assume next that the resident population is polymorphic which is the case if $\sigma_{sj} \geq \sigma_{cj}$. From the single-species model, the variance of a polymorphic resident population is $v_j = \sigma_{sj}^2 - \sigma_{cj}^2$ and population density is $N_j^* = K_j \sigma_{sj} / \sigma_{cj}$. In this case, if stabilizing selection in the invader is weak enough ($\sigma_{si}^2 \geq v_j + \sigma_{cij}^2$, region B in Figure 2.2a), it will invade always. Otherwise, invasion happens whenever

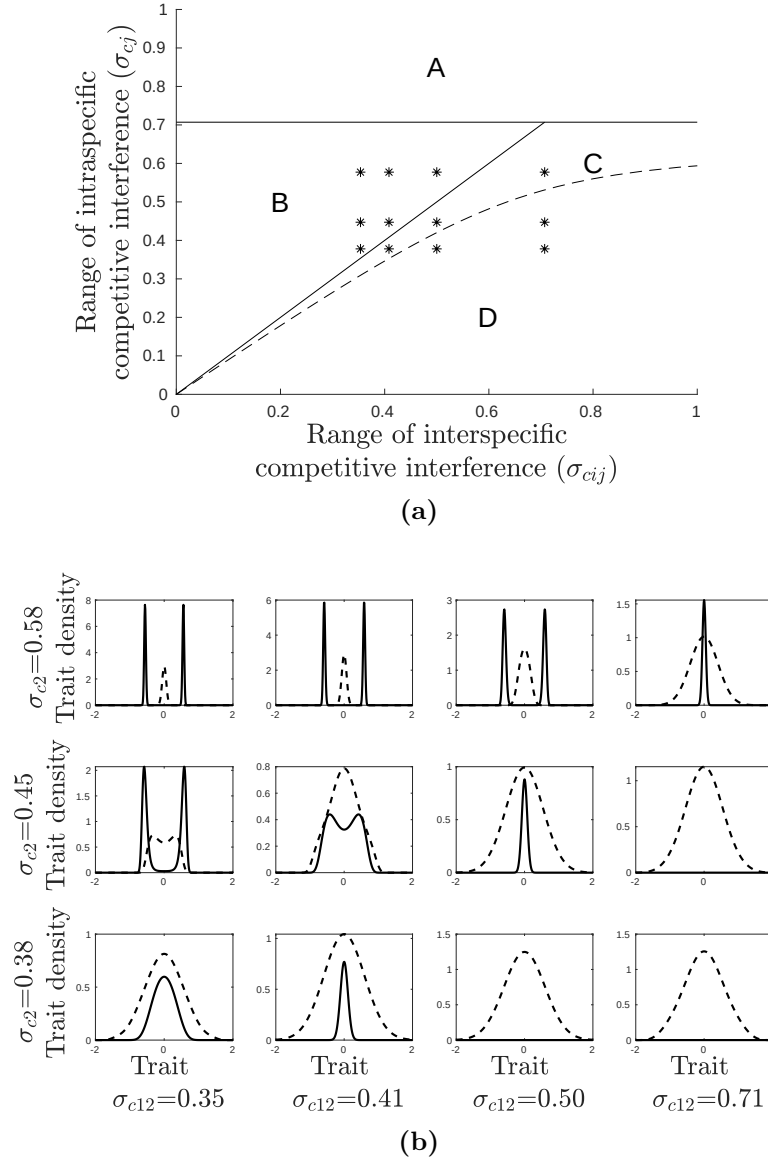


Figure 2.2: (a) Conditions for invasion as a function of the ranges of between-species σ_{cij} and within-species σ_{cj} competitive interference. In region A, $\sigma_{cj} > \sigma_{sj}$, the resident is monomorphic, and the invasion condition is based on inequality (2.5a). The line separating regions B and C is based on whether stabilizing selection on the invader is sufficiently weak to guarantee invasion ($\sigma_{si}^2 \geq v_j + \sigma_{cij}^2$ in region B). In region B, invasion is guaranteed for any choice of α_{ij} . Inequality (2.5b) determines the boundary between region C and D. It is satisfied in region C and not satisfied in region D. The black dots corresponds to the parameter choice for the numerical simulations illustrated in Figure 2.2b below. Other parameters: $\sigma_{sj} = 0.71, \delta = 0, \alpha_{ij} = 0.9$. (b) Equilibrium trait distributions of species 1 (solid line) and species 2 (dashed line) from numerical simulations for parameters values σ_{cij} and σ_{cj} marked by asterisks in Figure 2.2a. Identical initial phenotypic distributions $\phi(z) = 0.1 \exp(-10z^2)$. For all the simulations, $\alpha_{12} = 0.9, \alpha_{21} = 0.8, \sigma_{c1} = 0.71, \sigma_{c21} = 0.71, \sigma_{s1} = 0.71, \sigma_{s2} = 0.71, \delta = 0, r_1 = 1, r_2 = 1, K_1 = 1, K_2 = 1$.

$$\alpha_{ij} \frac{N_j^*}{K_i} < \sqrt{1 + \frac{v_j}{\sigma_{cij}^2}} \exp \left(\frac{\delta^2}{2(v_j + \sigma_{cij}^2 - \sigma_{si}^2)} \right). \quad (2.5b)$$

where $\delta = |\theta_i - \theta_j|$ is the difference between the optimum trait values. In Figure 2.2a, the inequality holds in region C and not in region D. Note that increasing the difference in the optimum trait values δ , decreasing the strength of stabilizing selection in the invader (i.e., increasing σ_{si}), or decreasing the range of between-species competitive interference σ_{cij} always increases the right-hand side of equation (2.5b) and, thus, makes invasion easier. If $\delta = 0$, increasing the ratio v_j/σ_{cij}^2 , i.e. decreasing the range of between-species competitive interference relative to the phenotypic variance in the resident species, makes invasion easier.

The invasion analysis provides only sufficient conditions for coexistence and does not tell anything about the equilibrium distributions of traits. Unlike the single-species model, the equilibrium distribution for the two-species model cannot be determined analytically. Therefore we supplement our results with a numerical study of the model dynamics for different combinations of parameters and initial conditions using an adaptive finite difference method (see Appendix A.2, also Doebeli (2011)). We describe our results next. In all cases considered, the system evolves to an equilibrium. If only one species survives, the equilibrium trait distribution matches the one predicted by equation (2.2).

At a coexistence equilibrium with both species polymorphic there are three possibilities: (i) both species are unimodal, (ii) one species is unimodal and another bimodal, or (iii) both species are bimodal. These outcomes are illustrated in Figure 2.2b. The values of range of interspecific (σ_{cij}) and intraspecific (σ_{ci}) competitive interference shown in these graphs are marked by asterisks in Figure 2.2a. Assuming that species 2 can invade a resident population of species 1 (i.e. that inequality (2.5b) holds for $i = 2, j = 1$), parameters relevant for species 1's invasion may lie in one of the regions A-C. In region B, species 1 is bimodal if the range of within-species competitive interference in species 2 is large and unimodal otherwise. Species 2 is bimodal only if the range of within-species competitive interference in species 2 is intermediate. In region A and C, both species are unimodal.

In the single species model, the equilibrium population size and variance decrease with the range of within-species competitive interaction σ_{ci} (see above). To explore the effects

of σ_{ci} in the case of two coexisting species, we varied σ_{c2} assuming that parameters for species 1 are in regions B or C, and for species 2 in region A of Figure 2.2a. Figure 2.3 shows the equilibrium variance and population size when the both species coexist (solid lines) and the analytical solution of the single-species model (dashed line). We find that the equilibrium variance of species 2 decreases with σ_{c2} and is always smaller when the other species is present. Equilibrium variances of the two species are inversely related. Similarly, the equilibrium population size of species 2 decreases with σ_{c2} whereas that of species 1 increases with σ_{c2} . Equilibrium population sizes of both the species are lower than their respective single-species equilibrium.

Summarizing, evolving phenotypic variance can promote coexistence of competitors when stabilizing selection is weak and the range of between-species interference is small. To survive, the weaker competitor reduces negative effects of competition by having a narrow trait distribution or by diversifying around the stronger competitor into a bimodal distribution. (Note that such a behavior is outside the realm of mean-focused approaches). At the coexistence equilibrium, the population size and phenotypic variance decrease with the range of intraspecific competitive interference similarly to what happens in the single species model.

2.4 Exploiter-victim interactions

Here we consider exploiter-victim interactions such as between a predator and a prey or a parasite and a host (Gavrilets, 1997, Clayton *et al.*, 1999, Soler *et al.*, 2001, Toju and Sota, 2006, Davies and Brooke, 1988). Writing the population density of the victim species as $N_1(t)$ and that of the exploiter species as $N_2(t)$, and assuming that the exploiter has an obligate relationship with the victim, we start with the model

$$\frac{dN_1}{dt} = \alpha N_1 \left(1 - \frac{N_1}{K_1} \right) - \zeta N_2 N_1, \quad (2.6a)$$

$$\frac{dN_2}{dt} = \beta N_1 N_2 - \gamma N_2 \left(1 + \frac{N_2}{K_2} \right). \quad (2.6b)$$

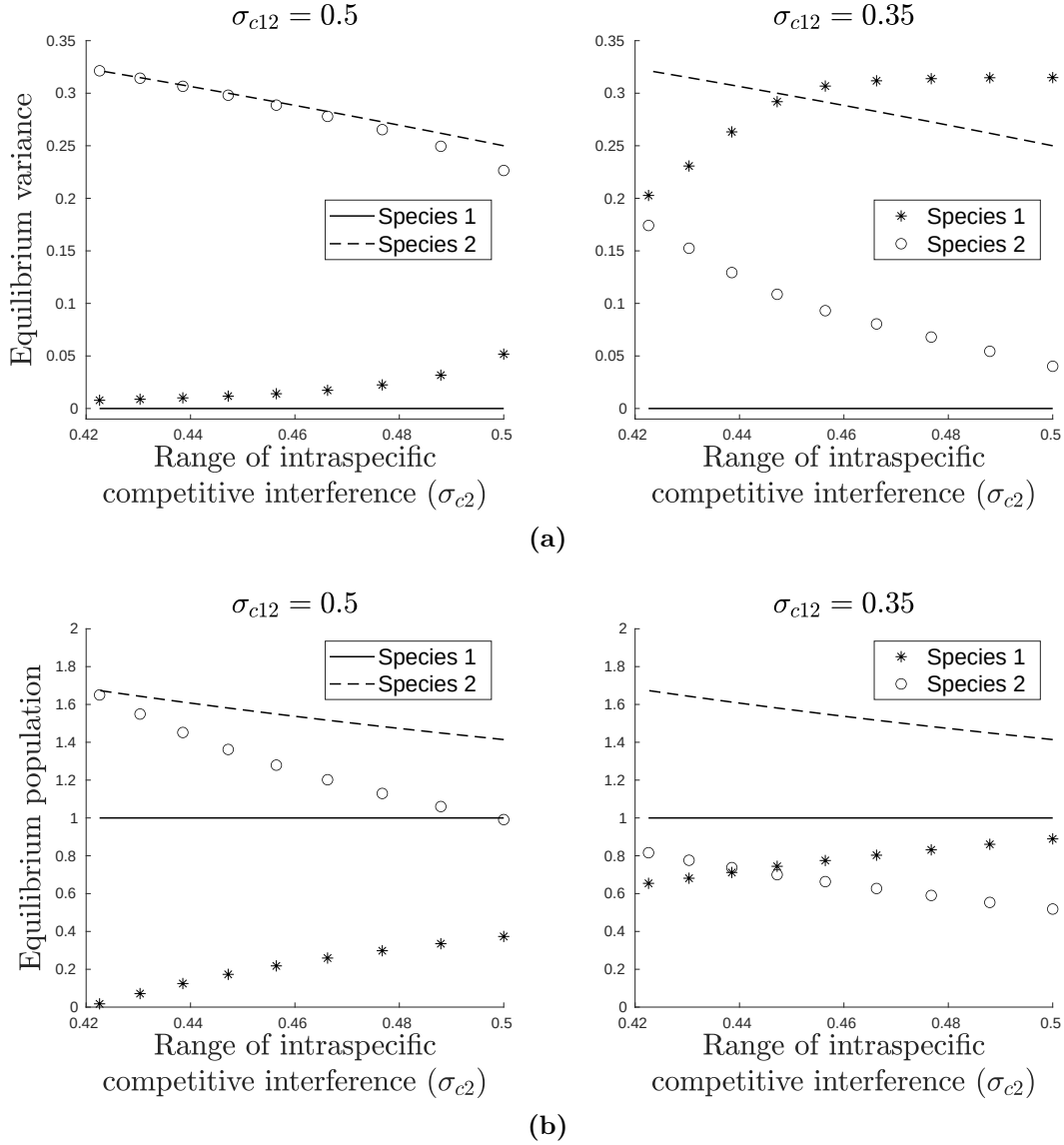


Figure 2.3: Effect of the range of within-species competitive interference σ_{c2} on the equilibrium variance and equilibrium population sizes of the two species. The dashed lines are based on the equilibrium of the single-species model and the solid lines are based on numerical solutions of the two-species model with identical initial phenotypic distributions $\phi(z) = 0.1 \exp(-10z^2)$. The left and right panels correspond to coexistence in region C and B in figure 2.2a, respectively. Other parameters: $\alpha_{12} = 0.9, \alpha_{21} = 0.8, \sigma_{c1} = 0.71, \sigma_{c21} = 0.71, \sigma_{s1} = 0.71, \sigma_{s2} = 0.71, \delta = 0, r_1 = 1, r_2 = 1, K_1 = 1, K_2 = 1$.

Here, the intrinsic growth rate of the victim is α , the intrinsic death rate of the exploiter is γ , β and ζ are the exploiter birth rate and victim death rate due to exploitation, K_1 and K_2 are characteristic population densities. This is a generalization of the classical [Lotka \(1920\)](#) model to which we have added a quadratic death rate term due to within-species competition. [This change also allows one to avoid structural instability inherent in the Lotka-Volterra model ([Kot, 2001](#)). We recover the Lotka-Volterra model in the limit of large K_1 and K_2 .]

In this model, both species coexist at an asymptotically stable equilibrium if

$$\frac{\beta K_1}{\gamma} > 1. \quad (2.7)$$

The numerator of the above ratio is the growth rate of the exploiter when the victim is at carrying capacity (i.e., $N_1 = K_1$) and the denominator is the exploiter's death rate at low densities. The corresponding equilibrium densities are $N_1^* = \gamma K_1(\alpha + \zeta K_2)/(\alpha\gamma + \beta\zeta K_1 K_2)$ and $N_2^* = \alpha K_2(\beta K_1 - \gamma)/(\alpha\gamma + \beta\zeta K_1 K_2)$. If the inequality above is reversed, only the victim species survives. This model does not lead to exploiter-victim cycles.

We extend the above model to individuals differing in continuous traits x in the victim and y in exploiter:

$$\frac{\partial \phi_1(x, t)}{\partial t} = \alpha \phi_1(x, t) \left(1 - \frac{\int C_1(x, z) \phi_1(z, t) dz}{\kappa_1(x)} \right) - \zeta \phi_1(x, t) \int D(x, z) \phi_2(z, t) dz, \quad (2.8a)$$

$$\frac{\partial \phi_2(y, t)}{\partial t} = \beta \phi_2(y, t) \int D(y, z) \phi_1(z, t) dz - \gamma \phi_2(y, t) \left(1 + \frac{\int C_2(y, z) \phi_2(z, t) dz}{\kappa_2(y)} \right). \quad (2.8b)$$

Here $\phi_1(x, t)$ and $\phi_2(y, t)$ are the corresponding densities of the traits, and parameters α, β, γ and ζ have the same meaning as above. Similar to the competition model with continuous traits, we assume that selection and within-species competition kernels are Gaussian: $\kappa_i(z) = K_i \exp(-(z - \theta_i)^2/2\sigma_{si}^2)$ and $C_i(x, y) = \exp(-(x - y)^2/2\sigma_{ci}^2)$, where parameters θ_i , σ_{ci}^2 and σ_{si}^2 have the same meaning as before. Assuming that exploiter-victim interactions are based on trait matching ([Gavrilets, 1997](#), [Yoder and Nuismer, 2010](#)), the exploitation kernel can be modelled as a Gaussian function: $D(x, y) = \exp(-(x - y)^2/2\sigma_d^2)$, where σ_d^2 measures the *range of exploitative interactions*. For example, with small σ_d^2 the exploiter can utilize only

victims with very similar trait values. This assumption restricts our model to exploitation based on trait-matching, such as brood parasitism where the egg colour, shape, texture should match between parasite and host.

2.4.1 Results

Using the invasibility analysis, for the exploiter to coexist with the victim, it should be able to grow from small densities. Consider first the case where stabilizing selection in the victim is strong ($\sigma_{c1} \geq \sigma_{s1}$). In this case, the victim is monomorphic and the sufficient conditions for coexistence is identical to the coexistence condition from the model with no individual variation (inequality (2.7)). In contrast, if $\sigma_{c1} < \sigma_{s1}$, the victim is polymorphic with equilibrium variance $v_1 = \sigma_{s1}^2 - \sigma_{c1}^2$ and equilibrium population size $N_1^* = K_1 \sigma_{s1} / \sigma_{c1}$ in the victim-only model. In this case, the sufficient condition for a successful invasion of the exploiter (and thus for coexistence) is

$$\frac{\beta N_1^*}{\gamma} > \sqrt{1 + \frac{v_1}{\sigma_d^2}}. \quad (2.9)$$

In particular, this shows that increasing the range of exploitative interactions σ_d simplifies survival of the exploiter.

We did not observe any non-equilibrium dynamics in numerical simulations. The equilibrium trait distribution of the victim matched the single-species equilibrium when the exploiter did not survive. Figure 2.4 illustrates trait distributions when the species coexist. The distributions are unimodal if the exploiter death rate (γ) is large. If it is small, the victim diversifies around the exploiter to survive. If stabilizing selection in the victim (σ_{s1}) is weak, the diversification in the victim can be followed by that in the exploiter (top right graph in Figure 2.4).

The equilibrium variance in both species increases with the range of competition in victims (Figure 2.5). The exploiter's variance is smaller than that in the victim even when the exploiter trait distribution is bimodal ($\sigma_{s1} = 1, \gamma = 0.5$ in Figure 2.4). Figure 2.5 also shows that if the exploiter survives, the victim's variance is larger and the population size is smaller than the equilibrium values of the victim-only model.

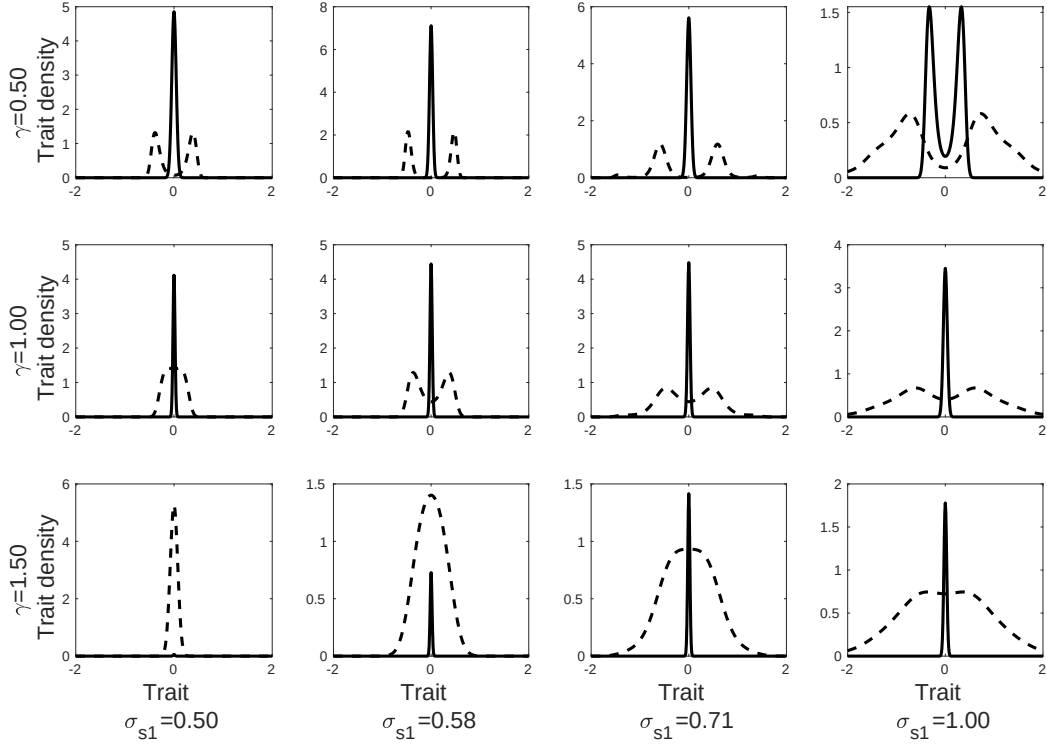


Figure 2.4: Examples of equilibrium trait distributions of the exploiter (solid line) and victim (dashed line) from numerical simulations with identical initial trait distributions $\phi(z) = 0.1 \exp(-10z^2)$. Other parameters: $\alpha = 1, \beta = 1.5, \zeta = 1, \sigma_{c1} = 0.5, \sigma_{c2} = 0.71, \sigma_d = 0.71, \delta = 0, K_1 = 1, K_2 = 1$.

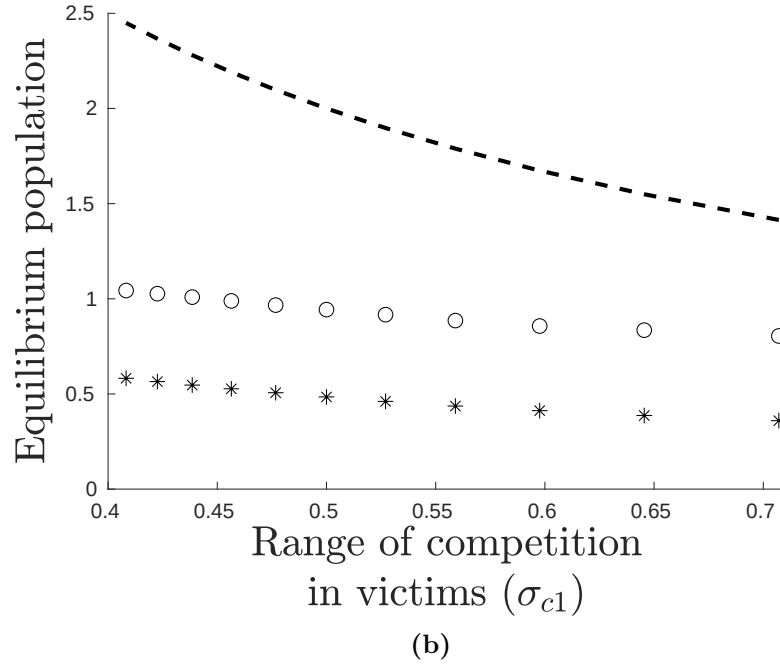
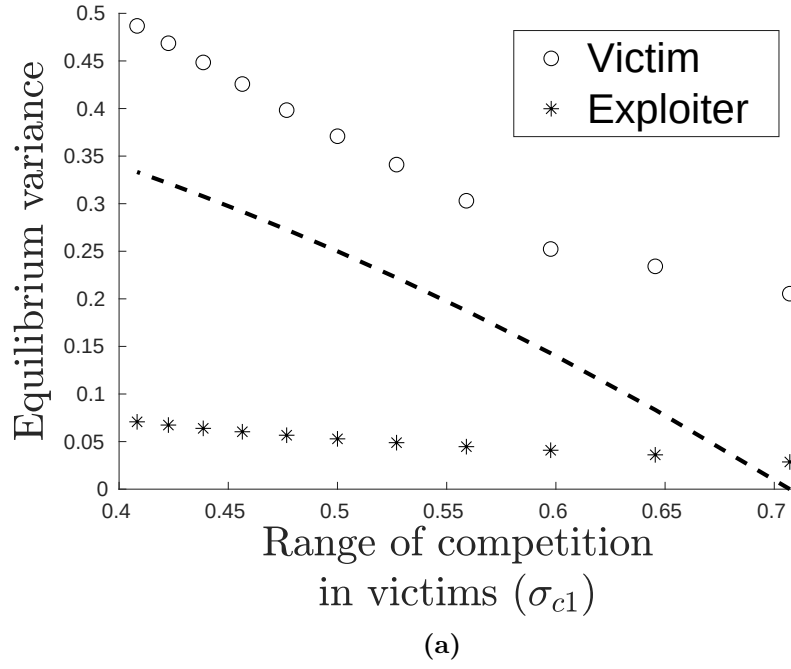


Figure 2.5: The equilibrium variances and population sizes of both species when the exploiter survives. The dashed line is the equilibrium variance and population of the victim in the single-species model, and the points are from numerical simulations using identical phenotypic distribution for the victim and exploiter ($\phi(z) = 0.1 \exp(-10z^2)$). Other parameters: $\alpha = 1, \beta = 2, \zeta = 1, \gamma = 1, \sigma_{c2} = 0.32, \sigma_{s1} = 0.71, \sigma_{s2} = 0.71, \delta = 0, K_1 = 1, K_2 = 1$.

Summarizing, evolving phenotypic variation can promote the exploiter’s persistence when stabilizing selection is weak and the range of exploitative interaction is large. Exploiters can persist by matching the (unimodal or bimodal) trait distribution in the victim. The conditions for the victim and exploiter to diversify are in agreement with those found using a mean-focused adaptive dynamics approach (Dieckmann and Doebeli, 1999). The equilibrium variance in the victim increases when the exploiter persists and it decreases with the range of intraspecific competition similarly to what happens in the single species model. The equilibrium population size and variance of the exploiter and victim are positively correlated.

2.5 Mutualism

In this section we consider mutualistic interactions where both species benefit from the interaction (Bronstein, 2015). We start with a classical mutualism model (Kostitzin, 1939, Gause and Witt, 1935) describing the dynamics of a pair of mutualistic partners with densities N_1 and N_2 :

$$\frac{dN_1}{dt} = r_1 N_1 \left(1 - \frac{N_1}{K_1} + B_1 N_2 \right), \quad (2.10a)$$

$$\frac{dN_2}{dt} = r_2 N_2 \left(1 - \frac{N_2}{K_2} + B_2 N_1 \right). \quad (2.10b)$$

Here, the mutualistic benefit for species i is denoted by B_i and the carrying capacity is denoted by K_i . A stable equilibrium exists if and only if $B_1 B_2 K_1 K_2 < 1$. At this equilibrium, the population density of species i is $K_i(1 + B_i K_j)/(1 - B_i B_j K_i K_j)$. If the above inequality does not hold, both species grow to infinite sizes. This unbounded growth is sometimes referred to as the “orgy of beneficial mutualism” (May, 1981).

Allowing for individual differences in continuous traits x in the first species and y in the second species, the corresponding dynamics of the mutualistic system are described by equations

$$\frac{\partial \phi_1(x, t)}{\partial t} = \phi_1(x, t) \left(r_1 - \frac{\int C_1(x, z) \phi_1(z, t) dz}{\kappa_1(x)} + B_1 \int M_1(x, z) \phi_2(z, t) dz \right), \quad (2.11a)$$

$$\frac{\partial \phi_2(y, t)}{\partial t} = \phi_2(y, t) \left(r_2 - \frac{\int C_2(y, z) \phi_2(z, t) dz}{\kappa_2(y)} + B_2 \int M_2(y, z) \phi_1(z, t) dz \right). \quad (2.11b)$$

Here ϕ_i , r_i , and κ_i represent the population density of the trait, the intrinsic growth rate, carrying capacity for species i , and C_i are the corresponding competition kernels. Similar to the other models, we assume carrying capacity and competition kernel functions are Gaussian: $\kappa_i(z) = K_{i,0} \exp(-(z - \theta_i)^2/2\sigma_{si}^2)$ and $C_i(x, y) = \exp(-(x - y)^2/2\sigma_{ci}^2)$. Assuming that mutualism is based on trait matching (Brouat *et al.*, 2001, Yoder and Nuismer, 2010), the mutualism kernel M_i can be modelled by a Gaussian function: $M_i(x, y) = \exp(-(x - y)^2/2\sigma_{mi}^2)$, where σ_{mi} measures the *range of mutualistic interactions*, *i.e.* range of phenotypes over which an individual receives mutualistic benefits. Parameters B_i represent mutualistic benefits due to other trait independent factors. Similar to our competition and exploiter-victim models, interspecific interaction is modelled as trait-matching. Many mutualisms based on morphological or genetic matching between mutualists will fit this description.

2.5.1 Results

Similar to the case of no intraspecific variation, in this model the two species either reach an equilibrium with finite population sizes or grow to infinite population sizes due a positive feedback of mutualistic benefits. To find the conditions for the population size to reach a stable equilibrium, an approach similar to the invasion analysis does not work. Instead we first find upper bounds for the time series of population sizes of the two species, and then determine conditions for the upper bound to converge. This gives us the necessary conditions for coexistence in a stable equilibrium (see Appendix A.1 for details).

Our results are as follows. If stabilizing selection is sufficiently strong ($\sigma_{ci} \geq \sigma_{si}$) in both species, then both species are monomorphic and the necessary condition for a stable coexistence equilibrium is identical to that in the model with no intraspecific variation:

$$B_1 B_2 K_1 K_2 < 1. \quad (2.12a)$$

If stabilizing selection is strong in only one of the species (say, 2nd), then the necessary condition for a stable coexistence equilibrium is

$$B_1 B_2 N_1^* K_2 < \sqrt{1 + \frac{v_1}{\sigma_{m2}^2}}. \quad (2.12b)$$

Here $N_i^* = K_i \sigma_{si} / \sigma_{ci}$ and $v_i = \sigma_{si}^2 - \sigma_{ci}^2$ are the equilibrium population size and variance of the single-species model for species 1. This shows that if stabilizing selection is strong in one of the species, the equilibrium is easier to achieve when the range of mutualistic interactions (σ_{m2}) is small.

Finally, if stabilizing selection is weak in both species, then the necessary condition for a stable coexistence equilibrium is

$$B_1 B_2 N_1^* N_2^* < \sqrt{\left(1 + \frac{v_1}{\sigma_{m2}^2}\right) \left(1 + \frac{v_2}{\sigma_{m1}^2}\right)}. \quad (2.12c)$$

In contrast to the case of strong stabilizing selection in only one of the species, here an equilibrium is easier to achieve when the range of mutualistic benefits is small in either of the two species. Overall, if variation is maintained (i one of both species), it is less likely that their population growth would be unbounded.

Numerical exploration of the model revealed two types of polymorphic equilibrium trait distributions: (i) unimodal (Figure 2.6a), or (ii) multimodal (Figure 2.6b). In the multimodal case, there is one large peak and two smaller peaks (Figure 2.6b) or one large and one small peak. The latter case is observed when δ is large and the former when δ is small. These additional smaller peaks exist because with large range of within-species competition σ_{ci} , mutualistic benefits do not decrease as quickly as competitive costs (decrease in growth rate due to competition) for traits farther away from the mean of the distribution.

We also found that as the range of within-species competition decreases, the equilibrium variance increases (Figure 2.7a). Figure 2.7 show that the equilibrium variance increases with smaller range of within-species competition irrespective of the range of within-species competition in the other species. Also, the equilibrium variance is always smaller than the equilibrium variance of the single-species model. In comparison to the single-species model,

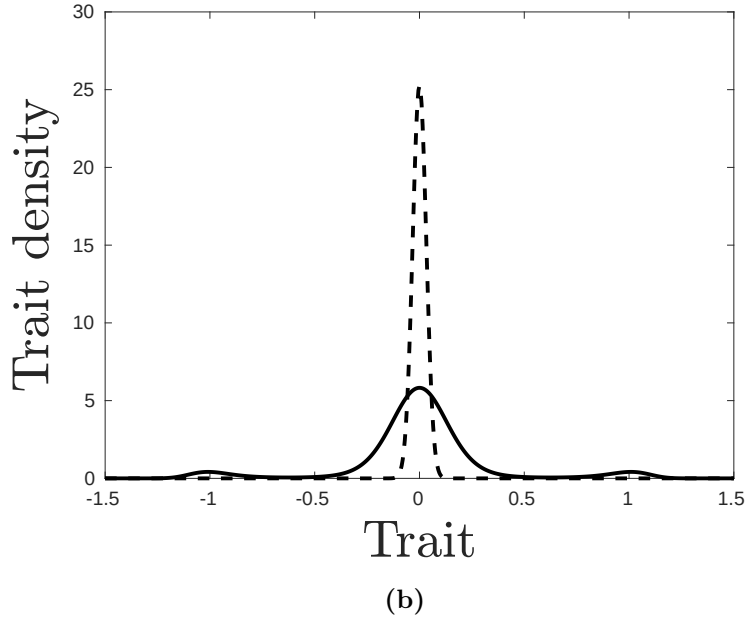
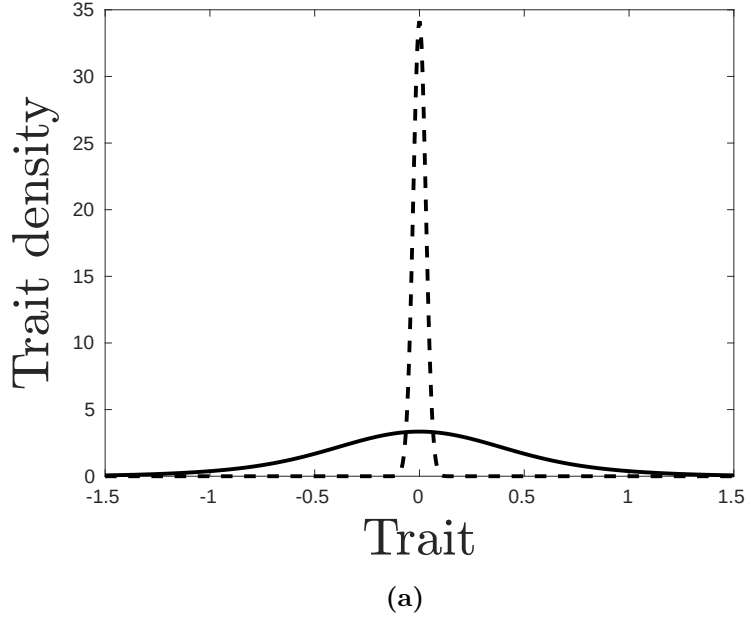


Figure 2.6: Common equilibrium trait distributions of mutualists. (a) $\sigma_{c1} = 0.35$ and (b) $\sigma_{c1} = 0.53$. Mutualist 1 (solid line) and mutualist 2 (dashed line); identical initial trait distributions $\phi(z) = 0.1 \exp(-10z^2)$; other parameters $r_1 = 1, r_2 = 1, B_1 = 0.5, B_2 = 0.5, \sigma_{c2} = 0.71, \sigma_{s1} = 0.71, \sigma_{s2} = 0.71, \sigma_{m1} = 0.71, \sigma_{m2} = 0.71, \delta = 0, K_1 = 1, K_2 = 1$. For $\delta > 0$, the mean of the distributions will shift but the shape remains the same.

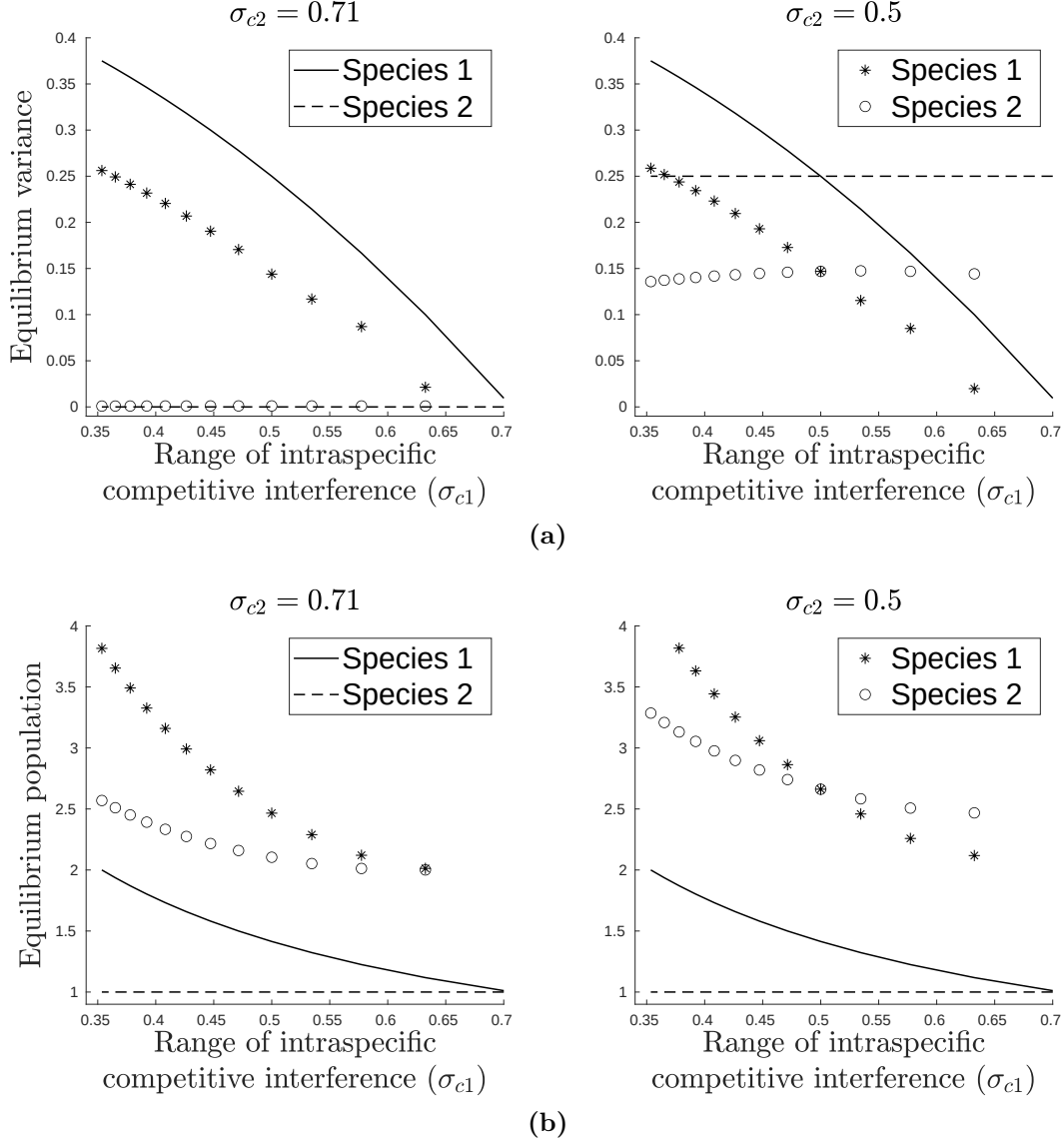


Figure 2.7: Effect of the range of within-species competitive interference on equilibrium variance and population size of the mutualists. The dashed and solid lines show the equilibrium of a single-species model, and the asterisks (species 1) and open circles (species 2) are based on the numerical solution of the two species model with initial phenotypic distributions $\phi(z) = 0.1 \exp(-10z^2)$. Other parameters: $r_1 = 1, r_2 = 1, \sigma_{s1} = 0.71, \sigma_{s2} = 0.71, \sigma_{m1} = 0.71, \sigma_{m2} = 0.71, B_1 = 0.5, B_2 = 0.5, \delta = 0, K_1 = 1, K_2 = 1$.

the equilibrium population sizes are higher when the mutualist partner is present. The equilibrium population size becomes larger as the range of within-species competition in either species decreases.

Summarizing, evolving phenotypic variation can stabilize mutualistic interactions when stabilizing selection is weak and the range of between-species mutualistic interaction is small. This happens because negative effects of competitive interactions become stronger than mutualistic benefits as the trait distributions align with each other. This is a novel mechanism for mutualists to coexist in a stable equilibrium which cannot be identified using a mean-focused approach. Phenotypic variance decreases with the range of intraspecific competition similarly to what happens in the single species model and the other two-species coevolutionary models. The equilibrium population size decreases with increasing the range of intraspecific competition in either species.

2.6 Discussion

Although the dynamic patterns of between-species interactions are expected to strongly depend on intraspecific variation, how exactly ecological and evolutionary processes interact is still largely an open question. Here we approached this question theoretically using three simple two-species models describing competition, exploiter-victim interaction, and mutualism. In our models, individuals differ with respect to a single quantitative character which controls both within- and between-species density-dependent interactions and, simultaneously, is subject to stabilizing natural selection. We analysed conditions for stable coexistence, equilibrium population sizes as well as the characteristics of trait distributions observed at equilibrium.

For intraspecific variation to be important in our models, it needs to be maintained. Our results show that without mutation, intraspecific variation is lost if stabilizing selection is strong enough, specifically, if the range of optimal traits values is narrower than the range of within-species competitive interference ($\sigma_s < \sigma_c$). In this case, the outcomes of population dynamics are the same as predicted by standard ecological models neglecting intraspecific variation. The condition $\sigma_s < \sigma_c$ for the loss of intraspecific variation is the same as the

one in single-species models (Roughgarden, 1972, Doebeli and Ispolatov, 2010). Intraspecific variation can be maintained if stabilizing selection is weak enough in at least one species of the pair. In the discussion below, we assume that this is the case.

We now summarize the results of our three models. Consider first between-species competition. In classical ecological models of competition, there are three possible outcomes: the extinction of a weaker competitor and persistence of a stronger competitor, survival of one species or another depending on initial densities, or coexistence. With intraspecific variation maintained, these three possible outcomes are still possible but the conditions for them to be observed depend on parameters characterizing ranges of interference. In particular, small ranges of between-species interference (σ_{cij}) make coexistence a more likely outcome. When the species coexist, their equilibrium trait distribution can be unimodal for both species, bimodal for both species, or unimodal for one species and bimodal for the other. The weaker competitor (based on ecological model) has a bimodal distribution when the range of between-species competition is large, and unimodal otherwise. The strong competitor has a unimodal trait distribution when the range of between-species competition is small or large, and a bimodal distribution for a narrow range of intermediate values.

Second, in classical ecological models of exploiter-victim interactions there are two possible outcomes: the victim species survives and the exploiter is extinct, or coexistence. These outcomes are also possible if within-species variation is maintained. In general, large ranges of exploitative interactions (σ_d) promotes survival of the exploiter. When the species coexist, their equilibrium trait distributions can be both unimodal, both bimodal, or unimodal in one species and bimodal in the other. When the exploiter's death rate (γ) is high, both the exploiter and victim have unimodal trait distributions at the coexistence equilibrium. When the exploiter's death rate is low, the victim diversifies and its equilibrium trait distribution is bimodal. If stabilizing selection in the victim is weak, its trait distribution becomes bimodal which can be followed by the evolution of bimodality in the exploiter.

Third, in classical ecological models of mutualism there are two possible outcomes: the two species coexist at finite population sizes, or both species grow indefinitely due to non-diminishing mutualistic benefits. Mutualists persist in both outcomes. With intraspecific variation maintained, a small range of mutualistic interactions (σ_m) promotes coexistence

in a stable equilibrium. The equilibrium trait distribution is typically unimodal, but can become multimodal (with two or three peaks) if stabilizing selection is strong enough. In this case, only one of the peaks has a high trait density, while other peak(s) have a much smaller trait density. When the mutualists' population size grows indefinitely, numerical instability makes it challenging to describe the limit of the normalized trait distribution.

In single-species models allowing for heritable intraspecific variation (such as given by equation (2.2)), the appearance of bimodal trait distributions or evolutionary branching require a non-Gaussian competition kernel, e.g. an asymmetric (Kisdi, 1999) or a platykurtic (Doebeli and Ispolatov, 2010). In contrast, our coevolutionary models show that species interactions could lead to bimodal trait distribution even for Gaussian competition and interaction functions. In our competition model, the equilibrium trait variances are smaller than those in the corresponding single-species model. For the exploiter-victim interaction, the equilibrium trait variance of the exploiter is always much smaller than that in the victim. In comparison to the single-species model, the victim always has a higher variance in presence of the exploiter. In the case of mutualism, equilibrium trait variances are smaller for both mutualist species compared to those in the corresponding single-species model. Overall, we find that coevolutionary interactions lead to smaller trait variances except for the victims in exploiter-victim interactions.

Both theoretical and empirical studies have explored the ecological consequences of intraspecific variation (Frankham, 1996, Gavrilets, 1997, Lichstein *et al.*, 2007, Breza *et al.*, 2012, Hart *et al.*, 2016, Des Roches *et al.*, 2018, Austin and Dunlap, 2019, Start, 2019, Start and Gilbert, 2019). However only few earlier theoretical studies allowed for within-species variation to evolve (Nuismer *et al.*, 2005, Kopp and Gavrilets, 2006). An interesting consequence of heritable trait variation is character displacement when between-species competition is reduced due to the divergence of mean phenotypes (Brown and Wilson, 1956, Schuler and McPhail, 1992, Dayan and Simberloff, 2005). Our results suggest competition can also be reduced due to a decrease in phenotypic variances, or when the distributions become multimodal. Evolving phenotypic variation can also have direct consequences for the mean trait values when the optimum traits of the coevolving species are different. For

example, the mean phenotype of the exploiter is predicted to be much closer to the victim than in the corresponding model with constant variances (see Appendix A.3 for details).

Our models show that the relationship between the range of optimum traits (which depends on the strength of stabilizing selection) and the ranges of within- and between-species interactions is an important determinant of coevolutionary dynamics. In general, a larger range of optimum traits relative to the range of within-species interactions leads to the maintenance of trait variation which in turn allows for competitors to coexist, exploiters to survive, and mutualists to coexist in a stable equilibrium. Increased evolutionary flexibility allowed for by intraspecific variation potentially offers a way to reconcile differences in empirical observations, some of which shows that intraspecific variation promotes coexistence (Jung *et al.*, 2010, Clark, 2010, Fricke *et al.*, 2019), while others suggest that it restricts it (Hausch *et al.*, 2018). The coexistence conditions we have derived extend coexistence theory (Chesson, 2000, Barabás *et al.*, 2018, Ellner *et al.*, 2019) by including the effect of heritable intraspecific variation, stabilizing selection, and trait-dependent competition. Our models also demonstrate that with heritable intraspecific variation maintained, the strength of trait-based interactions can change through time.

Cyclical dynamics in exploiter-victim interactions have been of great interest in ecological (Lotka, 1920, Turchin, 2003) and evolutionary (Gavrilets, 1997, Nuismer and Doebeli, 2004, Nuismer *et al.*, 2005, Kopp and Gavrilets, 2006) models. However cycling was not possible in our basic model (equations (2.6)) and we did not observe it in our extension of that model for the case of within-species variation. Nuismer *et al.* (2005) studied several coevolutionary models of exploiter-victim interactions which differed in their assumptions about the range of permissible phenotypes, presence of interaction-independent stabilizing selection, and whether genetic variance was fixed or evolved. They showed that evolutionary exploiter-victim cycles required evolving genetic variance or strong stabilizing selection when genetic variance was fixed or absent. Further evolutionary cycling was less likely when the trait interval was infinite, *i.e.* any trait value could be produced by mutation or was present initially (Nuismer and Doebeli, 2004, Nuismer *et al.*, 2005). In our exploiter-victim model, these conditions are satisfied so the lack of cycling is in line with earlier findings.

Our results show that exploiter-victim interactions and competition can lead to bimodal trait distributions when one species diversifies around the other one to reduce deleterious effects of ecological interactions. This situation is similar to the Buridan’s Ass regime in [Gavrilets and Waxman \(2002\)](#) where females diversify “around” males to reduce deleterious effects of sexual conflict. Bimodal distributions can emerge via the process of evolutionary branching ([Geritz *et al.*, 1998](#)) and can potentially lead to speciation. [Doebeli and Dieckmann \(2000\)](#) studied coevolutionary interaction between an exploiter and a victim using adaptive dynamics, and found that a large range of exploitative interaction and weak stabilizing selection in the victim can lead to evolutionary branching in exploiters. We find similar relationships between the range of exploitative interaction, the strength of stabilizing selection in the victim, and bimodality of the exploiter’s trait distribution when the exploiter’s death rate is high. [Yoder and Nuismer \(2010\)](#) studied phenotypic diversification in a metapopulation. They found that in coevolutionary interactions where fitness of at least one species is reduced when its traits match the other species, phenotypic diversity is higher compared to diversification by spatially variable selection without coevolution. We find that such costly trait matching leads to a higher trait variance in the victim, but can lead to a lower variance for a competitor. This is due to one of the competitor diversifying around the other competitor (*i.e.* bimodal distribution with the modes on either side of the mean trait of the other species), and competing from both directions of the mean trait value of the other species (see Figure 2.2b), $\sigma_{c2} = 0.58$, $\sigma_{c12} = 0.35$).

In all models considered here, species interactions are the strongest when traits match. But there are other ecological situations when interactions become stronger as the difference between trait values of the interactors increase. For example, differences in speed or toxicity can determine success of exploitation in exploiter-victim systems. The differences between mathematical models based on trait-matching and trait-difference have been extensively studied for exploiter-victim interactions ([Abrams and Matsuda, 1997](#), [Abrams, 2000](#), [Yoder and Nuismer, 2010](#), [Yamamichi *et al.*, 2019](#)). In these models, selective pressure on the victim induced by the exploiter is disruptive for trait-matching interactions but directional for trait-difference based interactions. Similar effects are expected in competitive and mutualistic

interactions. Expected ecological and evolutionary consequences of these effects should be addressed in future studies.

The main limitations to our approach in terms of biological realism are that we ignored mutation and sexual reproduction. Including mutation would make it easier to maintain intraspecific variation at low levels but is not expected to significantly change our results. For analytical convenience, we assumed clonal reproduction in all our models neglecting the homogenizing force of sexual reproduction. Earlier theoretical studies of coevolution find that genetic details can lead to novel dynamics (Kopp and Gavrillets, 2006, Nuismer and Doebeli, 2004, Nuismer *et al.*, 2005, Hayashi *et al.*, 2007). Some of the effects we observed (e.g., bimodal trait distributions) may be an artifact of our simplifying assumptions. Future work should focus on extending these models to sexually reproducing populations. Our findings will generalize to ecological interactions between different behavioral or morphological types of the same asexual species. They also should hold for sexually reproducing species with strong assortative mating between different behavioral or morphological types. Our approach can potentially be extended to sexually reproducing species and coevolution between the sexes using reproduction kernels method (Doebeli, 2011), or multilocus population genetics models (Gavrillets and Waxman, 2002, Bolnick and Doebeli, 2003). The analytical conditions we have found for stable coexistence are only sufficient (but not necessary) in the case of competition and exploiter-victim interaction, and necessary (but not sufficient) in the case of mutualism.

Our work adds to the toolkit of theoretical studies of coevolution a numerical approach (described in the SI) for modeling the dynamics of population densities and phenotypic distributions under different two-species interactions. We have also developed a novel application of the invasion analysis (Armstrong and McGehee, 1980), and derived an approximate analytical condition for existence of a stable coexistence equilibrium for mutualists (Appendix A.1). Our methods allowed us to obtain analytical results without the common assumption of weak selection in coevolutionary models (Gavrillets, 1997, Nuismer and Doebeli, 2004). Overall, our approach allows us to understand better the role of heritable trait variation in coevolutionary systems, which is the first step towards achieving a better

understanding of the implications of heritable trait variation in evolutionary community ecology (McPeck, 2017).

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Statement of authorship

A.S. designed the research. A.S. performed the research with contributions from S.G. A.S. and S.G. wrote the manuscript.

Chapter 3

Biotic-abiotic interactions

Publication statement

This chapter is a reprint of a paper that will be submitted for publication:

“Senthilnathan, A. and Gavrilets, S. (2021). Ecological and eco-evolutionary dynamics of biotic-abiotic feedbacks.”

Abstract

Interactions between organisms and their abiotic environment is fundamental to both ecology and evolution. The majority of the empirical and theoretical studies have focused either on the effects of organisms on their abiotic environment or on the effects of the abiotic environment on organisms. However, these effects are often present simultaneously and they form biotic-abiotic feedbacks which can affect population dynamics as well as trait evolution. Using mathematical models we demonstrate that the outcomes of the biotic-abiotic feedbacks depend on whether the abiotic factor is conditionable (like soil pH and temperature) or consumable (like inorganic nutrients and space). With consumable abiotic factors, intraspecific competition is exploitative, increasing resource capture rates leads to larger trait variance, and the equilibrium population size maximizes at an intermediate resource capture rate. With conditionable abiotic factors, there is intraspecific interference competition, and the equilibrium population size and trait variance depend on the initial state of the abiotic environment but not on the strength of conditioning. Our models provide the first steps towards building a theoretical framework for studying the trait basis of biotic-abiotic interactions and their consequences to ecosystems.

3.1 Introduction

Interactions of biological organisms with their abiotic environment is a fundamental process in ecology. For example, the conversion of inorganic matter to organic forms of energy by autotrophs, which is the first trophic level in any ecosystem, plays an important role in ecosystem functioning, resilience, community assembly, and several other ecological processes (Tilman, 1999, Bonanomi *et al.*, 2005, Matthews *et al.*, 2011). The abiotic environment also has an evolutionary significance as it can differentially affect the survival, growth and reproduction of organisms based on their phenotypes (Endler, 1986). Thus biotic-abiotic interactions are intrinsically eco-evolutionary. Characterizing these interactions is essential to the modern field of eco-evolutionary dynamics.

Studies of the effects of organisms on their abiotic environment are common in ecology. Resource consumption and competition emerging from it have been extensively studied over the years in several biological systems empirically and with a wide variety of mathematical models theoretically (Tilman, 1985, Grover, 1997, Svardal *et al.*, 2014, Haney and Siepielski, 2018). Besides consuming resources, organisms can also condition (i.e., change the properties of) the abiotic environment in a non-consumptive manner as in the case of plants conditioning the soil through selectively assembling their root microbial communities (Vitousek *et al.*, 1987, Johnson *et al.*, 2010). This effect is more broadly discussed in the literature on ecosystem engineers (Jones *et al.*, 1997, Odling-Smee *et al.*, 2013). However the ecological and evolutionary consequences of conditioning relative to consumption are not well understood.

Biotic-abiotic interactions can be viewed as one- or bi-directional. For example, changes in a population due to stabilizing environmental selection in a relatively stable environment can be viewed as a one-directional interaction. However if the environment is also changing specifically due to conditioning or consumptive effect of the biological organisms, then we need to study the system as a bidirectional interaction. Bidirectional relationships establish a feedback loop. Figure 3.1 conceptualizes such a feedback loop between a single species and a single abiotic state. In this Figure, the biotic component, which is represented by the abundances $N(x)$ of different phenotypes x in the population, affects their abiotic environment R which in turn affects the dynamics of the abundances. Importantly, the effects of the abiotic environment on the population are not only ecological but also evolutionary because of natural selection on the growth rate.

Community ecologists and ecosystem ecologists have only recently started exploring biotic-abiotic feedbacks with a focus on evolutionary processes (Post and Palkovacs, 2009, Loreau, 2010, Travis *et al.*, 2013, Ware *et al.*, 2019). Such feedbacks can have long-term large-scale consequences by affecting the evolutionary trajectory of organisms and contributing to geological change (Jones *et al.*, 1997, Odling-Smee *et al.*, 2003, Erwin, 2008) as recognized in the niche construction theory (Odling-Smee *et al.*, 2013, Laland *et al.*, 2014). For a better understanding of the feedback, we need an integrative approach including both ecological and evolutionary dynamics, and their effects on the environment which sets up the selection. An eco-evolutionary approach will also help us build a predictive framework which can be

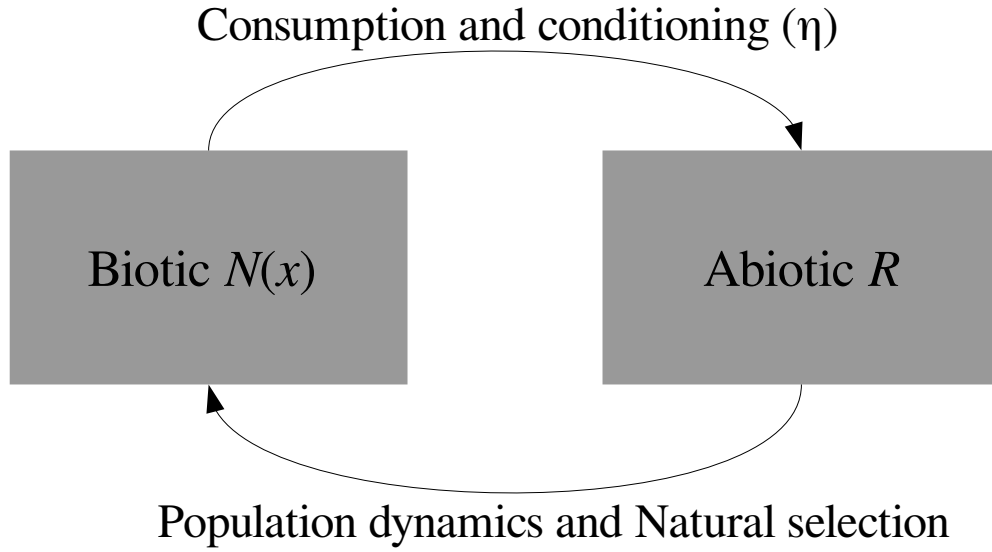


Figure 3.1: Conceptual representation of a simple biotic-abiotic feedback loop. The abundances $N(x)$ of individuals with different trait values x represent the biotic factor, and the abiotic state is represented by a single continuously varying quantity R . The abiotic environment influences population dynamics and natural selection. In turn, individuals affect their abiotic environment through consumption and conditioning. Parameter η represents the resource capture rate in the former case, and the strength of conditioning in the latter case. As the effect of biotic environment on the abiotic environment decreases, the feedback weakens and ultimately disappears. Therefore, more broadly, η represents the strength of the biotic-abiotic feedback.

used to generate hypotheses about the strength of the abiotic-abiotic feedback under different types of abiotic factors.

Intraspecific variation is an essential aspect of the biotic-abiotic interaction and is a part of both directions of the feedback loop. Recent theoretical and empirical studies suggest that phenotypic variation affects ecological dynamics and ecosystem services (Lecerf and Chauvet, 2008, Bolnick *et al.*, 2011, Albert *et al.*, 2011, Des Roches *et al.*, 2018, Senthilnathan and Gavrillets, 2021). Therefore models of biotic-abiotic feedback should explicitly capture the dynamics of intraspecific variation. Existing theoretical approaches to biotic-abiotic interactions based on population genetics (Laland *et al.*, 1999, Odling-Smee *et al.*, 2013), adaptive dynamics and evolutionary game theory (Krakauer *et al.*, 2009, Kylafis and Loreau, 2011) models need to be extended to account for changes in intraspecific variation traits involved in bi-directional biotic-abiotic interactions.

Here we focus on the role of abiotic factors in within-species competition. Competitive interactions can be broadly classified into apparent, exploitative, and interference competition (Gotelli, 2001, Begon *et al.*, 2006). Abiotic factors are downplayed in apparent competition which can be caused by a shared predator or parasite. In contrast, exploitative and interference competition can be due to both biotic and abiotic factors. Exploitative competition is the interaction between two species when they compete for a shared common resource which can be a common prey or abiotic consumables like inorganic nitrogen and minerals. Interference competition occurs when organisms hinder the survival and reproductive ability of other individuals due to non-trophic interactions. Interference competition typically involves abiotic conditionable factors such as soil pH and geological features. The differences in eco-evolutionary dynamics between exploitative and interference competition are not well studied.

In this paper we study biotic-abiotic interactions as an eco-evolutionary feedback using a mathematical framework similar to that in Roughgarden (1972), Slatkin (1979), Doebeli (2011), Senthilnathan and Gavrillets (2021). We consider exploitative and interference competition separately. For each case, we first build a population dynamics model to demonstrate ecological effects of the biotic-abiotic feedback. Then, we extend the population dynamics models to describe asexually reproducing species varying in a continuously

quantitative trait. We use our models to characterize the effects of the nature of abiotic factor, the strength of selection, and the strength of the feedback on phenotypic variance and population sizes.

3.2 Models and Results

3.2.1 Consumable abiotic factor and exploitative competition

Biotic-abiotic interactions involving a consumable abiotic factor have a lot of similarities with biotic-biotic exploiter-victim interactions. This allows us to utilize the rich literature on modeling ecological dynamics (Kot, 2001, Turchin, 2003). Specifically we will build on the MacArthur consumer-resource model (MacArthur, 1970, Chesson, 1990).

Ecological model

Adapting the MacArthur consumer-resource model, we consider a single resource with density $R(t)$ and two consumer species with densities $N_1(t)$ and $N_2(t)$. We assume that the resource replenishment follows a standard logistic model. The resource consumption rate f nonlinearly depends on the consumer and resource abundances. We also assume that the consumer growth rate depends on the rate of consumption while its death rate is constant. Our model is described by the following differential equations

$$\frac{dN_i}{dt} = \eta\omega f(N_i, R)N_iR - \mu_i N_i, \quad (3.1a)$$

$$\frac{dR}{dt} = \gamma R \left(1 - \frac{R}{K}\right) - \eta R (f(N_1, R)N_1 + f(N_2, R)N_2). \quad (3.1b)$$

Here the resource capture rate η characterizes the impact of the biotic-abiotic interaction on the resource, ω is conversion efficiency, μ_i is the species-specific per-capita mortality rate, and γ and K are the intrinsic growth rate and carrying capacity of the resource. The function $f(N, R)$ represents the “Beddington” functional response in which per-capita consumption

decreases with increasing consumer and resource abundance:

$$f(N, R) = \frac{\rho}{\alpha + R + \delta N}, \quad (3.2)$$

where parameters ρ , α , and δ represents the maximum consumption, the saturation constant, and the relative effect of consumer density-dependence.

Although Holling Type-II functional response is commonly used to model the dependence of consumption rate f on resource abundance R , we choose a more general “Beddington” functional response which also accounts for the dependence on consumer abundance N . The Beddington functional response has been mechanistically derived by considering handling times in predator-prey interactions (Huisman and De Boer, 1997) and using the optimal foraging theory with a sessile prey (Anderson, 2010). This choice of functional response is also supported empirically (Skalski and Gilliam, 2001, Novak *et al.*, 2017).

Single consumer. First, assume there is only one consumer. Then in our model, there are four equilibria out of which three are biologically feasible (non-negative): (i) both the consumer and the resource are extinct, (ii) the consumer is extinct and the resource is at the carrying capacity K , and (iii) both the consumer and the resource persist. The first equilibrium is never stable for finite resource capture rates, and the second is stable only when consumer mortality is large enough (specifically, if $\mu \geq \frac{K\eta\omega\rho}{K+\alpha}$). If consumer mortality is small ($\mu < \frac{K\eta\omega\rho}{K+\alpha}$), both the consumer and resource persist. The resource density at the coexistence equilibrium is inversely proportional to the resource capture rate η . The equilibrium consumer population reaches a maximum at an intermediate value of the resource capture rate: $\hat{\eta} = \frac{(K+2\alpha)\mu}{K\rho\omega} + \frac{\gamma\delta}{2\rho}$. Both the consumer and resource become extinct asymptotically as the resource capture rate η becomes very large. See SM section 1.8 for the details.

Two consumers. The full model with both consumers present has five equilibria. In one of these, both the consumers and the resource are extinct; this equilibrium is always unstable. At the four other equilibria, the resource is always present while each consumer can be either extinct or present. Similar to the single species case, a sufficient condition for

consumer i to become extinct is high enough mortality: $\mu_i \geq \frac{K\eta\omega\rho}{K+\alpha}$. A necessary condition for coexistence is low mortality: $\mu_i < \frac{K\eta\omega\rho}{K+\alpha}$ for both species.

To determine the region of stability for different equilibria, we used a combination of symbolic calculations and numerical evaluations. Figure 3.2a shows that the region of stability of all the four equilibria are mutually exclusive and exhaustive so that there is exactly one stable equilibrium for each combination of parameters. Figure 3.2a also shows that $\mu_i < \frac{K\eta\omega\rho}{K+\alpha}$ is not a sufficient condition for coexistence. Moreover, the coexistence equilibrium (which we find exactly in the SM section 1.1) has a slightly larger feasibility region than the stability region. Both these regions are strictly contained in the rectangle determined by the necessary condition. At the coexistence equilibrium, the consumer populations initially increases with increasing resource capture rate, η , but subsequently starts decreasing due to an inverse relationship between resource capture rate and the resource. (see Figure 3.2b). The equilibrium values in Figure 3.2b were evaluated using an analytical expression we derived for the case of coexistence. Naturally negative population densities are not biologically feasible. But we include them in the graph to show that coexistence is not feasible for very small and large resource capture rates.

Eco-evolutionary model

In an evolutionary interpretation, the two consumer species in the previous section can be viewed as two discrete morphs of a single asexual species. Here we generalize this model for a case where a single asexual consumer species exhibits continuous genetic variation in an ecologically important trait x . In the resulting model, the dynamics of resource R depend on its previous state and its relationship to the biological entities therein as captured by the notion of environmental inheritance (Odling-Smee *et al.*, 2013, Cuddington, 2011). As in the ecological consumer-resource model, we assume that the consumption is density-dependent and model it using the “Beddington” functional response (Huisman and De Boer, 1997).

The dynamics of the consumer with population density N for trait x , and resource amount R are

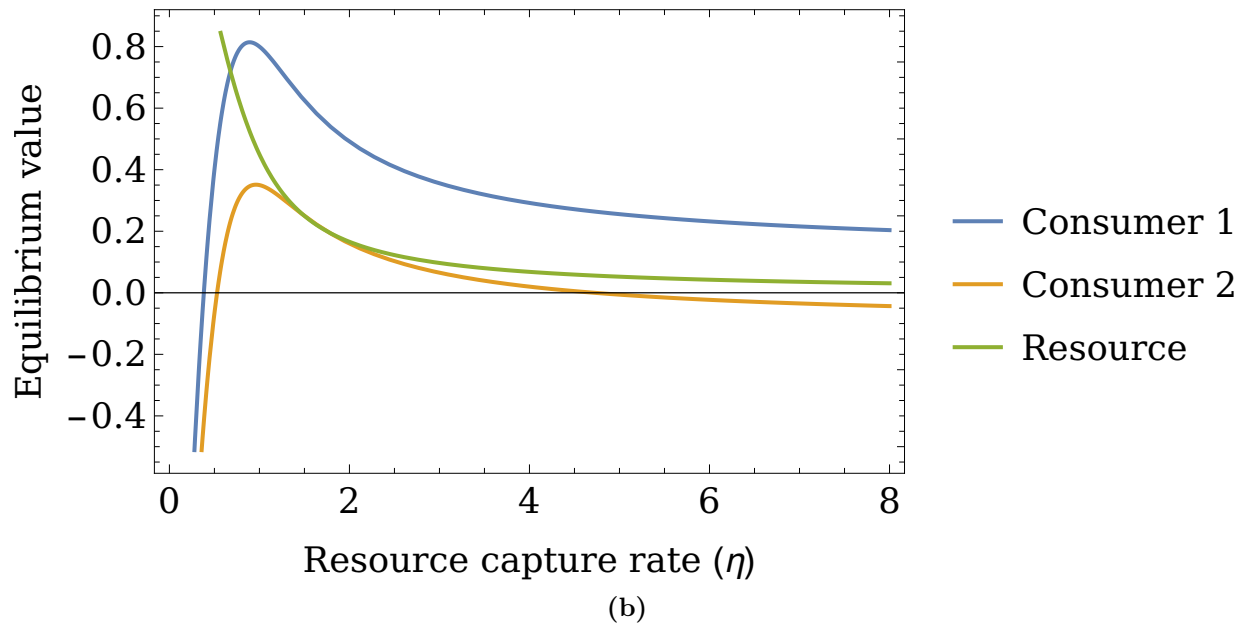
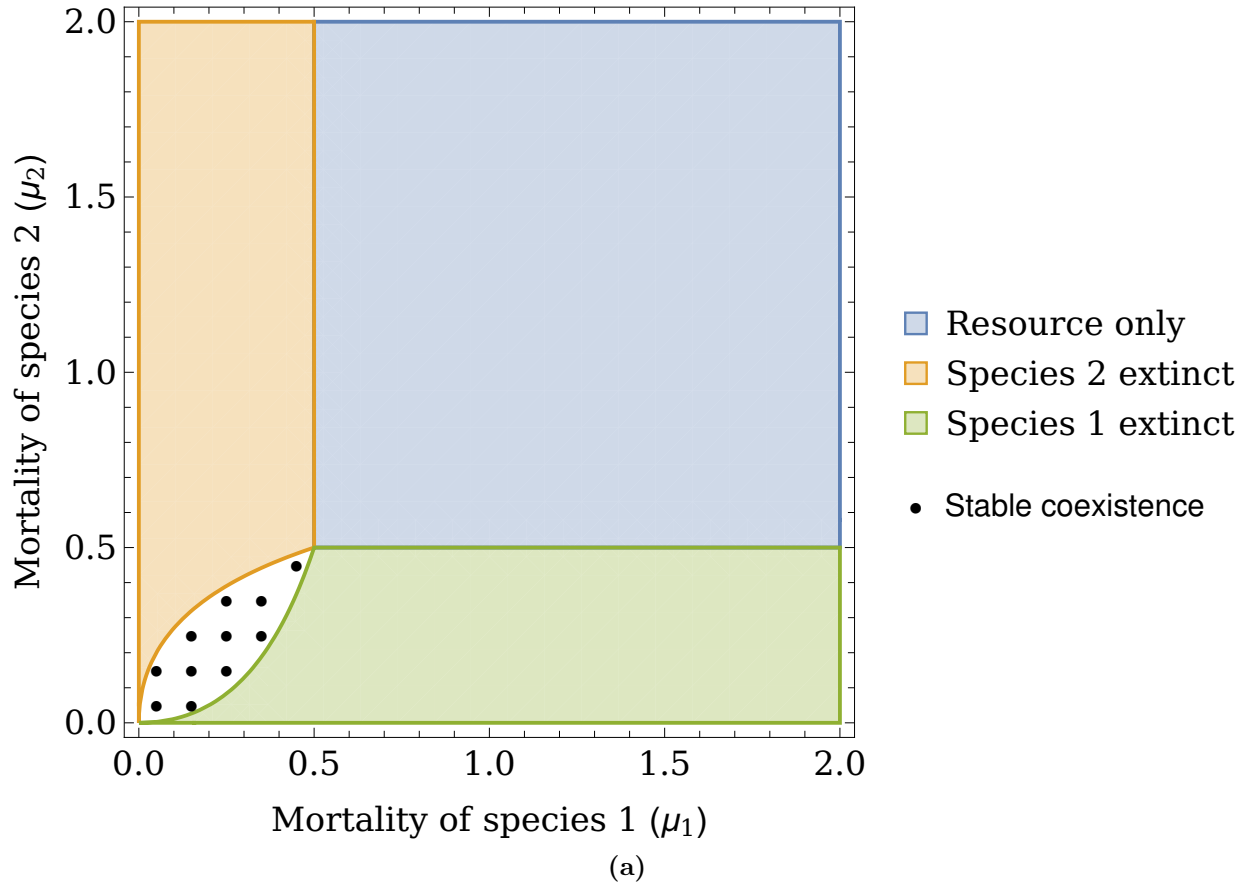


Figure 3.2: (a) Regions of stability of the four equilibria. (b) Effects of the resource capture rate η on the consumer populations N_1, N_2 and resource R at the coexistence equilibrium. Parameters: $\omega = 1, \gamma = 1, K = 1, \alpha = 1, \delta = 1, \rho = 1$, (a) $\eta = 1$ and (b) $\mu_1 = 0.2, \mu_2 = 0.25$.

$$\frac{\partial N(x, t)}{\partial t} = \eta\omega f(x)N(x, t)R(t) - m(x)N(x, t), \quad (3.3a)$$

$$\frac{dR(t)}{dt} = \gamma R(t) \left(1 - \frac{R(t)}{K}\right) - \eta R(t) \int f(x)N(x, t)dx. \quad (3.3b)$$

Here η, ω and K are constant parameters representing, as before, the resource capture rate, the number of births per unit resource consumed (conversion efficiency), and the carrying capacity of the resource respectively. Consumers die at the rate $m(x)$ which depends on their trait x . The functional response is given by equation (3.2) with an appropriate interpretation of N .

Resource consumption by individuals contributes to the growth rate and establishes a biotic-abiotic feedback. Below we start by summarizing the results for two special values of the maximum consumption rate, $\rho = 0$ and ∞ . Then we present some analytical and numerical results for the case of quadratic mortality function $m(x)$.

First, if there is no consumption ($\rho = 0$), it is clear that the consumer will be extinct while the resource will be at carrying capacity K . On the other extreme if there is no limit on consumption ($\rho \rightarrow \infty$), then consumption becomes independent of population density. In this case, only the phenotype with the smallest mortality survives while all other phenotypes are extinct at equilibrium.

Second, assume that the resource dynamics are fast ($\gamma \rightarrow \infty$) which is the standard quasi-equilibrium assumption in the analysis of ecological models of consumer-resource dynamics (MacArthur, 1970, Chesson, 1990). In this case the dynamics of the resource and consumer are decoupled and the consumer population does not change at the timescale of resource dynamics. So, we can analyze consumer dynamics at a slower timescale where the resource is always at equilibrium.

We choose the mortality function to be quadratic, $m(x) = \mu(x^2 + 1)$. This choice ensures that the population density of phenotypes at tails of the trait distributions is not increasing. In this case, there is exactly one equilibrium where the consumer persists. This equilibrium

consumer trait distribution is

$$N^*(x) = \max\left(\frac{1}{\delta} \left(\frac{K\eta\omega\rho}{\mu(x^2 + 1)} - (K + \alpha) \right), 0\right). \quad (3.4)$$

Note that the range of observed traits values in this distribution is finite: $N^*(x) > 0$ only for x values close to zero (specifically for $|x| < v_r/2 = \sqrt{\frac{K\eta\omega\rho}{\mu(K+\alpha)} - 1}$) and requires mortality to be small enough (specifically $\mu < \frac{K\eta\omega\rho}{K+\alpha}$). The last condition is also a necessary and sufficient condition for consumer persistence. For the rest of this section, we will use the trait range v_r as a measure of trait variation instead of statistical variance.

To analyze our model in the general case, we use a numerical method developed in [Senthilnathan and Gavrilets \(2021\)](#) for related eco-evolutionary models. Our numerical results show that the system reaches an equilibrium at which the consumer trait distribution resembles the one given by equation (3.4). Figure 3.3a shows such an equilibrium trait distribution of the consumer population when consumers persist. The equilibrium consumer population is hump-shaped, reaches a maximum at an intermediate resource capture rate $\hat{\eta}$, and vanishes outside a finite range similar to the ecological model. Importantly, there is a difference between values of η maximizing consumer density between the ecological and eco-evolutionary models as illustrated in Figure 3.3b. This can be explained by the effect of mortality rate on the trait range and population size in the presence of intraspecific variation. Lower mortality μ leads to larger trait range v_r , and higher mortality leads to smaller population sizes relative to the analytical results of the ecological model. Therefore η is larger in the eco-evolutionary model when mortality rates are low and, η is larger in the ecological model when mortality rates are high.

3.2.2 Conditioning abiotic factor and interference competition

In this section, we focus on biotic-abiotic interactions mediated by a conditionable abiotic factor. The conditioning of the abiotic environment by individuals to benefit themselves while harming others will represent interference competition. As above, we first consider an ecological model with two species and then generalize it to an eco-evolutionary model of a single species with intraspecific variation.

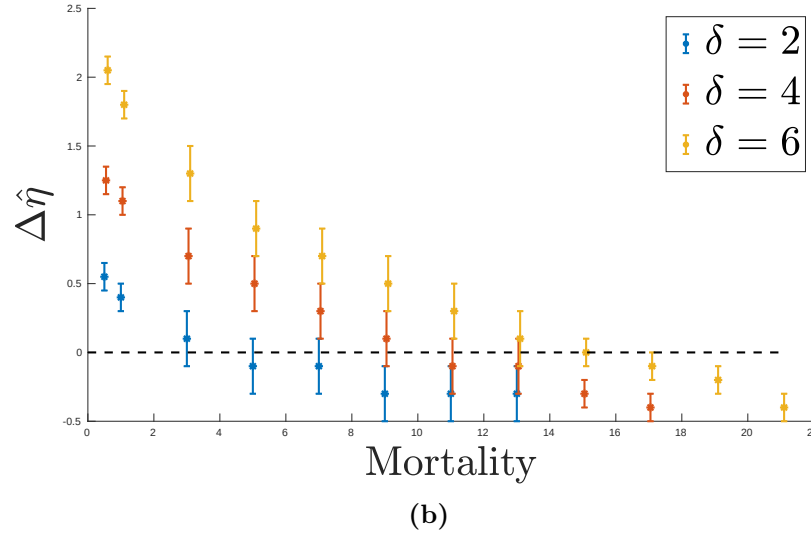
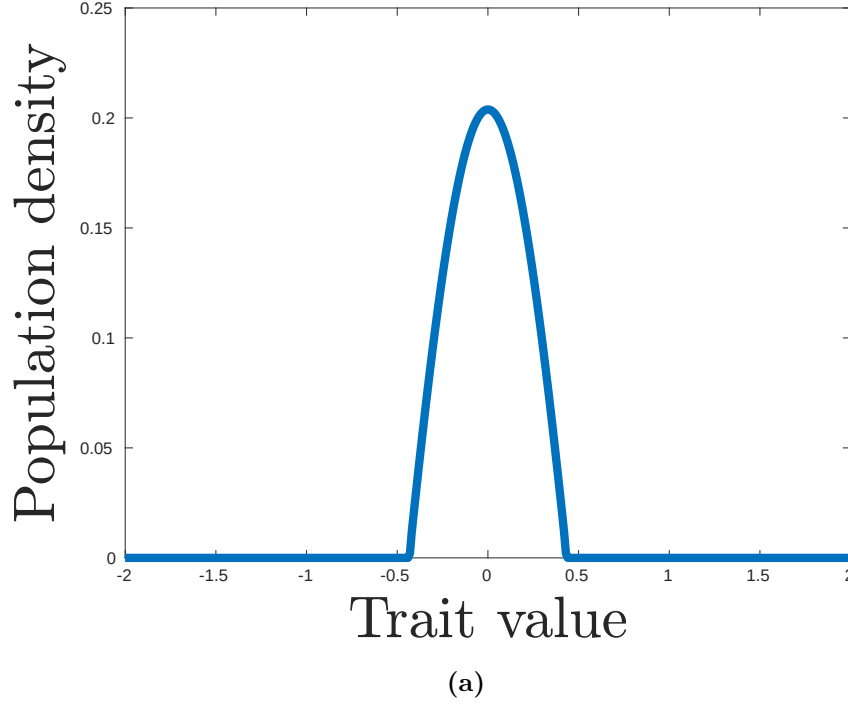


Figure 3.3: Summary of results for the model of biotic-abiotic feedback with a consumable abiotic factor. (a) Equilibrium trait distribution is unimodal centered at the trait with least mortality rate and has a finite range of traits with positive population density. (b) The difference between the resource capture rate at which the consumer population in the eco-evolutionary model and the ecological model, $\Delta\hat{\eta}$, decreases as the mortality rates become larger. Parameters: $K = 1, \omega = 2, \rho = 1, \alpha = 1$, (a) $\delta = 1, \eta = 5, \mu = 0.6$. Initial trait distribution and resources are $N(x, 0) = 0.1\sqrt{\frac{50}{\pi}}e^{-50x^2}$, $R(0) = 1$ respectively.

Ecological model

Consider a single abiotic factor whose value E determines the carrying capacity of the two species with population densities N_1 and N_2 . Let the optimum states of E for the two species be at E_1 and E_2 , respectively. Assume that the two species modify their environment so that they have a higher carrying capacity. An example would be plants conditioning their soil microbial community (Vitousek *et al.*, 1987, Johnson *et al.*, 2010, Van Nuland *et al.*, 2017). Let the effects of conditioning by a species be directly proportional to how far away the current environment is from the species' optimum. We describe the dynamics of such a community by the differential equations,

$$\frac{dN_i}{dt} = \beta N_i \left(1 - \frac{N_i}{\kappa_i(E)} \right), \quad (3.5a)$$

$$\frac{dE}{dt} = \eta ((E_1 - E)N_1 + (E_2 - E)N_2). \quad (3.5b)$$

where parameter η measures the strength of the conditioning. Parameter β in equation (3.5a) is a constant population growth rate while the carrying capacity κ_i of each species depends on the current abiotic state E . Specifically, we assume that the carrying capacity decreases from a maximum value K with the deviation from the corresponding optimum and write

$$\kappa_i(E) = K \exp \left(-\frac{(E_i - E)^2}{2\sigma^2} \right), \quad (3.6)$$

where the viability range σ measures the effect of the abiotic factor on carrying capacity. Small viability range σ means that the population can have large carrying capacity only if the environment E is close to the species' optimum E_i .

We show in the SM (section 2.1), that this model has five isolated equilibrium points and a line of equilibria where the two species are extinct while the abiotic state E can be an arbitrary number. The line of equilibria and the two equilibria with only one consumer species present are all unstable. The three equilibria with both species present differ with respect to the population sizes. A symmetric equilibrium with equal population sizes and the abiotic state at $E = 0$ is stable if and only if the difference between the optimum abiotic states is sufficiently small (precisely if $|E_1 - E_2| \leq 2\sigma$). Otherwise, depending on initial

conditions, the system evolves to one of the two asymmetric equilibria, at which one species has a higher density and “shifts” the abiotic state closer to its own optimum (R_1 or R_2). Figure 3.4 illustrates these results.

Eco-evolutionary model

In this section, we extend equations (3.5) from two species to a single asexually reproducing species with a phenotype which determines its optimal abiotic state (analogous to E_i in equations (3.5)). We continue to assume that each individual contributes to the dynamics of the abiotic state by shifting it closer to their phenotype. This establishes a density-dependent dynamics for the abiotic state. The dynamics of the population density N for trait x , and the abiotic state E is

$$\frac{\partial N(x, t)}{\partial t} = \beta N(x, t) \left(1 - \frac{N(x, t)}{\kappa(E, x)} \right) \quad (3.7a)$$

$$\frac{dE(t)}{dt} = \eta \int (x - E(t)) N(x, t) dx \quad (3.7b)$$

We continue to assume that the carrying capacity is a Gaussian function: $\kappa(E, x) = K e^{-\frac{(x-E)^2}{2\sigma^2}}$. This can be interpreted as stabilizing selection on trait x due to the abiotic environment with the viability range σ being inversely related to the strength of selection. Parameters β and σ are the intrinsic growth rate and the viability range, as above. The abiotic dynamics are population density-dependent as well as trait-dependent. Biotic-abiotic feedback is established by each individual modifying the abiotic environment to increase its carrying capacity. The strength of abiotic conditioning is determined by the parameter η . The abiotic state E does not change if there is no biotic-abiotic feedback ($\eta = 0$).

Expanding the integral in equation (3.7b) shows that at any equilibrium the mean trait will be equal to the abiotic state. Moreover, the carrying capacity function is an equilibrium for the trait distribution from equation (3.7). We can conclude that whenever $\eta = 0$ or if equilibrium is reached, the equilibrium trait distribution matches the carrying capacity function: $N^*(x) = \kappa(E^*, x)$ with some mean at E^* and variance equal to the viability range σ .

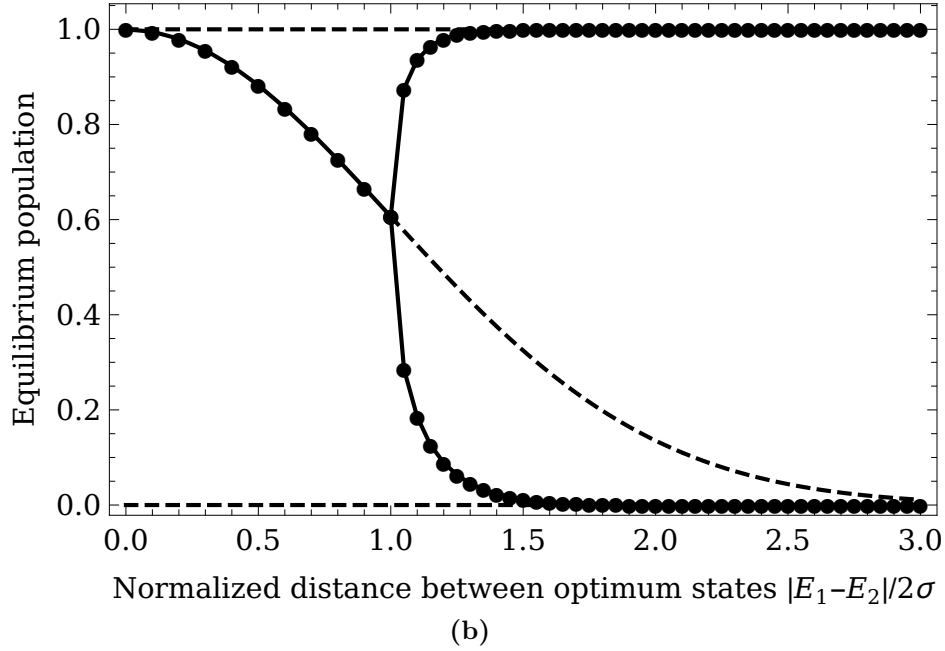
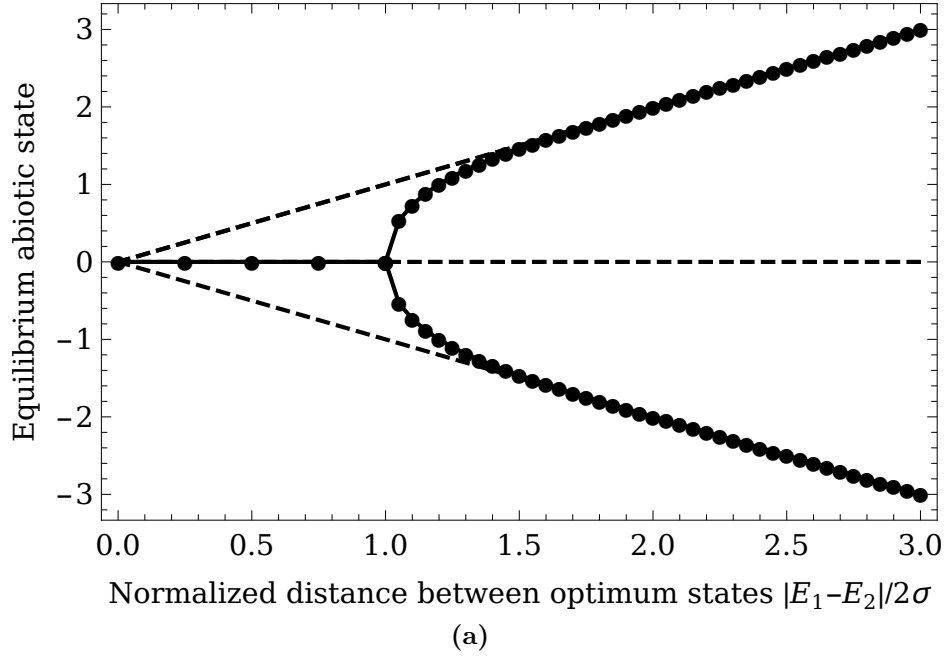


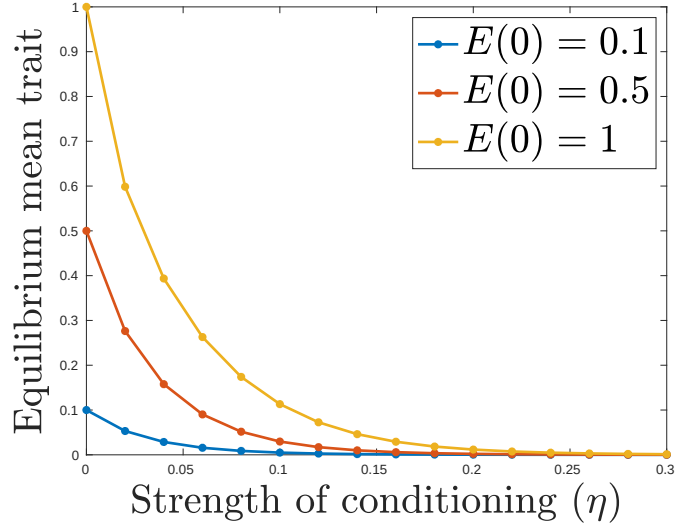
Figure 3.4: Bifurcation diagrams for (a) the abiotic state E and (b) the population density N in the model (3.5). The solid curves with dots represent the stable equilibria and the dashed curves represent unstable equilibria. In (b), both the species have the same population when $|E_1 - E_2|/(2\sigma) \leq 1$. When $|E_1 - E_2|/(2\sigma) > 1$, one of the species is on the upper branch and the other is on the lower branch depending on the initial conditions. This determines whether the abiotic value E is on the upper branch or the lower branch in (a). For example, if species 1 has the higher population, then the abiotic value is closer to E_1 .

Although these analytical results give us a trait distribution and the population size at equilibrium, they do not offer any insights about the stability of the equilibrium. We also cannot determine the values at which x^* and E^* equilibrate. Therefore we are forced to use numerical analysis. Using a method (similar to the consumer-resource case), we find that the system reaches an equilibrium for a wide range of parameters. Figure 3.5 shows the effect of the strength of abiotic conditioning, the viability range, and the initial abiotic value on the equilibrium mean trait. As expected, the abiotic state is shifted less from its initial value when the strength of abiotic conditioning is low (Figure 3.5a). Increasing the viability range increases the shift of the abiotic state from its initial value (Figure 3.5b). The abiotic state shifts least when the initial mean biotic trait and abiotic state are the farthest apart.

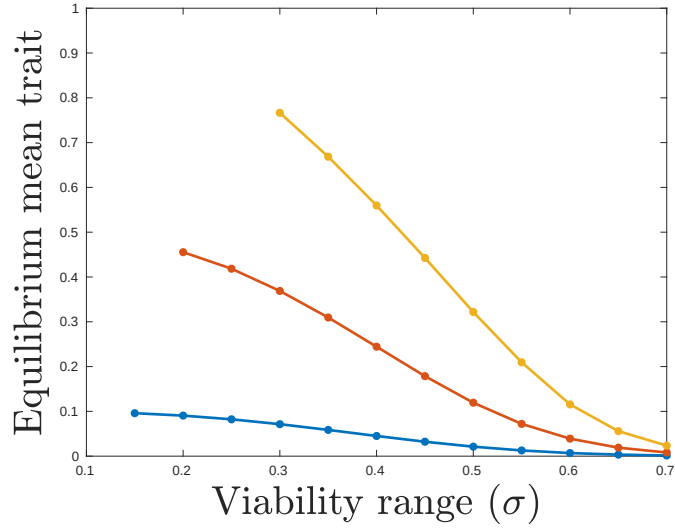
Overall, Figure 3.5 shows that the equilibrium mean trait and the abiotic state match the initial mean trait value regardless of the initial abiotic state when the strength of abiotic conditioning or the viability range is large. The latter means that when stabilizing selection is strong, the trait distribution shifts more from its initial condition in comparison to the abiotic state. This happens because the abiotic dynamics are population density-dependent, and equilibrium population size is smaller when selection is stronger (small σ). The trait variance also decreases with stronger stabilizing selection. The numerical solutions also show that there is no effect of the initial population size and trait variance on the equilibrium mean trait for a wide range of parameters. The equilibrium trait distribution is $\kappa(E^*, x)$ regardless on the initial trait distribution.

3.3 Discussion

Biotic-abiotic interactions are part of eco-evolutionary feedbacks which form the foundation of ecosystems. Here we aimed to understand better these interactions using mathematical models. Abiotic factors and resources can be categorized as consumable and conditionable. Consumable resources decrease in quantity due to consumption by organisms (e.g., nutrients, water, and habitable space). Organisms also interact with the environment by changing and conditioning it in a non-consumptive manner. We refer to such abiotic factors as conditionable. These include microclimate, soil pH, and other properties of the abiotic



(a)



(b)

Figure 3.5: The effect of the strength of conditioning and the viability range on the equilibrium mean trait value. These results were obtained from numerical solutions of the system of ordinary differential equations derived using a Gaussian approximation of the trait distribution. For all the simulations, the initial mean trait, trait variance, and population size were fixed at 0, 0.1, and 1 respectively. Other parameters: $\beta = K = 1$, (a) $\sigma = 0.5$, (b) $\eta = 0.05$.

environment which can be modified. We built and studied ecological and eco-evolutionary models to highlight the differences between biotic-abiotic feedback which involve consumable or conditionable factors.

For each type of the feedback, we first analyzed ecological models based on the population dynamics of two species with no intraspecific variation and a single abiotic factor. Then, we extended the ecological models from the two species to a single species with a continuously varying phenotype. When the abiotic factor is consumable, the phenotypic trait controls mortality rate while the rate of consumption depends on the population density of consumers with the same phenotype. When the abiotic factor is conditionable, the carrying capacity of each phenotype depends on how closely the abiotic state matches the phenotype. In this case, each individual conditions the environment in such a way that the abiotic factor shifts closer to its phenotype.

We found that the effect of the strength of biotic-abiotic feedback η on the equilibrium population size does not change qualitatively between the ecological and the corresponding eco-evolutionary models. However, the relationship between the strength of the feedback and equilibrium population size depends on the type of the abiotic factor. When the abiotic factor is consumable, the equilibrium population size initially increases with the consumption rate (η) and then decreases for large rates of consumption. When the abiotic factor is conditionable, the equilibrium population size does not depend on the strength of conditioning (η). In the eco-evolutionary model with a conditionable abiotic factor, the equilibrium trait distribution is always symmetric. But in the ecological model, the equilibrium can be asymmetric in the population of the two species when conditioning is sufficiently weak ($\sigma < |E_1 - E_2|/2$).

Specifically, there are two stable equilibria where one species has a larger population size than the other. Which species has the larger population size depends on the initial population sizes and abiotic state. Moreover as shown in Figure 3.5a, when conditioning is weak, the equilibrium mean trait which is equal to the abiotic state at equilibrium depends on the initial abiotic state. Such dependence on initial conditions might provide an explanation for the differences between similar ecosystems (dominated by the same species) at different geographical regions (Cuddington, 2011, Bayliss *et al.*, 2020, Crawford and Hawkes, 2020).

The effect of biotic-abiotic feedback on the equilibrium mean trait and the trait variation depends on the type of abiotic factor. When the abiotic factor is consumable, individuals outside a finite range v_r of trait values cannot survive. The trait range v_r increases with increasing consumption rate (η) which implies that exploitative competition has a diversifying effect on the consumers. In contrast, there is no effect of the strength of conditioning η on the equilibrium trait variance. The equilibrium mean trait is the trait with the smallest mortality when the abiotic factor is consumable, and it does not depend on the consumption rate. In the model of conditionable abiotic factor, the equilibrium mean trait depends on the initial difference between the mean trait and the abiotic state. As expected intuitively, the equilibrium mean trait is closer to the initial mean trait when the conditioning is strong, or closer to the initial abiotic state when conditioning is weak.

Our study of the biotic-abiotic feedbacks is closely related to the concept of the organism-environment feedback (Lewontin, 2000) which has developed into the niche construction theory (Odling-Smee *et al.*, 2013, Laland *et al.*, 2014) over the years. Two key concepts underlying the niche construction theory are (i) environmental inheritance and (ii) modification of the evolutionary niche. The former concept captures the idea that features of the environment are “inherited” through time analogously to biological inheritance of genes and traits of species living in that environment. By explicitly tracking the dynamics of the abiotic factor through time, our eco-evolutionary models account for environmental inheritance. The concept of the evolutionary niche of a species describes the collection of selection pressures it is under. The species in our eco-evolutionary models modify the selection pressure they are subject to through consumption or conditioning. In the case of consumption, the selection pressure is not modified to directly benefit the species. Therefore, the eco-evolutionary model with a consumable abiotic factor is a model of niche construction only under a broader definition which does not require a beneficial modification of the evolutionary niche (Post and Palkovacs, 2009, Odling-Smee *et al.*, 2013, Laland *et al.*, 2014). In the model with a conditionable abiotic factor, the modification is directly beneficial, and hence it is a model of niche construction according to a narrower definition (Fussmann *et al.*, 2007, Erwin, 2008, Krakauer *et al.*, 2009).

The models we have built here are also representative of two types of intraspecific competition. Exploitative competition is based on a shared resource whose consumption by one excludes it for the other individuals. Our models of biotic-abiotic feedback with a consumable abiotic factor can be viewed as describing exploitative competition. Interference competition is a broader class of competition where individuals affect the others' survival directly by mechanisms other than exploiting a shared resource. Our models of biotic-abiotic feedback with a conditionable abiotic factor can be viewed as describing interference competition.

Existing mathematical models of biotic-abiotic feedback can be broadly divided into three categories. The largest category (Ackermann and Doebeli, 2004, Fox and Vasseur, 2008, Svardal *et al.*, 2014, Haney and Siepielski, 2018) are the consumer-resource models whose ecological dynamics are based on the MacArthur model (MacArthur, 1970) similar to that in our approach. To model evolution, these models use the adaptive dynamics framework (Geritz *et al.*, 1998) or rely on individual-based simulations. The adaptive dynamics models assume fast resource dynamics and ignores intraspecific variation in the consumer. The individual-based simulations suffer from the lack of analytical treatment and computational complexity. Among the consumer-resource models, Ackermann and Doebeli (2004) is the closest to our eco-evolutionary model. The key difference is that Ackermann and Doebeli (2004) assume that there is a continuous variation in the resource and that consumption is based on how closely the resource “trait” matches the consumer trait. They found that the consumer can undergo adaptive speciation (i.e. evolve a bimodal trait distribution) or evolve to state with increased trait variance depending on the details of resource replenishment and trait-based consumption rates. In contrast, our model never leads to bimodality in the trait distribution. However, trait range increases with consumption rate.

Second, there are models of ecosystem engineering and niche construction. Models of ecosystem engineering typically only capture population dynamics and do not include any evolutionary processes (Gurney and Lawton, 1996, Vandermeer, 2008). Niche construction theory is built upon existing evolutionary theory, therefore it naturally includes evolutionary processes. Laland *et al.* (1999) built one of the early models of niche construction based on population genetics of two diallelic loci. They demonstrated that niche construction

can modify evolutionary trajectories and even overcome external selection pressures. Their model did not include population dynamics and density-dependent processes.

Instead of using the population genetics approach, [Kylafis and Loreau \(2008\)](#) and [Krakauer *et al.* \(2009\)](#) use the adaptive dynamics approach with a consumer-resource model as the underlying ecological model. Unlike other consumer-resource models, they explicitly include modification of an ecological niche through processes other than consumption. One of the main goals of their models was to determine whether niche construction was a stable strategy. In contrast, in our eco-evolutionary models niche construction is not a trait but a process.

Third, there are few models which include conditioning of the abiotic environment in a non-consumptive manner. [Bonanomi *et al.* \(2005\)](#) uses a simulation model where plants of different species occupy and condition discrete sites on a landscape. Conditioning results in that conspecifics have a lower germination probability compared to heterospecifics in a site previously occupied by a plant which is now dead. They find that such negative niche construction promotes species coexistence. This is in contrast to our ecological model with a conditionable abiotic factor where conditioning is beneficial to conspecifics. In our model, both species always persist. However, the population size of one of the species is close to zero when the strength of conditioning is small.

[Jiang and DeAngelis \(2013\)](#) also study a simulation model where species condition the site they inhabit. Conditioning in their model is similar to that in our models with species shifting the environment to be closer to their optimum. But in [Jiang and DeAngelis \(2013\)](#) conditioning is not density-dependent since they allow only for one individual to occupy a site at a time. As individuals perish, they are replaced by offspring of the species already present in the landscape through lottery competition or an immigration event. They find that strong conditioning increases species richness when there is immigration but the trend reverses in the absence of immigration. In our eco-evolutionary model, the strength of conditioning has no effect on phenotypic variance.

The differences in the biotic-abiotic feedback between consumable and conditionable abiotic factors that we have identified have direct implications for biomass and functional traits in ecosystem ecology. Our approach provides a template for building and analyzing

mathematical models which could be tailored to specific ecosystems with different sets of consumable and conditionable abiotic factors. These models can then be used to generate hypotheses regarding the effects of natural selection, strength of consumption and conditioning on population sizes (biomass) and functional traits. For example, our models can be used as a theoretical framework to study the abiotic side of plant-soil feedbacks (Van der Putten *et al.*, 2013). Plant-soil feedbacks could be positive or negative depending on their net effect on plant abundance or fitness. However, in the models we studied negative feedback is not possible. The feedback is positive in the case of abiotic resource consumption and the feedback has no effect on population size in the case of abiotic conditioning. Therefore our results imply that negative plant-soil feedback requires plant-microbe interaction or other complex biotic-abiotic interaction that our models do not represent.

The main limitation to our approach is the lack of intraspecific density-dependence in the eco-evolutionary models. In the model with a consumable abiotic factor (equations 3.3), consumption is independent of direct intraspecific effects. Specifically consumption is affected only by the population density of individuals with the same phenotype. This requires the assumption that intraspecific consumer interactions do not significantly affect consumption. In the model with a conditionable abiotic factor (equations 3.7), intraspecific density dependence is absent from the process of stabilizing natural selection and the per-capita growth rate is affected only by individuals with the same phenotype. Here, we again assumed that direct intraspecific interactions were restricted to individuals with the same phenotype.

Other limitations are related to specific modeling choices. The choice of “Beddington functional response” makes our consumer-resource ecological model unstable when the saturation constant (α), the relative effect of consumer density-dependence (δ) and mortality (μ) are all small. This is not surprising since small α and δ leads to an unrealistic situation where consumption is independent of the amount of resource. In the eco-evolutionary model with a conditionable abiotic factor, we assume that the phenotype only determines carrying capacity and the conditioning effect. However, Rueffler *et al.* (2006) show that carrying capacity and the growth rate are correlated when considering the underlying individual-based processes. This correlation could potentially affect the dynamics of conditioning and

hence change the equilibrium trait distribution. Future studies with explicit modeling of the individual-level processes will be required to understand the effect of the correlation between growth rate and carrying capacity on biotic-abiotic feedbacks.

[Yamamichi *et al.* \(2020\)](#) introduces the concept of intraspecific adaptation load which refers to the scenario where a species adapts to conspecific conflicts which subsequently has a negative effect on population growth. They suggest that intraspecific adaptation load might promote species coexistence. Our consumer-resource eco-evolutionary model describes intraspecific adaptation to resource competition. An extension to our model to two consumer species will offer further insights into the effect of intraspecific variation on species coexistence. Overall, we have provided a theoretical framework to understand the feedbacks between the biotic and abiotic components which are at the core of any ecosystem. Our models will contribute to a broader goal of integrating ecology and evolution to understand the processes which drive energy flows in ecosystem and determine community compositions ([Post and Palkovacs, 2009](#), [Loreau, 2010](#), [McPeck, 2017](#), [Lion, 2018](#), [Ware *et al.*, 2019](#)).

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Chapter 4

Body sizes and competition for space

Publication statement

This chapter is a reprint of a paper that will be submitted for publication:

“Senthilnathan, A. (2021). Smaller is better in competition for space.”

Abstract

Body size is a prominent morphological trait which affects many aspects of an organism's life. Although large body size is considered to be advantageous in many ecological scenarios and for a variety of reasons, ecologists have wondered about the benefits of being small. Many studies on body size depend on the metabolic theory of ecology since body size is irremovably part of an organism's energy budgets. Body size is also a spatial quantity and therefore irremovably linked to spatial processes. In this paper, I show that competition for space imposes a benefit to being small and hence selects for increasingly smaller body size. I build a deterministic population dynamics model and a stochastic model of birth, death and dispersal of a population of individuals with two different body sizes and show that only the smaller individuals survive. I also extend the population dynamics model to continuously varying body sizes and include a stabilizing natural selection for an intermediate body size. I find that the intrinsic preference for smaller body size in competition for space can only be overcome when natural selection is sufficiently strong for a large body size. Overall, my results point to a novel benefit to being small.

4.1 Introduction

Ecological processes that underlie the costs and benefits of having a large body size plays a fundamental role in understanding species interactions (Persson, 1985). The space that an individual organism utilizes is directly tied to its body size. Therefore, understanding ecological and evolutionary processes that influences body sizes offers a unifying perspective. Moreover, body size variation plays an important role in determining energy flows in ecosystems (Jennings *et al.*, 2002, Le Roux *et al.*, 2020). Although we have an extensive understanding of the proximate cause of body size variation, we are still working towards an eco-evolutionary understanding.

Evolutionary ecologists have been working on understanding the ecological processes which benefit small body sizes. These are primarily related to the costs of maintaining a large body size (Blanckenhorn, 2000). Larger individuals take longer time to reach maturity

and reproduce (Blanckenhorn, 2000). Competition studies show that despite having a higher feeding ability, larger individuals are inferior in exploitative competition (Persson, 1985, Van Buskirk *et al.*, 2017)). However, large body size is known to be advantageous in interference competition (Griffiths *et al.*, 2020).

The ubiquity of body size in sexual dimorphism makes it one of main traits of concern in studies of sexual selection and conflict (Andersson and Iwasa, 1996). Body size is also at the center of the metabolic theory of ecology which focuses on explaining ecological organization through flows of energy as well as matter (Brown *et al.*, 2004). This bioenergetic approach is also the basis of many mathematical models which are built to explain patterns of body size distribution (Uchmański, 1985, Pawar, 2015, Lee *et al.*, 2021). The importance of body size distribution and the consistency in the patterns we find in nature has led to many candidate mechanistic explanations (Blackburn and Gaston, 1999, Belgrano and Reiss, 2011). However none of them involves explicit competition for space.

Body size of organism are necessarily linked to the space they occupy. Empirical studies on space is skewed towards studying the movement and dispersal of an organism. While there are investigations of the role of body size in animal movement (Schmidt-Nielsen, 1984), there is no clear baseline expectation of the role of body size in competition for space. Understanding this role of body size is important for community and ecosystem ecology since size spectrum of species predicts community membership and energy flows in ecosystems (Jennings *et al.*, 2002, Arim *et al.*, 2011, Pawar, 2015).

In this paper, I use mathematical models to show that small body size has an intrinsic advantage in competition for space. First, I build a deterministic population dynamics with two phenotypes represented different body sizes and derive analytical conditions for persistence. Second, I build a spatially explicit stochastic birth-death model of a sessile organism. With this model I reinforce the results of the deterministic model and investigate the effects of dispersal. Third, I return to the deterministic model and extend it to include individuals which can vary continuously in body sizes. I use this model to study the effect of stabilizing natural selection and its interaction with the intrinsic benefit to small body size in competition for space. Specifically, I find the effects of mortality, viability range and optimal body size on body size distribution.

4.2 Population dynamics of two different body sizes

I begin with a simple population dynamics model of one species which has individuals of two different body sizes b_1 and b_2 . The goal of this model is to characterize the costs and benefits of different body sizes in competition for space. Therefore, I assume that all the needs of the species are fulfilled except for space which is the only limiting factor when the population size becomes large. I also assume that the individuals reproduce clonally to avoid the complexity of genetics and sexual reproduction. In this model the number of individuals of the two sizes (x and y) increases as long as there is space available for growth. They reproduce and consume space at rates c_1 and c_2 respectively. We call these parameters the (per-capita) space clearance rates. The two phenotypes also die at a constant rate m_1 and m_2 respectively. The dynamics of these two populations and the unused space r is represented by

$$\frac{dx}{dt} = \frac{c_1}{b_1}rx - m_1x \quad (4.1a)$$

$$\frac{dy}{dt} = \frac{c_2}{b_2}ry - m_2y \quad (4.1b)$$

$$\frac{dr}{dt} = b_1m_1x + b_2m_2y - (c_1x + c_2y)r \quad (4.1c)$$

Here, the birth rate of a phenotype is proportional to the total available space and the its abundance. The space clearance rate is proportional to the birth rate but is related to the amount of space available. The interpretation is more evident from the term in the parenthesis where c_i is per-capita rate at which each phenotype consumes space. As space gets consumed due to reproduction events, the number of individuals produced per unit space will be inversely proportional to the body size (equations 4.1a and 4.1b). In this model, the total space remains constant over time. For any initial conditions $x(0) = x_0$, $y(0) = y_0$ and $r(0) = r_0$, the total space $T = r_0 + b_1x_0 + b_2y_0$.

4.2.1 Results

Since total space is a constant over time, we have the identity $r(t) = T - b_1x(t) - b_2y(t)$. This is also evident from the differential equations 4.1 which satisfies the equation

$$\frac{dr}{dt} + b_1 \frac{dx}{dt} + b_2 \frac{dy}{dt} = 0.$$

Substituting for the available space $r(t)$ in equations 4.1a and 4.1b reduces the system of differential equations to two dimensions. Further algebraic manipulations allows us to rewrite the equations in the form of a standard two species Lotka-Volterra competition model (Chesson, 1990, Kot, 2001, Gotelli, 2001) with growth rates $\gamma_i = c_iT/b_i - m_i$, carrying capacities $K_i = T/b_i - m_i/c_i$, and competition coefficients $\alpha_{ii} = 1$ and $\alpha_{ij} = b_i/b_j$.

The outcome of Lotka-Volterra competition depends on the value of $\zeta_1 = \alpha_{12}K_2/K_1$ and $\zeta_2 = \alpha_{21}K_1/K_2$. Specifically if $\zeta_1 < 1$ and $\zeta_2 < 1$, then the two phenotypes coexist in a stable equilibrium. However in our model, $\zeta_1 = 1/\zeta_2$. Therefore exactly one of ζ_1 and ζ_2 is less than 1 and the other is greater than 1. In this case, the individuals with the lower value of ζ_1 survives at its carrying capacity and the other type becomes extinct. In terms of the parameters in equations 4.1,

$$\zeta_i = \frac{T - \frac{b_j m_j}{c_j}}{T - \frac{b_i m_i}{c_i}}.$$

Therefore the phenotype with a smaller $b_i m_i / c_i$ value survives while the other phenotype become extinct. Equivalently we can say that a phenotype has better chance of winning the competition for space if it is smaller, faster reproducing, or has lower mortality. Assuming that space clearance rate and death rate are the same for the two phenotypes, we can conclude that only the phenotype producing the smaller individuals survives. However, notice that the assumption of identical space clearance rate means that the smaller individuals have a higher per-capita growth rate.

4.3 Spatial population dynamics of two different body sizes

There are two limitations of the model given by equations 4.1. First, individuals do not occupy a contiguous unit of space. Spatial structure was ignored for a mathematically convenient description. This assumption can be justified in the case of animals which can move around to maximally utilize the total space available. However in most cases this is biologically and physically unrealistic. Second, smaller individuals had an intrinsic birth rate advantage since the space clearance rate did not depend on the body size. In this section I provide a spatially explicit model of two-dimensional sessile organisms where birth and death are independent stochastic processes. Therefore space clearance rates are not low-level prescribed parameters but varies indirectly due to stochastic dynamics.

Similar to the population dynamics model I will continue to model a single species with individuals of two different body sizes b_1 and b_2 . I will assume that reproduction and death are modelled by independent and exponentially distributed random variables. We can describe this as each individual carrying two hypothetical clocks which contains the next time to reproduce and the time at which the individual dies (Nair *et al.*, 2019). The rates at which b_1 and b_2 individuals gives birth are γ_1 and γ_2 respectively. The rates at which they die are m_1 and m_2 as before.

To avoid complexity at the edges of the space available to the species, I model the individuals and the space they occupy as circles. Body sizes b_1 and b_2 are the areas of individuals with circular bodies of radius β_1 and β_2 respectively. The total space is also a circle with the radius l . When an individual reproduces, I assume that they give birth to a single offspring which disperses to a random location. The offspring survives if the location it disperses to is unoccupied and within the bounds of the total space. Using simulations I studied the effect of body size, death rate, growth rate, and dispersal on the long-term population sizes of two phenotypes.

I studied two dispersal modes: (i) local and (ii) global dispersal. When the phenotypes disperses locally, the probability distribution of the offspring location (dispersal kernel) is represented by an uncorrelated two-dimensional normal distribution. I also assumed that the

standard deviations in both directions were the same and represent it by σ_i for phenotype i . When the phenotypes disperses globally, the dispersal kernel is represented by the uniform distribution on a disc with radius l .

4.3.1 Results

I simulated the spatially explicit model for a wide range of parameters. In almost all of the simulations, individuals with one body size survives while the other becomes extinct. Figure 4.1 shows these results. The condition for survival closely (black line in the figure) matches the analytical condition for survival from the population dynamics model. Specifically, the type of individuals with smaller value of $\beta_i^2 m_i / \gamma_i$ ($b_i m_i / c_i$ in the population dynamics model) survives. In simulations near the boundary of survival the two phenotypes seem to coexist. This is just a transient phenomenon due to the large time to extinction near this boundary.

The condition for survival matches the deterministic population dynamics model only when the death rates are large. When the death rates are small, the phenotype with the smaller body size has an advantage. Figure 4.2 shows that the smaller phenotype drives the larger phenotype to extinction at death rates where the smaller phenotype would have become extinct in the deterministic model.

When dispersal is local, we can expect increased death rates for small and large dispersal ranges (standard deviation σ). At low dispersal range, the offspring might not be sufficiently far from the parent whereas at high dispersal range the offspring might end up outside the total range. Figure 4.2 shows these results. When dispersal rates are equal ($\sigma = 5$), the figure shows the advantage to smaller individuals in comparison to the deterministic model. However when dispersal range is sufficiently low or high ($\sigma_2 = 0.1$ and $\sigma_2 = 50$ in the figure) the advantage due to small body size is overcome. Lower dispersal range particularly increases the time to extinction which explains the noisiness in Figure 4.2. The outcomes of global dispersal is qualitatively similar to the case of local dispersal at intermediate dispersal ranges but with shorter time to extinction.

The equilibrium population size of the surviving phenotype is lower in comparison to the population dynamics model. This is expected since the total space cannot be optimally

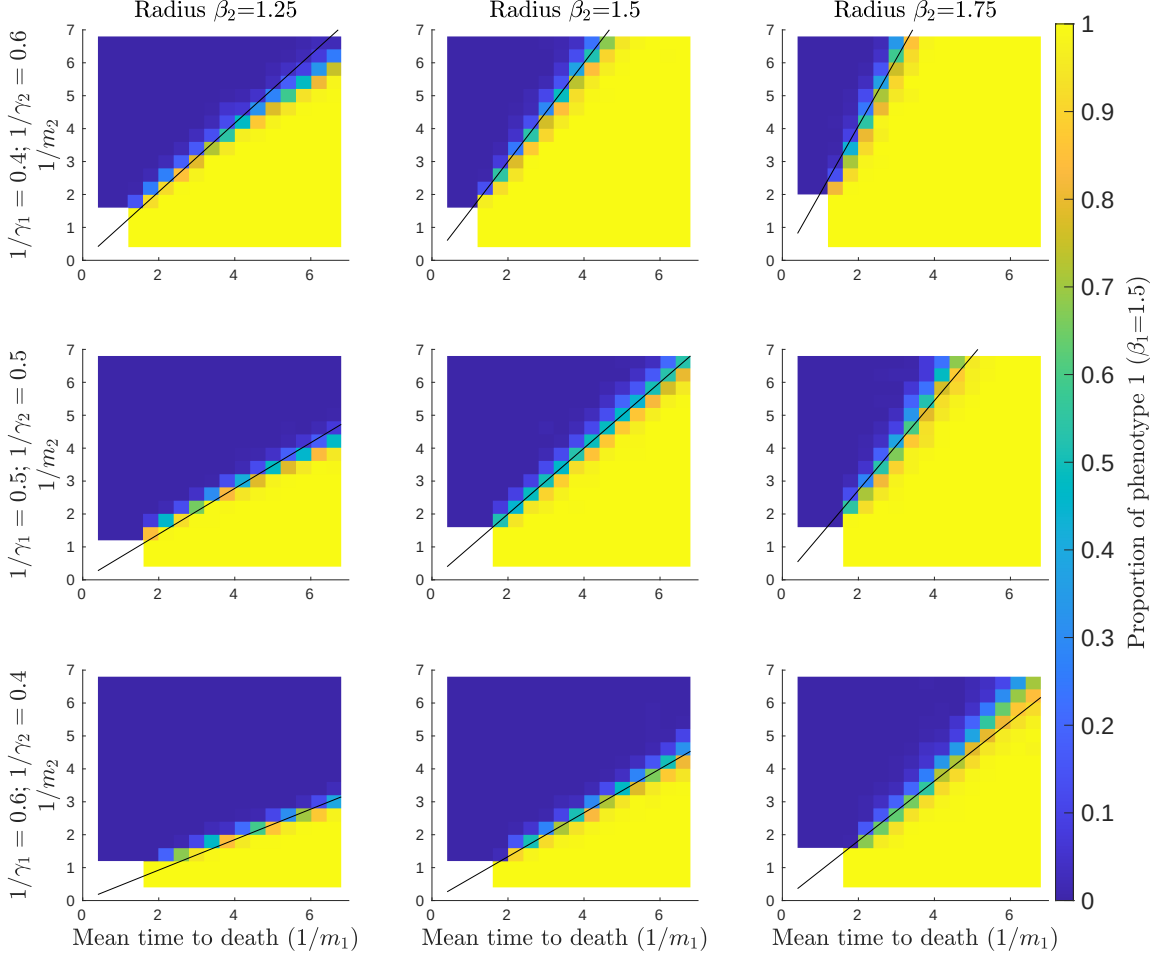


Figure 4.1: Death rates, birth rates and body sizes determines which of the two phenotypes survive. Yellow denotes the parameter combinations for which first phenotype survived whereas blue denotes that the second phenotype survived. The solid black line is the boundary of survival calculated from the deterministic population dynamics model, $1/m_2 = \beta_1^2/\gamma_1 1/m_1$. Offspring dispersal for both phenotypes was local with equal standard deviations ($\sigma = 5$) in a disc of radius $l = 20$. All simulations were initiated with 10 individuals of each phenotype and ran for 1000 units of time. The output (proportion of phenotype 1) is the average of 200 simulation per parameter combination.

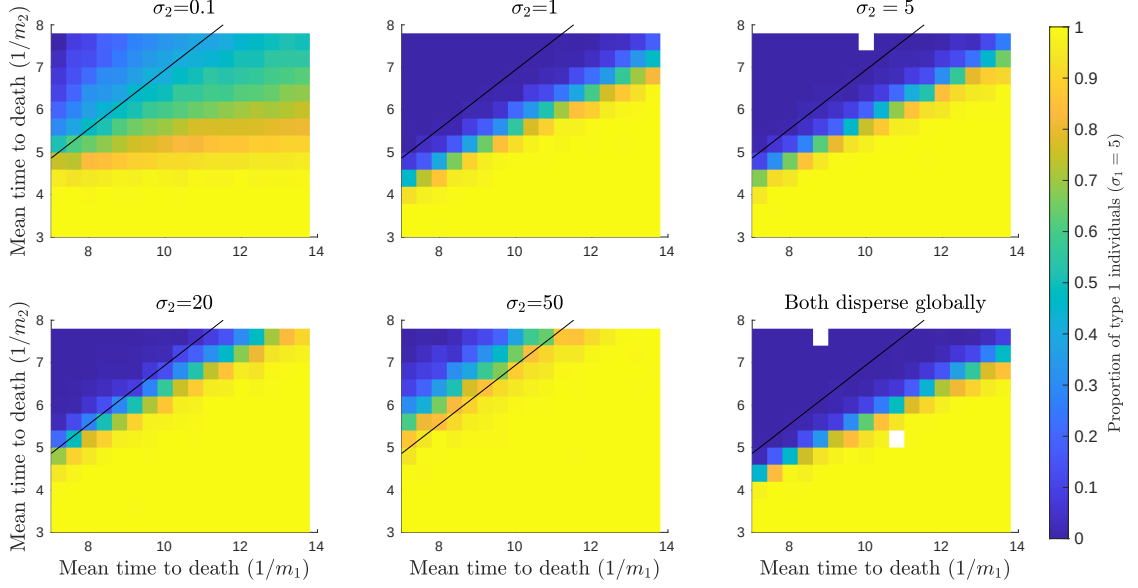


Figure 4.2: Region of persistence for small death rates and different dispersal ranges. Yellow denotes the parameter combinations for which first phenotype survived whereas blue denotes that the second phenotype survived. The solid black line is the boundary of survival calculated from the deterministic population dynamics model, $1/m_2 = \beta_1^2/\gamma_1 1/m_1$. All simulations were initiated with 10 individuals of each phenotype and ran for 5000 units of time. The output (proportion of phenotype 1) is the average of 200 simulation per parameter combination. Other parameters: $\gamma_1 = \gamma_2 = 0.5$, $\beta_1 = 1.5$ and $\beta_2 = 1.25$.

filled in the simulation model unlike the population dynamics model. The dependence of the equilibrium population on the body size and death rate are still qualitatively similar between the two models despite the population size being always lower in the simulation model. Specifically the population size increases as death rate and body size becomes smaller whereas it increases as growth rate increases (Figure 4.3). We also notice that the population size reaches its maximum value as the death rate decreases. This maximum population size is only related to the body size and does not depend on the growth rate.

Overall the conclusion that smaller body size is advantageous continues to be the case in the stochastic model. Large bodied individuals can outcompete the smaller individuals only when they have smaller death rates, larger birth rates, or an advantage due to dispersal. The stochastic simulations also provide the intuition that when space is limiting, an offspring of smaller individuals is more likely to find space to occupy compared to an offspring of a larger individual. In addition to this benefit, the disadvantages of stochastic death is stronger for larger individuals as they are fewer in number compared to smaller individuals when space is finite.

4.4 Dynamics of body size distribution

In this final model section I will extend the deterministic population dynamics model to an integro-differential equation which accounts for individuals with any body size. Specifically the differential equation will describe the dynamics of the body size distribution $N(z)$ which denotes the number of individuals with body size z . I model the stabilizing natural selection on body size as a decrease in second-order death rate. This is analogous to the differences in carrying capacity of individuals based on their body sizes. In contrast, modelling stabilizing selection in the first order birth or death term would lead to a homogeneous population with only the optimal phenotype surviving. The dynamics of body size distribution and available space is described by the following equations

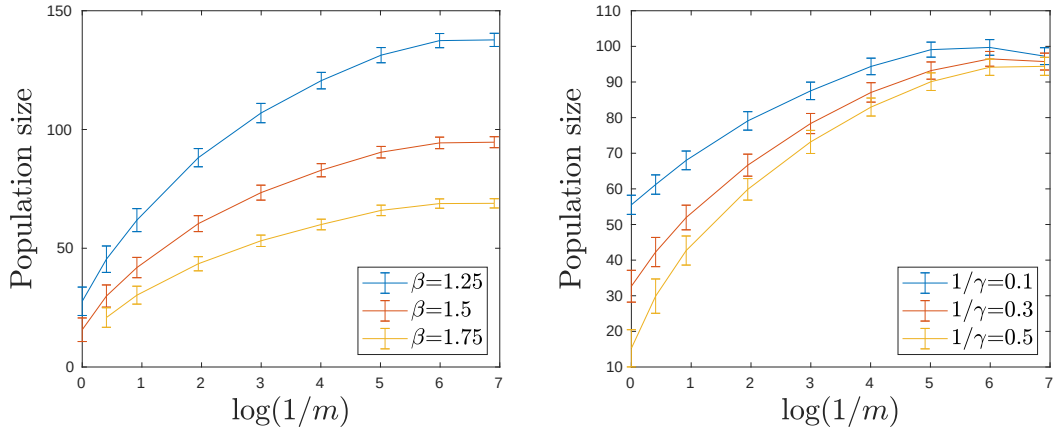


Figure 4.3: Equilibrium population size of the surviving phenotype increases with decreasing mortality (m) and body size (β), and increasing birth rate (γ). Simulations were initiated with 10 individuals of each phenotype and ran for 1000 units of time. The population size and the standard deviation error bars are based on 200 repetitions. Offspring dispersal for both phenotypes was local with equal standard deviations ($\sigma = 5$) in a disc of radius $l = 20$. Other parameters for the extinct phenotype: $\beta = 1.5, 1/\gamma = 0.5, 1/m = 0.1$. Other parameters for the surviving phenotype: (left panel) $1/\gamma = 0.5$ and (right panel) $\beta = 1.5$.

$$\frac{\partial N(z)}{\partial t} = \frac{c}{z} r N(z) - m N(z) \left(1 + \frac{N(z)}{\kappa(z)} \right), \quad (4.2a)$$

$$\frac{dr}{dt} = \int z m \left(1 + \frac{N(z)}{\kappa(z)} \right) N(z) dz - r \int c N(z) dz. \quad (4.2b)$$

Here, the stabilizing selection kernel $\kappa(z)$ is modelled as a Gaussian function $K \exp(-(z - \theta)^2 / 2\sigma^2)$ following other phenotypic models (Gavrilets, 1997, Roughgarden, 1972, Senthilnathan and Gavrilets, 2021). The parameters θ and σ represent the optimal body size and viability range respectively. Apart from mathematical convenience, modelling stabilizing selection in this manner captures density-dependent population regulation which can maintain variation. However, I consider density-dependent regulation within a phenotype and not across the entire population for simplicity. The full version of density-dependent regulation will involve accounting for density of all the phenotypes in the numerator instead of just $N(z)$.

Body sizes cannot be arbitrarily small as the limit of 0 body size will necessarily cause a blow up in the population size. Therefore to keep population size bounded, the domain of equations 4.2 needs to be restricted away from 0 which will be the minimum body size z_m . In the analysis which follows I set $z_m = 0.1$.

4.4.1 Results

I can analytically solve for the equilibrium from equation 4.2a to show that the zero function and

$$N^*(z) = \max \left(\kappa(z) \left(\frac{cr^*}{\mu z} - 1 \right), 0 \right) \quad (4.3)$$

are the only equilibrium body size distributions. However, we cannot analytically solve for the equilibrium space available r^* . Since $r^* \geq 0$ and by inspecting the equilibrium body size distribution given by equation 4.3, we can conclude that there is a maximum body size $\hat{z}$. *i.e.* There are no individuals with body size larger than $\hat{z}$.

I studied the dynamics of the model numerically using an adaptive finite difference method developed for similar integro-differential equations (Senthilnathan and Gavrilets, 2021). Numerical solutions for a wide range of parameters converged to an equilibrium

which qualitatively resemble the analytical solution in equation 4.3. Specifically there was a maximum body size $\hat{z}$. Figure 4.4 shows three qualitatively different equilibrium body size distributions. First, if optimal body size (θ) is small then the population density strictly decreases with increasing body sizes. Second, if optimal body size is large then there is an inflection point in the distribution which makes the distribution wider. This is due to strong stabilizing selection moving the weight of the distribution away from 0. Third, if optimal body size and the total space (T) is large then there is a local maxima or a second mode in the body size distribution. *i.e.* The density of individuals slightly smaller and larger than this body size (say $\tilde{z}$) is lower.

Figure 4.5 shows the effect of stabilizing natural selection on the population size ($\int N(z)dz$) and the two main aspects of the equilibrium distribution: mode ($\tilde{z}$) and maximum ($\hat{z}$) body size. Strong stabilizing selection is characterized by three parameters in this model. First, increasing death rate (m) intensifies selection by making death rate for all body sizes higher. Second, decreasing viability range intensifies selection by increasing death rate of individuals smaller or larger than the optimal body size. Third, increasing the optimal body size counters the intrinsic advantage to smaller individuals by increasing their death rates. Increasing death rate, optimal body size or decreasing viability range decreases the population size due to stronger selection. As expected, increasing the optimal body size increases the mode and maximum body size of the population. Wider viability range dilutes the disadvantages to smaller body sizes from stabilizing selection. Therefore the mode and maximum size decreases for increasing viability range. Increasing the total space increases population size, mode and maximum body size.

Overall, the smallest sized individuals are found at the highest densities in a population of individuals varying continuously in body sizes. This advantage can be weakened by strong stabilizing selection for an intermediate body size. Specifically, a sufficiently large death rate, optimal body size or a small viability range can lead to an intermediate body size have a population density.

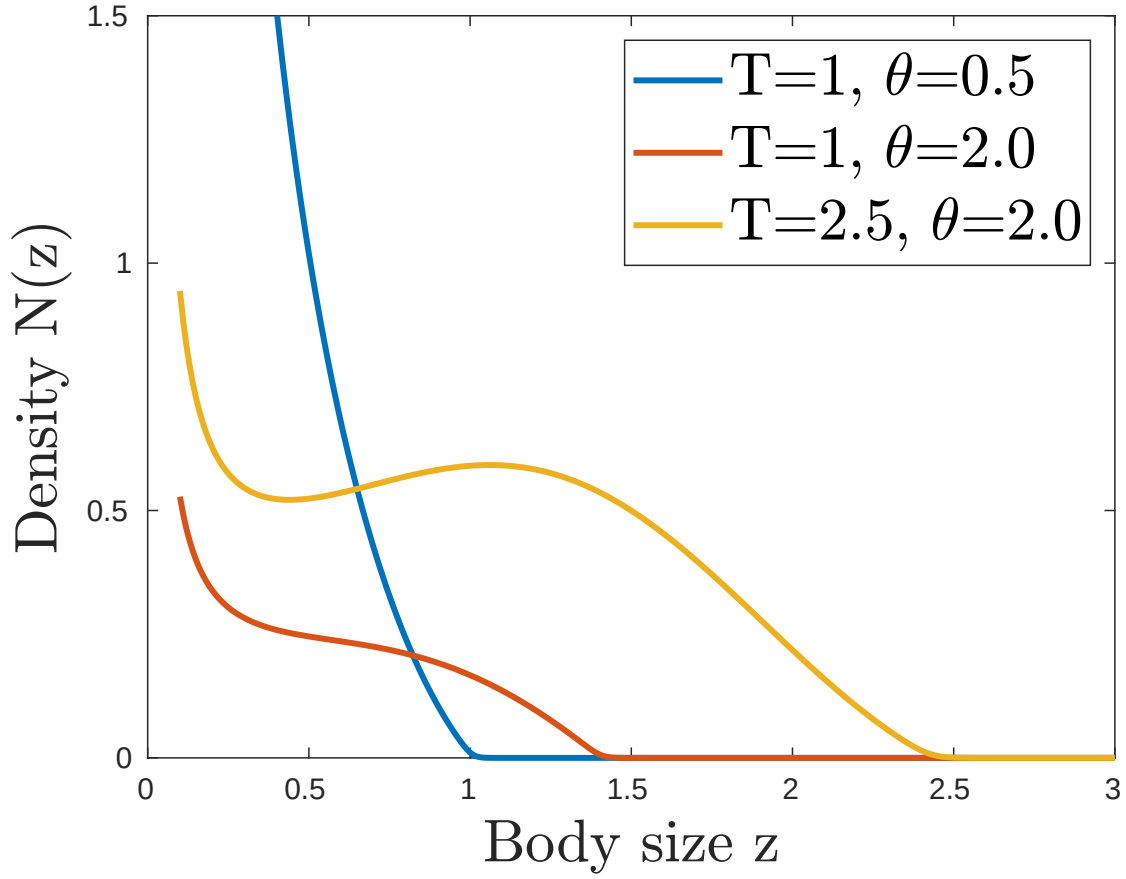


Figure 4.4: Examples of equilibrium body size distributions from numerical solutions of the model for continuous body sizes (Equations 4.2). The three numerical solutions were obtained with initial conditions $N(z) = \exp(-z)/z$ after 200 time steps. Other parameters: $c = 1$, $m = 0.6$, $K = 1$ and $\sigma = 0.75$.

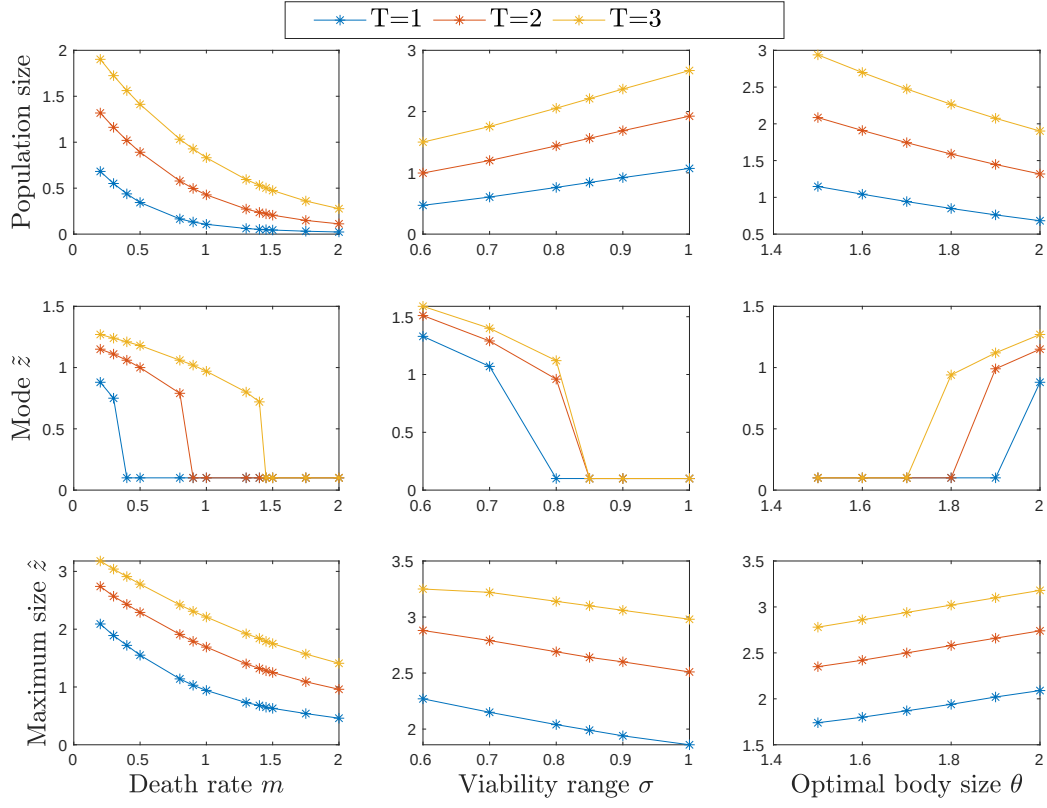


Figure 4.5: Effect of stabilizing natural selection on equilibrium body size distribution. All numerical solutions were obtained with initial conditions $N(z) = \exp(-z)/z$ after 200 time steps. Other parameters: $c = 1$ and $K = 1$.

4.5 Discussion

Being a prominent morphological trait and relatively easy to measure, body size has been studied extensively in ecology relating it to many questions. Among the one of the most simple but challenging question is whether being smaller or larger is better for organisms. In this paper, I study this question in the context of competition for space. I build three different mathematical models whose analysis leads to the conclusion that small body size has an intrinsic advantage in competition for space. Only significantly strong selection for a large body size can make the smallest possible size less abundant in the population.

My models are general representation of competition for space among sessile organism which differ in assumptions of stochasticity and stabilizing selection. In the first two models, there are only two phenotypes representing one small and one large body size. The first model is a simple deterministic model (equations 4.1) in which only the phenotype with the smaller value of $(body\ size) \times (mortality\ rate) / (space\ clearance\ rate)$ survives. Note that if the two phenotypes differ only in body size, then only the smaller body size individuals survive. This result continues to hold in the case of the spatially explicit model where individuals reproduce, disperse and die randomly (Figure 4.1). However, stochasticity further favors the smaller phenotype when death rates are small. This advantage to the smaller phenotype is lost when its dispersal range is very small or large (Figure 4.2).

My third model is an extension of the deterministic model with phenotypes which can continuously vary in body sizes instead of just two different sizes. This model also includes stabilizing selection through a density-dependent mechanism. Specifically, there is an optimal body size whose mortality is the low even at high abundances. The smallest phenotype is still very likely to be abundant while the abundance strictly decreases as body size increases. As selection intensifies due to an increase in the optimal body size and a decrease in viability range, the body size distribution become heavier at the tail. The analysis also shows that there is a maximum body size for the population which becomes larger as selection intensifies (Figure 4.5).

Body size distributions represent a wide range of patterns in ecology based on the scale at which the study is conducted as well as the scale of the biological question (White

et al., 2007, Belgrano and Reiss, 2011). For example the truncated power law pattern in body size distribution is typically observed at scales higher than the population-level and a pattern where abundance increases with body size can be observed at the within-taxa level (Nee *et al.*, 1991). Several mechanisms have been proposed to explain these patterns among which those based on bioenergetics and the metabolic theory of ecology are the most common (Uchmański, 1985, Blackburn and Gaston, 1999, Pawar, 2015). However these mechanisms equate body size and body mass, and do not specifically include the spatial aspect of competition due to body sizes.

The mechanism I propose is the competition for space. Simulations of my individual-based model illustrates that once the total space is nearly filled, only small individuals can occupy the few empty spaces available although both large and small individuals eventually perish. Such an advantage could generate body size distributions that resemble a truncated power-law distribution. However, analytical results of the continuous body size model show that the distribution is indeed not a truncated power law. Moreover there is a maximum body size beyond which larger individuals are absent in the population. When there is sufficient strong stabilizing selection for a large body size, body size distributions could have a local maxima or even become unimodal. These are reminiscent of within-taxa and intraspecific body size distributions (Nee *et al.*, 1991, Gouws *et al.*, 2011). Since clonal reproduction was assumed in the models presented here, competition for space could be used as an explanation for these wide variety of patterns in body size distributions with appropriate higher-level interpretation of natural selection.

Although existing mathematical models do not consider competition for space, they contribute a lot to our understanding of patterns of body sizes. Allometric scaling laws based on bioenergetics and metabolic theory are among the strongest statistical patterns we have in ecology (Peters, 1983, Brown *et al.*, 2004). West *et al.* (1997) has a general model of energy flows in branching networks to explain the power law dependence of several biological variable on body mass. These scaling laws form the basis of a class of models explaining body size distributions (Uchmański, 1985, Pawar, 2015, Lee *et al.*, 2021). Development and growth have been ignored in the models presented in this paper. Stage-structured models (often using a version of the McKendrick-vonFoerster equation) accounting for individuals

growth form another class of models describing body size distributions (Castle *et al.*, 2011, Datta *et al.*, 2011, Xu *et al.*, 2021).

Incorporating the evolutionary dynamics of body size in models has further enriched theoretical studies. Loeuille and Loreau (2005) presents an eco-evolutionary model which generates complex food webs from simple consumer-resource interaction through evolution. In their model, each species is characterized by a single phenotype. They choose this to be the body size which determines ecological interactions as well as is prone to mutation. Apart from showing that their model could generate food webs which resemble the structure of food webs observed in nature, they also characterize the body size distributions which appear in their model (Loeuille and Loreau, 2006). They find patterns which resemble the truncated power law and body size distributions where there is an intermediate body size with high abundance (local maximum). The latter pattern emerges when the range of consumption is large and the optimal size difference for predation is small. These patterns can immediately be contrasted with the body size patterns in Figure 4.4. However, the conditions for the different patterns to emerge are not comparable since these studies model fundamentally different ecological processes - space competition and predator-prey interactions. Luhning and DeLong (2020) is another example of an eco-evolutionary model of a community (food chain) of organisms which are under predator-prey interactions. In this model, body size determines death rate, prey handling time and other parameters which simultaneously rapidly evolving in response to the predator-prey interactions. The eco-evolutionary nature of this model allowed them to study not only classical trophic cascades of alternating shifts in abundance due removal of top predator but also shifts in body mass of the rest of the species. Overall, this shows that extending the models presented in this paper to more than one species should be an important next step in the study of body sizes.

The deterministic model represented by equations 4.1 has parallels to the theory on resource competition and R^* condition for survival (Tilman, 1982). Specifically, the quantity $(\text{body size}) \times (\text{mortality rate}) / (\text{space clearance rate})$ is the equivalent of R^* . A major limitation to this result is that it holds only when there are no other limiting resources. Moreover we ignore many other ecological processes which are known to be affected by body size (Peters, 1983, Schmidt-Nielsen, 1984). For example, Brown and Maurer (1986)

found that body size is positively correlated with resource acquisition which can explain the selection towards larger body sizes.

The assumption that the population is sessile and clonally reproducing are the other two major limitations. First, it is well known that body size affects locomotion (Schmidt-Nielsen, 1984, Cloyd and Dell, 2020). This could have interesting implications for our results particularly to the effects of dispersal. We could still expect an intrinsic advantage to small body size with some minor corrections (similar to the comparison between the first two models here). Second, although clonal reproduction relieves us from the mathematical complexity it severely limits the model by not being able to study sexual dimorphism. Differences in body sizes between the sexes is a classic example of a body size pattern (Fairbairn, 1997). A model with two sexes could also allow us to explore the effects of sexual selection on body size. This provides an interesting opportunity to study whether large body size advantage in any of these of ecological processes could sufficiently counteract the small body size advantage in space competition.

Overall, I studied the effect of space competition on body sizes using three different mathematical models. The results from all of them reveal an advantage that smaller individuals have in competition for space. With the third model I show how space competition and stabilizing natural selection on body size can interact to generate different patterns of body size distributions. Since body size is an prominent morphological trait which can be measured in units of space for any species on the tree of life, and the significant role body size has in different subdisciplines of ecology mathematical models like these could be the key to understanding general patterns in ecology.

Chapter 5

Conclusions

In this dissertation, I have build a series of eco-evolutionary models combining population dynamics models and evolutionary biology models to study the consequences of heritable intraspecific variation. I investigated the dynamic behavior as well as equilibrium properties of the corresponding integro-differential equations models using analytical approximations and numerical methods. I illustrated the utility of my mathematical framework for studying the ecological consequences of heritable intraspecific variation in biotic-biotic and biotic-abiotic interactions. Specifically (i) in chapter 2 I studied coevolutionary systems, (ii) in chapter 3 I studied the feedback between a single species and a single abiotic factor in its environment, and (iii) in chapter 4 I studied a specific trait of a single species (body size) and a specific abiotic factor (space). In all these studies I found that novel ecological dynamics emerged as a consequence of heritable variation within species.

My analytical approximations include novel applications of the invasion analysis (Armstrong and McGehee, 1980, Geritz *et al.*, 1998) and quasi-equilibrium approximations. In chapter 2 I derived sufficient conditions for stable coexistence in two-species competition and exploiter-victim system and necessary condition for two-species mutualism using an invasion analysis (Appendix A.1). In chapter 3, I used a quasi-equilibrium approximation where resource dynamics is assumed to be faster than consumer dynamics to derive equilibrium consumer trait distribution. I analyzed all the underlying population dynamics models analytically using standard linear stability analysis and other techniques (see Mathematica

notebook in SM). I also solved for equilibrium trait distribution of the eco-evolutionary models whenever it was feasible.

I supplemented all my analytical approximations with numerical solutions based on finite difference methods. These are standard numerical techniques for the population dynamics where derivatives are approximated using Euler forward difference (Simmons and Krantz, 2007, Kreyszig, 2009). This method reduces the ordinary differential equations to a system of difference equations which can then be solved using an iteration scheme. For the eco-evolutionary models I adapted a finite difference method from Doebeli (2011) (Appendix A.2). The idea here is similar to the Euler method for ordinary differential equations. The two key differences are the need to truncate the trait space to an appropriate interval on the real line and compute integrals using trapezoidal rule. The numerical solutions reinforced the analytical results as well as provided insights into the dynamic behavior of the models. Moreover this numerical method is also computationally faster than simulations of stochastic birth and death processes which can also generate the dynamics of entire trait distributions. Note that none of the models presented in this dissertation led to nonequilibrium dynamics. This is likely due to the addition of a second order term in models which typically show nonequilibrium behaviour without that term.

In the rest of this chapter I summarize the key results from my dissertation and outline some future directions.

5.1 Weak stabilizing selection promotes species coexistence

In chapter 2 I study coevolutionary systems but focus on ecological consequences. Coevolution is typically studied using evolutionary models where species coexistence is not tested but assumed (Nuismer, 2017). However that is not necessarily the case in ecological theory. Using my eco-evolutionary approach I found that coexistence conditions need to be revised while considering coevolution. Specifically I found that weak stabilizing selection promotes stable coexistence in two-species competition, exploiter-victim interaction and mutualism.

The intuition behind this result is that heritable intraspecific variation and weak selection allows for evolutionary flexibility - coevolving species can adjust their trait distribution in a way which allows them to coexist in a stable equilibrium. The inverse conclusion that strong stabilizing selection weakens coexistence is also true.

5.2 Classifying biotic-abiotic feedbacks

In chapter 3 I differentiate biotic-abiotic feedbacks where the abiotic factor is conditionable or consumable. This study is motivated by the fact that interference competition and exploitative competition are typically mediated by a conditionable or a consumable abiotic factor, respectively. Moreover, a consumable factor like soil nitrogen is vulnerable to monopolization and can be totally removed from the system while a conditionable factor like soil pH can only be manipulated by different individuals. I also consider the biological scenarios where the abiotic factor affects population dynamics and natural selection thereby establishing a biotic-abiotic feedback. Using my eco-evolutionary framework I show that both ecological and evolutionary dynamics are different between the two types of feedbacks. Specifically equilibrium population size is not affected by the strength of feedback when the abiotic factor is conditionable. However population size is maximized at intermediate values of strength of feedback when the abiotic factor is consumable. A novel conclusion from the study is that initial differences in abiotic state (abiotic origin) between two populations is reflected in the mean trait value of the populations over time only when the abiotic factor is conditionable.

5.3 Smaller is better in a competition for space

In chapter 4 I narrow down to a special case of biotic-abiotic feedback with a consumable abiotic factor. The trait and abiotic factor I study are body size and space, respectively. I use a stochastic birth-death model as well as the eco-evolutionary framework I used in the earlier chapters to show that being smaller is intrinsically beneficial in competition for space. The eco-evolutionary model also shows that known patterns of body size distributions can

emerge from the interaction of space competition and stabilizing natural selection. This is a novel mechanism which does not invoke metabolic theory to explain patterns of body size distributions (Blackburn and Gaston, 1999). I show that an intermediate body size can have a larger abundance than the smallest body size when there is strong stabilizing selection for a large body size as per expectations.

5.4 Future directions

As with any mathematical model, the eco-evolutionary models I have used in this dissertation has several limitations due to the assumptions made to simplify the complexity of nature. Although the goal of mathematical modelling is not to build an exact replica capturing every detail of the natural world, adding certain details might be rewarding. Incorporating sexual reproduction and explicitly keeping track of the sexes will expand the applicability of my results as well as allow for exploring questions regarding sex. Recent advances in the infinitesimal model which describes inheritance of quantitative traits (Barton *et al.*, 2017) and utilizing reproduction kernel (Doebeli, 2011) in a manner similar to competition kernel offer a path forward.

Both theoretical approaches in eco-evolutionary dynamics and empirical studies of rapid evolution suffer from an issue of timescales. Mathematical models typically assume a timescale separation between ecological and evolutionary dynamics or assume that they are on the same timescales (Lion, 2018). The former assumption is closely related to the commonly used quasi-equilibrium assumption (see Section 3.2.1). The latter assumption is typically justified based on empirical studies of rapid evolution. Although quantitative methods exists to infer whether evolution is rapid (Hairston *et al.*, 2005, DeLong *et al.*, 2016), there are differences between these methods and most studies do not utilize them. Instead they infer rapid evolution from observations of concurrent evolutionary (like trait evolution) as well as ecological (like population) dynamics. Future studies need to develop a standard quantitative approach to determine timescales of ecological and evolutionary dynamics and explore whether the two current timescale assumptions are at the ends of a continuum. This

could be of great value to our study of legacy effects and the consequences of evolutionary histories.

Our knowledge of ecology has benefited a lot from studies of pairwise interactions. A vast majority of theoretical studies are indeed based on pairwise interactions. However we need to go beyond studying two species to understand communities and ecosystems. Communities are more than a collection of pairwise interactions and studying more than two species at a time are often prohibitively complex. Network approaches have filled this niche effectively for many years. It is however primarily a phenomenological approach and cannot completely replace mechanistic studies of complex communities. There has been some promising efforts in understand species packing in communities using extension of standard population dynamics models (Leimar *et al.*, 2013, Barabás and D’Andrea, 2016). There are also research programs which are built towards integrating various aspects of ecology and evolution (Loreau, 2010, McPeck, 2017). Future studies developing the eco-evolutionary framework presented in this dissertation to include more than two species will be of relevance to these research programs.

Extensions involving more than two species is just one way to increase the dimensionality of the problem. The other major way is to increase the number of traits in each species. This could potentially be a harder problem since fewer studies have taken this direction. Moreover modelling how interactions depend on a vector of quantities as opposed to a single quantitative trait in itself could be a challenge. However Doebeli and Ispolatov (2010), Yamamichi *et al.* (2019) are just two examples where a multi-trait approach has been useful for studying eco-evolutionary dynamics. There are many interesting directions to take the eco-evolutionary framework proposed here and most of them will add novel perspectives to our understanding of ecology and evolution.

Bibliography

- Abrams, P. A. (2000). The Evolution of Predator-Prey Interactions: Theory and Evidence. *Annual Review of Ecology and Systematics*, **31**(1), 79–105. [10](#), [35](#)
- Abrams, P. A. and Matsuda, H. (1994). The evolution of traits that determine ability in competitive contests. *Evolutionary Ecology*, **8**(6), 667–686. [11](#)
- Abrams, P. A. and Matsuda, H. (1997). Fitness minimization and dynamic instability as a consequence of predator-prey coevolution. *Evolutionary Ecology*, **11**(1), 1–20. [35](#)
- Ackermann, M. and Doebeli, M. (2004). Evolution of niche width and adaptive diversification. *Evolution*, **58**(12), 2599–2612. [57](#)
- Aguilée, R., de Bédélièvre, B., Lambert, A., and Claessen, D. (2011). Under which conditions is character displacement a likely outcome of secondary contact? *Journal of Biological Dynamics*, **5**(2), 135–146. [3](#)
- Akçay, E. (2016). Evolutionary Models of Mutualism. In *Mutualism*, pages 57–76. Oxford University Press. [10](#)
- Albert, C. H., Grassein, F., Schurr, F. M., Vieilledent, G., and Violle, C. (2011). When and how should intraspecific variability be considered in trait-based plant ecology? *Perspectives in Plant Ecology, Evolution and Systematics*, **13**(3), 217–225. [1](#), [10](#), [42](#)
- Allen, W. J., Meyerson, L. A., Flick, A. J., and Cronin, J. T. (2018). Intraspecific variation in indirect plantsoil feedbacks influences a wetland plant invasion. *Ecology*, **99**(6), 1430–1440. [1](#), [11](#)
- Anderson, B. and Johnson, S. D. (2008). The geographical mosaic of coevolution in a plant-pollinator mutualism. *Evolution*, **62**(1), 220–225. [10](#)
- Anderson, J. J. (2010). Ratio- and predator-dependent functional forms for predators optimally foraging in patches. *The American Naturalist*, **175**(2), 240–249. [44](#)
- Andersson, M. and Iwasa, Y. (1996). Sexual selection. *Trends in Ecology and Evolution*, **11**(2), 53–58. [63](#)

- Arim, M., Berazategui, M., Barreneche, J. M., Ziegler, L., Zarucki, M., and Abades, S. R. (2011). Determinants of Density-Body Size Scaling Within Food Webs and Tools for Their Detection. In *Advances in Ecological Research*, volume 45, pages 1–39. Academic Press/Elsevier. [63](#)
- Armstrong, R. A. and McGehee, R. (1980). Competitive Exclusion. *The American Naturalist*, **115**(2), 151–170. [17](#), [36](#), [80](#)
- Austin, M. W. and Dunlap, A. S. (2019). Intraspecific variation in worker body size makes North American bumble bees (*Bombus* spp.) less susceptible to decline. *The American Naturalist*, **194**(3), 381–394. [1](#), [11](#), [33](#)
- Barabás, G. and D’Andrea, R. (2016). The effect of intraspecific variation and heritability on community pattern and robustness. *Ecology Letters*, **19**(8), 977–986. [84](#)
- Barabás, G., D’Andrea, R., and Stump, S. M. (2018). Chesson’s coexistence theory. *Ecological Monographs*, **88**(3), 277–303. [34](#)
- Barton, N. H. (1986). The maintenance of polygenic variation through a balance between mutation and stabilizing selection. *Genetical Research*, **47**(3), 209–216. [11](#)
- Barton, N. H., Etheridge, A. M., and Véber, A. (2017). The infinitesimal model: Definition, derivation, and implications. *Theoretical Population Biology*, **118**, 50–73. [83](#)
- Bayliss, S. L. J., Mueller, L. O., Ware, I. M., Schweitzer, J. A., and Bailey, J. K. (2020). Plant genetic variation drives geographic differences in atmosphere-plant-ecosystem feedbacks. *Plant-Environment Interactions*, **1**(3), 166–180. [55](#)
- Begon, M., Townsend, C. R., and Harper, J. L. (2006). *Ecology: from individuals to ecosystems*. Blackwell Publishing Ltd. [42](#)
- Belgrano, A. and Reiss, J. (2011). *Advances in ecological research - The role of body size in multispecies systems*. Academic Press/Elsevier. [63](#), [77](#)

- Benkman, C. W., Parchman, T. L., Favis, A., and Siepielski, A. M. (2003). Reciprocal selection causes a coevolutionary arms race between crossbills and lodgepole pine. *The American Naturalist*, **162**(2), 182–194. [10](#)
- Blackburn, T. M. and Gaston, K. J. (1999). The Relationship between Animal Abundance and Body Size: A Review of the Mechanisms. In *Advances in Ecological Research*, volume 28, pages 181–210. Academic Press. [63](#), [77](#), [83](#)
- Blanckenhorn, W. U. (2000). The evolution of body size: What keeps organisms small? *Quarterly Review of Biology*, **75**(4), 385–407. [6](#), [62](#), [63](#)
- Bolnick, D. I. and Doebeli, M. (2003). Sexual dimorphism and adaptive speciation: two sides of the same ecological coin. *Evolution*, **57**(11), 2433–2449. [36](#)
- Bolnick, D. I., Amarasekare, P., Araújo, M. S., Bürger, R., Levine, J. M., Novak, M., Rudolf, V. H. W., Schreiber, S. J., Urban, M. C., and Vasseur, D. A. (2011). Why intraspecific trait variation matters in community ecology. *Trends in Ecology and Evolution*, **26**(4), 183–192. [1](#), [10](#), [42](#)
- Bonanomi, G., Giannino, F., and Mazzoleni, S. (2005). Negative plant-soil feedback and species coexistence. *Oikos*, **111**(2), 311–321. [39](#), [58](#)
- Breza, L. C., Souza, L., Sanders, N. J., and Classen, A. T. (2012). Within and between population variation in plant traits predicts ecosystem functions associated with a dominant plant species. *Ecology and Evolution*, **2**(6), 1151–1161. [1](#), [33](#)
- Bronstein, J. L. (2015). *Mutualism*. Oxford University Press. [26](#)
- Brouat, C., Garcia, N., Andary, C., and McKey, D. (2001). Plant lock and ant key: Pairwise coevolution of an exclusion filter in an ant-plant mutualism. *Proceedings of the Royal Society B: Biological Sciences*, **268**(1481), 2131–2141. [9](#), [27](#)
- Brown, J. H. and Maurer, B. A. (1986). Body size, ecological dominance and cope’s rule. *Nature*, **324**(6094), 248–250. [78](#)

- Brown, J. H., Gillooly, J. F., Allen, A. P., Savage, V. M., and West, G. B. (2004). Toward a metabolic theory of ecology. *Ecology*, **85**(7), 1771–1789. [63](#), [77](#)
- Brown, W. L. and Wilson, E. O. (1956). Character Displacement. *Systematic Zoology*, **5**(2), 49–64. [2](#), [11](#), [33](#)
- Buckling, A. and Rainey, P. B. (2002). The role of parasites in sympatric and allopatric host diversification. *Nature*, **420**(6915), 496–499. [2](#), [11](#)
- Bulmer, M. G. (1971). The Effect of Selection on Genetic Variability. *The American Naturalist*, **105**(943), 201–211. [11](#), [12](#)
- Bulmer, M. G. (1985). *The mathematical theory of quantitative genetics*. Clarendon Press. [2](#), [3](#)
- Castle, M. D., Blanchard, J. L., and Jennings, S. (2011). Predicted Effects of Behavioural Movement and Passive Transport on Individual Growth and Community Size Structure in Marine Ecosystems. In *Advances in Ecological Research*, volume 45, pages 41–66. Elsevier Ltd., 1 edition. [78](#)
- Chesson, P. (1990). MacArthur’s consumer-resource model. *Theoretical Population Biology*, **37**(1), 26–38. [43](#), [47](#), [65](#)
- Chesson, P. (2000). Mechanisms of Maintenance of Species Diversity. *Annual Review of Ecology and Systematics*, **31**, 343–366. [34](#)
- Clark, J. S. (2010). Individuals and the Variation Needed for High Species Diversity in Forest Trees. *Scientific Reports*, **327**(February), 1129–1132. [34](#)
- Clarke, B. C. (1979). The evolution of genetic diversity. *Proceedings of the Royal Society of London - Biological Sciences*, **205**(1161), 453–474. [2](#), [11](#)
- Clayton, D. H., Lee, P. L. M., Tompkins, D. M., and Brodie III, E. D. (1999). Reciprocal Natural Selection on Host-Parasite Phenotypes. *The American Naturalist*, **154**(3), 261–270. [9](#), [20](#)

- Cloyed, C. S. and Dell, A. I. (2020). The body size and temperature dependence of organismal locomotion. *Ecology*, **101**(10). [79](#)
- Crawford, K. M. and Hawkes, C. V. (2020). Soil precipitation legacies influence intraspecific plantsoil feedback. *Ecology*, **101**(10), e03142. [55](#)
- Cuddington, K. (2011). Legacy Effects: The Persistent Impact of Ecological Interactions. *Biological Theory*, **6**(3), 203–210. [45](#), [55](#)
- Datta, S., Delius, G. W., Law, R., and Plank, M. J. (2011). A stability analysis of the power-law steady state of marine size spectra. *Journal of Mathematical Biology*, **63**(4), 779–799. [78](#)
- Davies, N. B. and Brooke, M. d. L. (1988). Cuckoos versus reed warblers: adaptation and counteradaptations. *Animal Behaviour*, **36**, 262–284. [9](#), [20](#)
- Dayan, T. and Simberloff, D. (2005). Ecological and community-wide character displacement: the next generation. *Ecology Letters*, **8**(8), 875–894. [3](#), [11](#), [33](#)
- Delong, J. P., Forbes, V. E., Galic, N., Gibert, J. P., Laport, R. G., Phillips, J. S., and Vavra, J. M. (2016). How fast is fast? Eco-evolutionary dynamics and rates of change in populations and phenotypes. *Ecology and Evolution*, **6**(2), 573–581. [83](#)
- Des Roches, S., Post, D. M., Turley, N. E., Bailey, J. K., Hendry, A. P., Kinnison, M. T., Schweitzer, J. A., and Palkovacs, E. P. (2018). The ecological importance of intraspecific variation. *Nature Ecology & Evolution*, **2**(1), 57–64. [1](#), [11](#), [33](#), [42](#)
- Des Roches, S., Pendleton, L. H., Shapiro, B., and Palkovacs, E. P. (2021). Conserving intraspecific variation for nature’s contributions to people. *Nature Ecology and Evolution*, **5**(5), 574–582. [1](#)
- Dieckmann, U. and Doebeli, M. (1999). On the origin of species by sympatric speciation. *Nature*, **400**(22), 354–357. [4](#), [11](#), [12](#), [26](#)
- Diekmann, O. (2004). A beginner’s guide to adaptive dynamics. *Banach Center Publications*, **63**, 47–86. [4](#), [12](#)

- Doebeli, M. (1996a). An explicit genetic model for ecological character displacement. *Ecology*, **77**(2), 510–520. [3](#)
- Doebeli, M. (1996b). Quantitative genetics and population dynamics. *Evolution*, **50**(2), 532–546. [3](#)
- Doebeli, M. (1997). Genetic variation and the persistence of predator-prey interactions in the Nicholson-Bailey model. *Journal of Theoretical Biology*, **188**(1), 109–120. [10](#)
- Doebeli, M. (2011). *Adaptive diversification (MPB-48)*. Princeton University Press. [19](#), [36](#), [42](#), [81](#), [83](#), [110](#)
- Doebeli, M. and Dieckmann, U. (2000). Evolutionary Branching and Sympatric Speciation Caused by Different Types of Ecological Interactions. *The American Naturalist*, **156**(S4), S77–S101. [10](#), [35](#)
- Doebeli, M. and Ispolatov, I. (2010). Complexity and diversity. *Science (New York, N.Y.)*, **328**(5977), 494–7. [13](#), [14](#), [32](#), [33](#), [84](#)
- Ellner, S. P., Snyder, R. E., Adler, P. B., and Hooker, G. (2019). An expanded modern coexistence theory for empirical applications. *Ecology Letters*, **22**(1), 3–18. [34](#)
- Endler, J. A. (1986). *Natural selection in the wild (MPB-21)*. Princeton University Press. [39](#)
- Erwin, D. H. (2008). Macroeolution of ecosystem engineering, niche construction and diversity. *Trends in Ecology and Evolution*, **23**(6), 304–310. [40](#), [56](#)
- Fairbairn, D. J. (1997). Allometry for sexual size dimorphism: Pattern and process in the coevolution of body size in males and females. *Annual Review of Ecology and Systematics*, **28**(1), 659–687. [79](#)
- Ferrière, R., Bronstein, J. L., Rinaldi, S., Law, R., and Gauduchon, M. (2002). Cheating and the evolutionary stability of mutualisms. *Proceedings of the Royal Society B: Biological Sciences*, **269**(1493), 773–780. [4](#), [10](#)

- Ferrière, R., Gauduchon, M., and Bronstein, J. L. (2007). Evolution and persistence of obligate mutualists and exploiters: Competition for partners and evolutionary immunization. *Ecology Letters*, **10**(2), 115–126. [10](#)
- Fleischer, S. R., TerHorst, C. P., and Li, J. (2018). Pick your trade-offs wisely: Predator-prey eco-evo dynamics are qualitatively different under different trade-offs. *Journal of Theoretical Biology*, **456**, 201–212. [10](#)
- Foster, K. R. and Kokko, H. (2006). Cheating can stabilize cooperation in mutualisms. *Proceedings of the Royal Society B: Biological Sciences*, **273**(1598), 2233–2239. [10](#)
- Fox, J. W. and Vasseur, D. A. (2008). Character convergence under competition for nutritionally essential resources. *The American Naturalist*, **172**(5), 667–680. [57](#)
- Frankham, R. (1996). Relationship of Genetic Variation to Population Size in Wildlife. *Conservation Biology*, **10**(6), 1500–1508. [1](#), [11](#), [33](#)
- Fricke, E. C., Tewksbury, J. J., and Rogers, H. S. (2019). Linking intraspecific trait variation and plant function: seed size mediates performance tradeoffs within species. *Oikos*, **128**(12), 1716–1725. [34](#)
- Fussmann, G. F., Loreau, M., and Abrams, P. A. (2007). Eco-evolutionary dynamics of communities and ecosystems. *Functional Ecology*, **21**(3), 465–477. [56](#)
- Futuyma, D. J. and Slatkin, M. (1983). *Coevolution*. Sinauer Associates. [9](#)
- Gause, G. F. and Witt, A. A. (1935). Behavior of Mixed Populations and the Problem of Natural Selection. *The American Naturalist*, **69**(725), 596–609. [5](#), [26](#)
- Gavrilets, S. (1997). Coevolutionary chase on exploiter-victim systems with polygenic characters. *Journal of Theoretical Biology*, **186**(4), 527–534. [xii](#), [10](#), [13](#), [20](#), [22](#), [33](#), [34](#), [36](#), [72](#), [112](#), [113](#)
- Gavrilets, S. (2004). *Fitness Landscapes and the Origin of Species (MPB-41)*. Princeton University Press. [2](#), [11](#)

- Gavrilets, S. and Hastings, A. (1994a). Dynamics of genetic variability in two-locus models of stabilizing selection. *Genetics*, **138**(2), 519–532. [3](#), [11](#), [12](#)
- Gavrilets, S. and Hastings, A. (1994b). Maintenance of multilocus variability under strong stabilizing selection. *Journal of Mathematical Biology*, **32**(4), 287–302. [11](#)
- Gavrilets, S. and Hastings, A. (1995). Dynamics of polygenic variability under stabilizing selection, recombination, and drift. *Genetical Research*, **65**(1), 63–74. [3](#), [11](#), [12](#)
- Gavrilets, S. and Michalakis, Y. (2008). Effects of environmental heterogeneity on victim-exploiter coevolution. *Evolution*, **62**(12), 3100–3116. [10](#)
- Gavrilets, S. and Waxman, D. (2002). Sympatric speciation by sexual conflict. *Proceedings of the National Academy of Sciences of the United States of America*, **99**(16), 10533–10538. [35](#), [36](#)
- Geritz, S. A. H., Kisdi, É., Meszéna, G., and Metz, J. A. (1998). Evolutionarily Singular Strategies and the Adaptive Growth and Branching of the Evolutionary Tree. *Evolutionary Ecology*, **12**(1), 35–57. [4](#), [11](#), [12](#), [17](#), [35](#), [57](#), [80](#)
- Gómez, J. M., Perfectti, F., Bosch, J., and Camacho, J. P. (2009). A geographic selection mosaic in a generalized plant-pollinator-herbivore system. *Ecological Monographs*, **79**(2), 245–263. [10](#)
- Gomulkiewicz, R., Thompson, J. N., Holt, R. D., Nuismer, S. L., and Hochberg, M. E. (2000). Hot Spots, Cold Spots, and the Geographic Mosaic Theory of Coevolution. *The American Naturalist*, **156**(2), 156–174. [10](#)
- Gotelli, N. J. (2001). *A Primer of Ecology*. Sinauer Associates. [42](#), [65](#)
- Gouws, E. J., Gaston, K. J., and Chown, S. L. (2011). Intraspecific body size frequency distributions of insects. *PLoS ONE*, **6**(3), 1–8. [77](#)
- Govaert, L., Fronhofer, E. A., Lion, S., Eizaguirre, C., Bonte, D., Egas, M., Hendry, A. P., De Brito Martins, A., Melián, C. J., Raeymaekers, J. A. M., Ratikainen, I. I., Saether, B.,

- Schweitzer, J. A., and Matthews, B. (2018). Ecoevolutionary feedbacks Theoretical models and perspectives. *Functional Ecology*, **33**(1), 1365–2435.13241. [2](#)
- Griffiths, J. I., Childs, D. Z., Bassar, R. D., Coulson, T., Reznick, D. N., and Rees, M. (2020). Individual differences determine the strength of ecological interactions. *Proceedings of the National Academy of Sciences of the United States of America*, **117**(29), 17068–17073. [63](#)
- Grover, J. P. (1997). *Resource Competition*. Springer. [40](#)
- Gurney, W. S. C. and Lawton, J. H. (1996). The Population Dynamics of Ecosystem Engineers. *Oikos*, **76**(2), 273–283. [57](#)
- H Hairston, N. G., Ellner, S. P., Geber, M. A., Yoshida, T., and Fox, J. A. (2005). Rapid evolution and the convergence of ecological and evolutionary time. [83](#)
- Haney, S. D. and Siepielski, A. M. (2018). Tipping Points in Resource Abundance Drive Irreversible Changes in Community Structure. *The American Naturalist*, **191**(5), 668–675. [40](#), [57](#)
- Hart, S. P., Schreiber, S. J., and Levine, J. M. (2016). How variation between individuals affects species coexistence. *Ecology Letters*, **19**(8), 825–838. [33](#)
- Hausch, S., Vamosi, S. M., and Fox, J. W. (2018). Effects of intraspecific phenotypic variation on species coexistence. *Ecology*, **99**(6), 1453–1462. [1](#), [11](#), [34](#)
- Hayashi, T. I., Vose, M., and Gavrillets, S. (2007). Genetic differentiation by sexual conflict. *Evolution*, **61**(3), 516–529. [36](#)
- Huisman, G. and De Boer, R. J. (1997). A formal derivation of the 'Beddington' functional response. *Journal of Theoretical Biology*, **185**(3), 389–400. [44](#), [45](#)
- Hutchinson, G. E. (1959). Homage to Santa Rosalia or Why Are There So Many Kinds of Animals? *The American Naturalist*, **93**(870), 145–159. [2](#)
- Iwasa, Y., Pomiankowski, A., and Nee, S. (1991). The evolution of costly mate preferences II. The "Handicap" principle. *Evolution*, **45**(456), 1431–1442. [3](#)

- Jacquemyn, H., Vandepitte, K., Roldán-Ruiz, I., and Honnay, O. (2009). Rapid loss of genetic variation in a founding population of *Primula elatior* (Primulaceae) after colonization. *Annals of Botany*, **103**(5), 777–783. [2](#), [11](#)
- Janzen, D. H. (1980). When is it coevolution? *Evolution*, **34**(3), 611–612. [9](#)
- Jennings, S., Warr, K. J., and Mackinson, S. (2002). Use of size-based production and stable isotope analyses to predict trophic transfer efficiencies and predator-prey body mass ratios in food webs. *Marine Ecology Progress Series*, **240**, 11–20. [62](#), [63](#)
- Jiang, J. and DeAngelis, D. L. (2013). Strong species-Environment feedback shapes plant community assembly along environmental gradients. *Ecology and Evolution*, **3**(12), 4119–4128. [58](#)
- Johnson, N. C., Wilson, G. W. T., Bowker, M. A., Wilson, J. A., and Miller, R. M. (2010). Resource limitation is a driver of local adaptation in mycorrhizal symbioses. *Proceedings of the National Academy of Sciences*, **107**(5), 2093–2098. [40](#), [50](#)
- Jones, C. G., Lawton, J. H., and Shachak, M. (1997). Positive and Negative Effects of Organisms as Physical Ecosystem Engineers. *Ecology*, **78**(7), 1946–1957. [40](#)
- Jung, V., Violle, C., Mondy, C., Hoffmann, L., and Muller, S. (2010). Intraspecific variability and trait-based community assembly. *Journal of Ecology*, **98**(5), 1134–1140. [34](#)
- Keller, I. and Largiadèr, C. R. (2003). Recent habitat fragmentation caused by major roads leads to reduction of gene flow and loss of genetic variability in ground beetles. *Proceedings of the Royal Society B: Biological Sciences*, **270**(1513), 417–423. [2](#), [11](#)
- Kisdi, É. (1999). Evolutionary branching under asymmetric competition. *Journal of Theoretical Biology*, **197**(2), 149–162. [33](#)
- Kopp, M. and Gavrillets, S. (2006). Multilocus genetics and the coevolution of quantitative traits. *Evolution*, **60**(7), 1321–1336. [2](#), [3](#), [10](#), [33](#), [34](#), [36](#)
- Kostitzin, V. (1939). *Mathematical biology*. George G. Harrap And Company Ltd. London. [5](#), [26](#)

- Kot, M. (2001). *Elements of Mathematical Ecology*. Cambridge University Press. [13](#), [22](#), [43](#), [65](#)
- Krakauer, D. C., Page, K. M., and Erwin, D. H. (2009). Diversity, Dilemmas, and Monopolies of Niche Construction. *The American Naturalist*, **173**(1), 26–40. [42](#), [56](#), [58](#)
- Kremer, C. T. and Klausmeier, C. A. (2017). Species packing in eco-evolutionary models of seasonally fluctuating environments. *Ecology Letters*, **20**(9), 1158–1168. [4](#), [10](#)
- Kreyszig, E. (2009). *Advanced engineering mathematics*. Wiley. [81](#), [110](#)
- Kylafis, G. and Loreau, M. (2008). Ecological and evolutionary consequences of niche construction for its agent. *Ecology Letters*, **11**(10), 1072–1081. [4](#), [58](#)
- Kylafis, G. and Loreau, M. (2011). Niche construction in the light of niche theory. *Ecology Letters*, **14**(2), 82–90. [42](#)
- Laland, K. N., Odling-Smee, F. J., and Feldman, M. W. (1999). Evolutionary consequences of niche construction and their implications for ecology. *Proceedings of the National Academy of Sciences*, **96**(18), 10242–10247. [42](#), [57](#)
- Laland, K. N., Uller, T., Feldman, M. W., Sterelny, K., Müller, G. B., Moczek, A., Jablonka, E., Odling-Smee, F. J., Wray, G. A., Hoekstra, H. E., Futuyma, D. J., Lenski, R. E., Mackay, T. F. C., Schluter, D., and Strassmann, J. E. (2014). Does evolutionary theory need a rethink? *Nature*, **514**(7521), 161–164. [40](#), [56](#)
- Lande, R. (1975). The maintenance of genetic variability by mutation in a polygenic character with linked loci. *Genetical Research*, **26**(3), 221–235. [11](#)
- Lande, R. (1976). Natural Selection and Random Genetic Drift in Phenotypic Evolution. *Evolution*, **30**(2), 314–334. [3](#)
- Le Roux, E., Van Veenhuisen, L. S., Kerley, G. I., and Croomsigt, J. P. (2020). Animal body size distribution influences the ratios of nutrients supplied to plants. *Proceedings of the National Academy of Sciences of the United States of America*, **117**(36), 22256–22263. [62](#)

- Lecerf, A. and Chauvet, E. (2008). Intraspecific variability in leaf traits strongly affects alder leaf decomposition in a stream. *Basic and Applied Ecology*, **9**(5), 598–605. [42](#)
- Lee, E. D., Kempes, C. P., and West, G. B. (2021). Growth, death, and resource competition in sessile organisms. *Proceedings of the National Academy of Sciences*, **118**(15), 1–9. [63](#), [77](#)
- Leimar, O., Sasaki, A., Doebeli, M., and Dieckmann, U. (2013). Limiting similarity, species packing, and the shape of competition kernels. *Journal of Theoretical Biology*, **339**, 3–13. [10](#), [84](#)
- Lewontin, R. (2000). *The triple helix: gene, organism, and environment*. Harvard University Press. [56](#)
- Lichstein, J. W., Dushoff, J., Levin, S. A., and Pacala, S. W. (2007). Intraspecific variation and species coexistence. *The American Naturalist*, **170**(6), 807–818. [33](#)
- Lion, S. (2018). Theoretical Approaches in Evolutionary Ecology: Environmental Feedback as a Unifying Perspective. *The American Naturalist*, **191**(1), 21–44. [2](#), [4](#), [60](#), [83](#)
- Lloyd-Smith, J. O., Schreiber, S. J., Kopp, P. E., and Getz, W. M. (2005). Superspreading and the impact of individual variation on disease emergence. *Nature*, **438**(7066), 355–9. [1](#), [11](#)
- Loeuille, N. and Loreau, M. (2005). Evolutionary emergence of size-structured food webs. *Proceedings of the National Academy of Sciences of the United States of America*, **102**(16), 5761–5766. [78](#)
- Loeuille, N. and Loreau, M. (2006). Evolution of body size in food webs: Does the energetic equivalence rule hold? *Ecology Letters*, **9**(2), 171–178. [78](#)
- Loreau, M. (2010). *From Populations to Ecosystems : Theoretical Foundations for a New Ecological Synthesis*. Princeton University Press. [40](#), [60](#), [84](#)

- Losos, J. B. (2000). Ecological character displacement and the study of adaptation. *Proceedings of the National Academy of Sciences of the United States of America*, **97**(11), 5693–5. [2](#)
- Lotka, A. J. (1920). Analytical Note on Certain Rhythmic Relations in Organic Systems. *Proceedings of the National Academy of Sciences*, **6**(7), 410–415. [5](#), [22](#), [34](#)
- Luhring, T. M. and DeLong, J. P. (2020). Trophic cascades alter eco-evolutionary dynamics and body size evolution. *Proceedings of the Royal Society B: Biological Sciences*, **287**(1938), 20200526. [78](#)
- MacArthur, R. H. (1970). Species packing and competitive equilibrium for many species. *Theoretical Population Biology*, **1**(1), 1–11. [43](#), [47](#), [57](#)
- MacArthur, R. H. and Levins, R. (1967). The Limiting Similarity, Convergence, and Divergence of Coexisting Species. *The American Naturalist*, **101**(921), 377–385. [17](#)
- Matthews, B., Narwani, A., Hausch, S., Nonaka, E., Peter, H., Yamamichi, M., Sullam, K. E., Bird, K. C., Thomas, M. K., Hanley, T. C., and Turner, C. B. (2011). Toward an integration of evolutionary biology and ecosystem science. *Ecology Letters*, **14**(7), 690–701. [39](#)
- May, R. (1981). Models for Two Interacting Populations. In *Theoretical Ecology: Principles and Applications*, pages 78–104. Sinauer Associates. [26](#)
- McPeck, M. A. (2017). *Evolutionary community ecology*. Princeton University Press. [37](#), [60](#), [84](#)
- Mitrovski, P., Hoffmann, A. A., Heinze, D. A., and Weeks, A. R. (2008). Rapid loss of genetic variation in an endangered possum. *Biology Letters*, **4**(1), 134–138. [2](#), [11](#)
- Nair, G. G., Senthilnathan, A., Iyer, S. K., and Guttal, V. (2019). Fission-fusion dynamics and group-size dependent composition in heterogeneous populations. *Physical Review E*, **99**(3), 032412. [66](#)

- Nee, S., Read, A. F., Greenwood, J. J. D., and Harvey, P. H. (1991). The relationship between abundance and body size in British birds. *Nature*, **351**(May), 312–313. [77](#)
- Nijhawan, S., Mitapo, I., Pulu, J., Carbone, C., and Rowcliffe, J. M. (2019). Does polymorphism make Asiatic golden cat the most adaptable predator in Eastern Himalayas? *Ecology*, **100**(10), e02768. [2](#), [11](#)
- Novak, M., Wolf, C., Coblenz, K. E., and Shepard, I. D. (2017). Quantifying predator dependence in the functional response of generalist predators. *Ecology Letters*, **20**(6), 761–769. [44](#)
- Nuismer, S. L. (2017). *Introduction to coevolutionary theory*. Macmillan Higher Education. [5](#), [9](#), [81](#)
- Nuismer, S. L. and Doebeli, M. (2004). Genetic correlations and the coevolutionary dynamics of three-species systems. *Evolution*, **58**(6), 1165–1177. [34](#), [36](#)
- Nuismer, S. L., Thompson, J. N., and Gomulkiewicz, R. (2000). Coevolutionary clines across selection mosaics. *Evolution*, **54**(4), 1102–1115. [10](#)
- Nuismer, S. L., Doebeli, M., and Browning, D. (2005). The coevolutionary dynamics of antagonistic interactions mediated by quantitative traits with evolving variances. *Evolution*, **59**(10), 2073–2082. [10](#), [33](#), [34](#), [36](#)
- Nuismer, S. L., Gomulkiewicz, R., and Ridenhour, B. J. (2010). When is correlation coevolution? *The American Naturalist*, **175**(5), 525–537. [9](#), [13](#)
- Odling-Smee, F. J., Laland, K. N., and Feldman, M. W. (2003). *Niche construction : the neglected process in evolution*. Princeton University Press. [40](#)
- Odling-Smee, F. J., Erwin, D. H., Palkovacs, E. P., Feldman, M. W., and Laland, K. N. (2013). Niche construction theory; A practical guide for ecologists. *The Quarterly Review of Biology*, **88**(1), 3–28. [6](#), [40](#), [42](#), [45](#), [56](#)

- Patel, S., Cortez, M. H., and Schreiber, S. J. (2018). Partitioning the effects of eco-evolutionary feedbacks on community stability. *American Naturalist*, **191**(3), 381–394. [2](#)
- Pawar, S. (2015). The Role of Body Size Variation in Community Assembly. In *Advances in Ecological Research*, volume 52, pages 201–248. Academic Press Inc. [63](#), [77](#)
- Persson, L. (1985). Asymmetrical Competition: Are Larger Animals Competitively Superior? *The American Naturalist*, **126**(2), 261–266. [62](#), [63](#)
- Peters, R. H. (1983). *The Ecological Implications of Body Size*. Cambridge University Press. [6](#), [77](#), [78](#)
- Pfennig, K. S. and Pfennig, D. W. (2009). Character displacement: ecological and reproductive responses to a common evolutionary problem. *Quarterly Review of Biology*, **84**(3), 253–276. [10](#)
- Post, D. M. and Palkovacs, E. P. (2009). Eco-evolutionary feedbacks in community and ecosystem ecology: interactions between the ecological theatre and the evolutionary play. *Philosophical transactions of the Royal Society of London. Series B, Biological sciences*, **364**(1523), 1629–40. [40](#), [56](#), [60](#)
- Roughgarden, J. (1972). Evolution of Niche Width. *The American Naturalist*, **106**(952), 683–718. [4](#), [13](#), [17](#), [32](#), [42](#), [72](#)
- Roughgarden, J. (1976). Resource partitioning among competing species -A coevolutionary approach. *Theoretical Population Biology*, **9**(3), 388–424. [10](#)
- Rueffler, C., Egas, M., and Metz, J. A. J. (2006). Evolutionary predictions should be based on individual-level traits. *The American Naturalist*, **168**(5), E148–E162. [59](#)
- Sasaki, A. and Dieckmann, U. (2011). Oligomorphic dynamics for analyzing the quantitative genetics of adaptive speciation. *Journal of Mathematical Biology*, **63**(4), 601–635. [4](#)
- Schmidt-Nielsen, K. (1984). *Scaling: Why is Animal Size so Important*. Cambridge University Press. [6](#), [63](#), [78](#), [79](#)

- Schreiber, S. J., Patel, S., and TerHorst, C. P. (2018). Evolution as a coexistence mechanism: does genetic architecture matter? *The American Naturalist*, **191**(3), 407–420. [10](#)
- Schulter, D. and McPhail, J. D. (1992). Ecological Character Displacement and Speciation in Sticklebacks. *The American Naturalist*, **140**(1), 85–1098. [2](#), [11](#), [33](#)
- Senthilnathan, A. and Gavrillets, S. (2021). Ecological consequences of intraspecific variation in coevolutionary systems. *The American Naturalist*, **197**(1), 1–17. [42](#), [48](#), [72](#)
- Simberloff, D. and Boecklen, W. (1981). Santa Rosalia Reconsidered: Size Ratios and Competition. *Evolution*, **35**(6), 1206. [2](#)
- Simmons, G. F. and Krantz, S. G. (2007). *Differential Equations : Theory, Technique, and Practice*. McGraw-Hill Higher Education. [81](#), [110](#)
- Skalski, G. T. and Gilliam, J. F. (2001). Functional responses with predator interference: Viable alternatives to the Holling type II model. *Ecology*, **82**(11), 3083–3092. [44](#)
- Slatkin, M. (1979). Frequency- and density-dependent selection on a quantitative character. *Genetics*, **93**(3), 755–771. [42](#)
- Slatkin, M. (1980). Ecological Character Displacement. *Ecology*, **61**(1), 163–177. [2](#), [4](#), [10](#)
- Smith, P. J., Francis, R. I., and McVeagh, M. (1991). Loss of genetic diversity due to fishing pressure. *Fisheries Research*, **10**(3-4), 309–316. [2](#), [11](#)
- Soler, J. J., Martínez, J. G., Soler, M., and Møller, A. P. (2001). Coevolutionary interactions in a host-parasite system. *Ecology Letters*, **4**(5), 470–476. [9](#), [20](#)
- Start, D. (2019). Individual and Population Differences Shape Species Interactions and Natural Selection. *The American Naturalist*, **194**(2), 183–193. [1](#), [11](#), [33](#)
- Start, D. and Gilbert, B. (2019). Trait variation across biological scales shapes community structure and ecosystem function. *Ecology*, **100**(9), e02769. [1](#), [11](#), [33](#)
- Summers, K., McKeon, S., Sellars, J., Keusenkothen, M., Morris, J., Gloeckner, D., Pressley, C., Price, B., and Snow, H. (2003). Parasitic exploitation as an engine of diversity. [2](#), [11](#)

- Svardal, H., Rueffler, C., and Doebeli, M. (2014). Organismal complexity and the potential for evolutionary diversification. *Evolution*, **68**(11), 3248–3259. [40](#), [57](#)
- Taper, M. L. and Case, T. J. (1992). Models of Character Displacement and the Theoretical Robustness of Taxon Cycles. *Evolution*, **46**(2), 317–333. [3](#), [4](#), [10](#)
- ten Brink, H., de Roos, A. M., and Dieckmann, U. (2019). The Evolutionary Ecology of Metamorphosis. *The American Naturalist*, **193**(5), E116–E131. [4](#)
- Thompson, J. N. (1994). *The coevolutionary process*. University of Chicago Press. [5](#), [9](#)
- Thompson, J. N. (2005). *The geographic mosaic of coevolution*. University of Chicago Press. [10](#)
- Tilman, D. (1982). Space as a Resource, Disturbance, and Community. In *Resource competition and community structure*, pages 205–227. Princeton University Press. [78](#)
- Tilman, D. (1985). The Resource-Ratio Hypothesis of Plant Succession. *The American Naturalist*, **125**(6), 827–852. [40](#)
- Tilman, D. (1999). The ecological consequences of changes in biodiversity: A search for general principles. *Ecology*, **80**(5), 1455–1474. [39](#)
- Toju, H. and Sota, T. (2006). Adaptive divergence of scaling relationships mediates the arms race between a weevil and its host plant. *Biology Letters*, **2**(4), 539–542. [9](#), [20](#)
- Travis, J., Leips, J., and Rodd, F. H. (2013). Evolution in Population Parameters: Density-Dependent Selection or Density-Dependent Fitness? *The American Naturalist*, **181**(S1), S9–S20. [40](#)
- Troost, T. A., van Dam, J. A., Kooi, B. W., and Tuenter, E. (2009). Seasonality, Climate Cycles and Body Size Evolution. *Mathematical Modelling of Natural Phenomena*, **4**(6), 135–155. [4](#)
- Turchin, P. (2003). *Complex Population Dynamics: A Theoretical/Empirical Synthesis (MPB-35)*. Princeton University Press. [34](#), [43](#)

- Uchmański, J. (1985). Differentiation and frequency distributions of body weights in plants and animals. [63](#), [77](#)
- Van Buskirk, J., Cereghetti, E., and Hess, J. S. (2017). Is bigger really better? Relative and absolute body size influence individual growth rate under competition. *Ecology and Evolution*, **7**(11), 3745–3750. [63](#)
- Van der Putten, W. H., Bardgett, R. D., Bever, J. D., Bezemer, T. M., Casper, B. B., Fukami, T., Kardol, P., Klironomos, J. N., Kulmatiski, A., Schweitzer, J. A., Suding, K. N., Van de Voorde, T. F., and Wardle, D. A. (2013). Plant-soil feedbacks: The past, the present and future challenges. *Journal of Ecology*, **101**(2), 265–276. [59](#)
- Van Nuland, M. E., Bailey, J. K., and Schweitzer, J. A. (2017). Divergent plant-soil feedbacks could alter future elevation ranges and ecosystem dynamics. *Nature Ecology and Evolution*, **1**(6), 1–10. [50](#)
- Vandermeer, J. (2008). The niche construction paradigm in ecological time. *Ecological Modelling*, **214**(2-4), 385–390. [57](#)
- Vellend, M. (2006). The consequences of genetic diversity in competitive communities. *Ecology*, **87**(2), 304–311. [1](#), [11](#)
- Violle, C., Enquist, B. J., McGill, B. J., Jiang, L., Albert, C. H., Hulshof, C., Jung, V., and Messier, J. (2012). The return of the variance: Intraspecific variability in community ecology. *Trends in Ecology and Evolution*, **27**(4), 244–252. [1](#), [10](#)
- Vitale, C. and Best, A. (2019). The paradox of tolerance: Parasite extinction due to the evolution of host defence. *Journal of Theoretical Biology*, **474**, 78–87. [4](#)
- Vitousek, P. M., Walker, L. R., Whiteaker, L. D., Mueller-Dombois, D., and Matson, P. A. (1987). Biological invasion by *Myrica faya* alters ecosystem development in Hawaii. *Science*, **238**(4828), 802–804. [40](#), [50](#)
- Walsh, B. and Lynch, M. (2018). Maintenance of Quantitative Genetic Variation. In *Evolution and Selection of Quantitative Traits*, volume 1. Oxford University Press. [11](#)

- Ware, I. M., Fitzpatrick, C. R., Senthilnathan, A., Bayliss, S. L. J., Beals, K. K., Mueller, L. O., Summers, J. L., Wooliver, R. C., Van Nuland, M. E., Kinnison, M. T., Palkovacs, E. P., Schweitzer, J. A., Bailey, J. K., Summers, J. L., Schweitzer, J. A., Mueller, L. O., Bayliss, S. L. J., Beals, K. K., Van Nuland, M. E., Bailey, J. K., Kinnison, M. T., and Palkovacs, E. P. (2019). Feedbacks link ecosystem ecology and evolution across spatial and temporal scales: Empirical evidence and future directions. *Functional Ecology*, **33**(1), 31–42. [2](#), [6](#), [40](#), [60](#)
- Waxman, D. and Gavrillets, S. (2005). 20 Questions on Adaptive Dynamics. *Journal of Evolutionary Biology*, **18**(5), 1139–1154. [12](#)
- West, G. B., Brown, J. H., and Enquist, B. J. (1997). A general model for the origin of allometric scaling laws in biology. *Science*, **276**(5309), 122–126. [77](#)
- White, E. P., Ernest, S. K., Kerkhoff, A. J., and Enquist, B. J. (2007). Relationships between body size and abundance in ecology. *Trends in Ecology and Evolution*, **22**(6), 323–330. [76](#)
- Winkelmann, K., Genner, M. J., Takahashi, T., and Rüber, L. (2014). Competition-driven speciation in cichlid fish. *Nature Communications*, **5**(1), 1–8. [11](#)
- Wright, J. P., Ames, G. M., and Mitchell, R. M. (2016). The more things change, the more they stay the same? When is trait variability important for stability of ecosystem function in a changing environment. *Philosophical Transactions of the Royal Society B: Biological Sciences*, **371**(1694), 20150272. [1](#)
- Xu, N., Delius, G. W., Zhang, L., Thygesen, U. H., and Andersen, K. H. (2021). Spatial drivers of instability in marine size-spectrum ecosystems. *Journal of Theoretical Biology*, **517**, 110631. [78](#)
- Yamamichi, M., Lyberger, K., and Patel, S. (2019). Antagonistic coevolution between multiple quantitative traits: Matching dynamics can arise from difference interactions. *Population Ecology*, **61**(4), 362–370. [35](#), [84](#)
- Yamamichi, M., Kyogoku, D., Iritani, R., Kobayashi, K., Takahashi, Y., Tsurui-Sato, K., Yamawo, A., Dobata, S., Tsuji, K., and Kondoh, M. (2020). Intraspecific Adaptation

Load: A Mechanism for Species Coexistence. *Trends in Ecology and Evolution*, **35**(10), 897–907. [60](#)

Yoder, J. B. and Nuismer, S. L. (2010). When Does Coevolution Promote Diversification? *The American Naturalist*, **176**(6), 802–817. [2](#), [10](#), [13](#), [22](#), [27](#), [35](#)

Zu, J. and Wang, J. (2013). Adaptive evolution of attack ability promotes the evolutionary branching of predator species. *Theoretical Population Biology*, **89**, 12–23. [4](#), [12](#)

Appendices

A Appendix for Chapter 2

A.1 Analytical solution

Competition

Based on mutual invasibility analysis, we can derive conditions for each species to grow from low density in a population of the other species. In the absence of the species 1, species 2 will reach the equilibrium of the one species model (equation (2.2)). If $\sigma_{s2}^2 > \sigma_{c2}^2$, the trait distribution at the single-species equilibrium will be

$$\phi_2^*(y) = \frac{\sigma_{s2}}{\sigma_{c2}} \frac{K_2}{\sqrt{2\pi(\sigma_{s2}^2 - \sigma_{c2}^2)}} \exp\left(-\frac{(y - \theta_2)^2}{2(\sigma_{s2}^2 - \sigma_{c2}^2)}\right).$$

Else, it would be a delta function at θ_2 . Invasion criteria for species 1 in that case turns out to be identical to the population dynamics model (*i.e.*, $\alpha_{12} < K_1/K_2$).

For species i to coexist with species j, atleast individuals of some trait x needs to survive when $\phi_1(x) \approx 0$ and $\phi_2(y) = \phi_1^*(y)$. From equation (2.4),

$$\phi_1(x) \left(1 - \frac{\alpha_{12} \int \exp\left(-\frac{(x-z)^2}{2\sigma_{c12}^2}\right) \phi_2^*(z) dz}{K_1 e^{-\frac{(x-\theta_1)^2}{2\sigma_{s1}^2}}} \right) > 0$$

$$\Rightarrow f(x) = \alpha_{12} \frac{\sigma_{s2}}{\sigma_{c2}} \frac{\sigma_{c12}}{\sqrt{\sigma_{s2}^2 - \sigma_{c2}^2 + \sigma_{c12}^2}} \frac{K_2}{K_1} \exp\left(\frac{(x - \theta_1)^2}{2\sigma_{s1}^2} - \frac{(x - \theta_2)^2}{2(\sigma_{s2}^2 - \sigma_{c2}^2 + \sigma_{c12}^2)}\right) < 1$$

If $\sigma_{c2}^2 - \sigma_{c12}^2 \geq \sigma_{s2}^2 - \sigma_{s1}^2$ (inequality $\mathcal{A}$), then $\lim_{x \rightarrow \pm\infty} f(x) = 0$ and the inequality holds for some x . Else, $\lim_{x \rightarrow \pm\infty} f(x) = \infty$ and the condition holds only if $\min f(x) < 1$. This gives the invasion criteria for species 1,

$$\alpha_{12} \frac{\sigma_{s2}}{\sigma_{c2}} \frac{\sigma_{c12}}{\sqrt{\sigma_{s2}^2 - \sigma_{s1}^2 + \sigma_{c12}^2}} \frac{K_2}{K_1} \exp\left(-\frac{(\theta_1 - \theta_2)^2}{2((\sigma_{s2}^2 - \sigma_{s1}^2) - (\sigma_{c2}^2 - \sigma_{c12}^2))}\right) < 1 \quad (\text{A1})$$

Since the system is symmetric, a similar analysis yields the invasion criteria for species 2.

Exploiter-victim

For exploiter to coexist with the victim, the exploiter should be able to grow from low density in a population of victim. In the absence of the exploiter and strong competition among victims ($\sigma_{s1}^2 > \sigma_{c1}^2$), the victim trait distribution will be

$$\phi_1^*(x) = \frac{\sigma_{s1}}{\sigma_{c1}} \frac{K_1}{\sqrt{2\pi(\sigma_{s1}^2 - \sigma_{c1}^2)}} \exp\left(-\frac{(x - \theta_1)^2}{2(\sigma_{s1}^2 - \sigma_{c1}^2)}\right).$$

From equation (2.8), the condition for coexistence is,

$$\begin{aligned} & \left(\beta \int \exp\left(-\frac{(y-z)^2}{2\sigma_d^2}\right) \phi_1^*(z) dz \right) - \gamma > 0, \\ \Rightarrow & \frac{\beta K_1 \sigma_{s1} \sigma_d}{\sigma_{c1} \sqrt{\sigma_{s1}^2 - \sigma_{c1}^2 + \sigma_d^2}} \exp\left(-\frac{(y-\delta)^2}{2(\sigma_{s1}^2 - \sigma_{c1}^2 + \sigma_d^2)}\right) > \gamma. \end{aligned}$$

This is a Gaussian function. Therefore for some trait y of the exploiter to coexist with the victim, the maximum value of the function should satisfy the condition. This gives the sufficient conditions for coexistence,

$$\frac{\beta K_1 \sigma_{s1} \sigma_d}{\sigma_{c1} \sqrt{\sigma_{s1}^2 - \sigma_{c1}^2 + \sigma_d^2}} > \gamma. \quad (\text{A2})$$

equation (A2) is the sufficient condition for coexistence when intraspecific variation is allowed to change over time, and intraspecific competition in victim is stronger than the stabilizing selection acting on them ($\sigma_{s1}^2 > \sigma_{c1}^2$). If $\sigma_{s1}^2 \leq \sigma_{c1}^2$, then the sufficient condition for coexistence is $\beta > \gamma$ which is identical to the model with no intraspecific variation.

Mutualism

To obtain conditions for the two species to exist at finite population sizes at equilibrium, we find a sequence of trait distributions for species 1 and species 2 which is the upper bound for the dynamics. The equilibrium population will be finite only if the sequence of population sizes obtained from the sequence of trait distributions converges. If $\phi_1(x, 0) \approx 0$ and $\phi_2(y, 0) \approx 0$, $\phi_1(x, \tau)$ will be smaller than its one species equilibrium trait distribution.

Assume $\sigma_{si}^2 > \sigma_{ci}^2$. Then, for some small τ_1 ,

$$\phi_1(x, \tau_1) \leq \frac{r_1 K_1 \sigma_{s1}}{\sigma_{c1} \sqrt{2\pi(\sigma_{s1}^2 - \sigma_{c1}^2)}} \exp\left(-\frac{(x - \theta_1)^2}{2(\sigma_{s1}^2 - \sigma_{c1}^2)}\right) = f_{1,1}(x).$$

Mutualistic benefit for species 2 at time τ is bounded by

$$\begin{aligned} B_2 \int \exp\left(-\frac{(y - z)^2}{2\sigma_{m2}^2}\right) f_{1,1}(z) dz &= \frac{B_2 r_1 K_1 \sigma_{s1} \sigma_{m2}}{\sigma_{c1} \sqrt{\sigma_{s1}^2 - \sigma_{c1}^2 + \sigma_{m2}^2}} \exp\left(-\frac{(y - \theta_1)^2}{2(\sigma_{s1}^2 - \sigma_{c1}^2 + \sigma_{m2}^2)}\right) \\ &\leq \frac{B_2 K_1 \sigma_{s1} \sigma_{m2}}{\sigma_{c1} \sqrt{\sigma_{s1}^2 - \sigma_{c1}^2 + \sigma_{m2}^2}} r_1 \end{aligned}$$

Therefore for $\tau_i < \tau_{i+1}$,

$$\phi_2(y, \tau_2) \leq \left(r_2 + \frac{B_2 K_1 \sigma_{s1} \sigma_{m2}}{\sigma_{c1} \sqrt{\sigma_{s1}^2 - \sigma_{c1}^2 + \sigma_{m2}^2}} r_1\right) \frac{K_2 \sigma_{s2}}{\sigma_{c2} \sqrt{2\pi(\sigma_{s2}^2 - \sigma_{c2}^2)}} \exp\left(-\frac{(y - \theta_2)^2}{2(\sigma_{s2}^2 - \sigma_{c2}^2)}\right) = f_{2,1}(y),$$

$$\begin{aligned} \phi_1(x, \tau_3) &\leq \left(r_1 + \frac{B_1 K_2 \sigma_{s2} \sigma_{m2}}{\sigma_{c2} \sqrt{\sigma_{s2}^2 - \sigma_{c2}^2 + \sigma_{m2}^2}} \left(r_2 + \frac{B_2 K_1 \sigma_{s1} \sigma_{m2}}{\sigma_{c1} \sqrt{\sigma_{s1}^2 - \sigma_{c1}^2 + \sigma_{m2}^2}} r_1\right)\right) \\ &\quad \frac{K_1 \sigma_{s1}}{\sigma_{c1} \sqrt{2\pi(\sigma_{s1}^2 - \sigma_{c1}^2)}} \exp\left(-\frac{(x - \theta_1)^2}{2(\sigma_{s1}^2 - \sigma_{c1}^2)}\right) = f_{1,2}(x), \end{aligned}$$

$$\begin{aligned} \phi_2(y, \tau_4) &\leq \left(r_2 + \frac{B_2 K_1 \sigma_{s1} \sigma_{m2}}{\sigma_{c1} \sqrt{\sigma_{s1}^2 - \sigma_{c1}^2 + \sigma_{m2}^2}} \left(r_1 + \frac{B_1 K_2 \sigma_{s2} \sigma_{m2}}{\sigma_{c2} \sqrt{\sigma_{s2}^2 - \sigma_{c2}^2 + \sigma_{m2}^2}} \left(r_2 + \dots \right. \right. \right. \\ &\quad \left. \left. \left. \dots \frac{B_2 K_1 \sigma_{s1} \sigma_{m2}}{\sigma_{c1} \sqrt{\sigma_{s1}^2 - \sigma_{c1}^2 + \sigma_{m2}^2}} r_1\right)\right)\right) \frac{K_2 \sigma_{s2}}{\sigma_{c2} \sqrt{2\pi(\sigma_{s2}^2 - \sigma_{c2}^2)}} \exp\left(-\frac{(y - \theta_2)^2}{2(\sigma_{s2}^2 - \sigma_{c2}^2)}\right) = f_{2,2}(y), \end{aligned}$$

$$\begin{aligned} \phi_1(x, \tau_5) &\leq \left(r_1 + \frac{B_1 K_2 \sigma_{s2} \sigma_{m2}}{\sigma_{c2} \sqrt{\sigma_{s2}^2 - \sigma_{c2}^2 + \sigma_{m2}^2}} \left(r_2 + \frac{B_2 K_1 \sigma_{s1} \sigma_{m2}}{\sigma_{c1} \sqrt{\sigma_{s1}^2 - \sigma_{c1}^2 + \sigma_{m2}^2}} \left(r_1 + \dots \right. \right. \right. \\ &\quad \left. \left. \left. \dots \frac{B_1 K_2 \sigma_{s2} \sigma_{m2}}{\sigma_{c2} \sqrt{\sigma_{s2}^2 - \sigma_{c2}^2 + \sigma_{m2}^2}} \left(r_2 + \frac{B_2 K_1 \sigma_{s1} \sigma_{m2}}{\sigma_{c1} \sqrt{\sigma_{s1}^2 - \sigma_{c1}^2 + \sigma_{m2}^2}} r_1\right)\right)\right)\right) \frac{K_1 \sigma_{s1} \exp\left(-\frac{(x - \theta_1)^2}{2(\sigma_{s1}^2 - \sigma_{c1}^2)}\right)}{\sigma_{c1} \sqrt{2\pi(\sigma_{s1}^2 - \sigma_{c1}^2)}} \\ &= f_{1,3}(x), \end{aligned}$$

The time series of population sizes of the two species are bounded by the sequences $N_{1,i} = \int f_{1,i}(x)dx$ and $N_{2,i} = \int f_{2,i}(y)dy$ respectively. From the sequence of trait distributions, we can infer that

$$N_{1,1} = \frac{\sigma_{s1}}{\sigma_{c1}} K_1 r_1,$$

$$N_{1,i} = \frac{\sigma_{s1}}{\sigma_{c1}} K_1 \left(r_1 + \frac{B_1 K_2 \sigma_{s2} \sigma_{m2}}{\sigma_{c2} \sqrt{\sigma_{s2}^2 - \sigma_{c2}^2 + \sigma_{m2}^2}} \left(r_2 + \frac{B_2 K_1 \sigma_{s1} \sigma_{m2}}{\sigma_{c1} \sqrt{\sigma_{s1}^2 - \sigma_{c1}^2 + \sigma_{m2}^2}} \frac{\sigma_{c1}}{\sigma_{s1}} \frac{N_{1,i-1}}{K_1} \right) \right), \forall i \geq 2.$$

The equilibrium population size is finite only if these sequences converge. Real-valued sequences converge if and only if they are Cauchy.

$$\begin{aligned} N_{1,i} - N_{1,i-1} = & \frac{\sigma_{s1}}{\sigma_{c1}} K_1 \left(r_1 + \frac{B_1 K_2 \sigma_{s2} \sigma_{m2}}{\sigma_{c2} \sqrt{\sigma_{s2}^2 - \sigma_{c2}^2 + \sigma_{m2}^2}} r_2 + \dots \right. \\ & \left. \dots \left(\frac{B_1 B_2 K_1 K_2 \sigma_{s1} \sigma_{s2} \sigma_{m1} \sigma_{m2}}{\sigma_{c1} \sigma_{c2} \sqrt{(\sigma_{s1}^2 - \sigma_{c1}^2 + \sigma_{m2}^2)(\sigma_{s2}^2 - \sigma_{c2}^2 + \sigma_{m1}^2)}} - 1 \right) \frac{\sigma_{c1}}{\sigma_{s1}} \frac{N_{1,i-1}}{K_1} \right). \end{aligned}$$

Since $N_{1,i}$ is an increasing sequence, for it to be Cauchy,

$$\frac{B_1 B_2 K_1 K_2 \sigma_{s1} \sigma_{s2} \sigma_{m1} \sigma_{m2}}{\sigma_{c1} \sigma_{c2} \sqrt{(\sigma_{s1}^2 - \sigma_{c1}^2 + \sigma_{m2}^2)(\sigma_{s2}^2 - \sigma_{c2}^2 + \sigma_{m1}^2)}} < 1 \quad (\text{A3})$$

The condition for convergence of $N_{2,i}$ is identical. Equation (A3) is a necessary condition for the equilibrium population of the two species to be finite.

A.2 Numerical Method

We solved our dynamic equations using a finite difference method (Simmons and Krantz, 2007, Kreyszig, 2009, Doebeli, 2011). Specifically, we first truncate the phenotype space to a finite interval $[-\lambda, \lambda]$. λ needs to be chosen such that ϕ and $\partial\phi/\partial\tau$ are small at the boundaries. Second, we partition the truncated phenotype space into intervals of length l . This gives a partition of size $N = 2\lceil\lambda/l\rceil$. We can now discretise the dynamic equations. For example, in the two species competition model (2.4),

$$\frac{\partial\phi_1(x_i, \tau)}{\partial\tau} \approx r_1 \phi_1(x_i, \tau) \left(1 - \frac{\int C_{11}(x_i, z) \phi_1(z, \tau) dz + \alpha_{12} \int C_{12}(x_i, z) \phi_2(z, \tau) dz}{\kappa_1(x_i)} \right), \quad (\text{A4a})$$

$$\frac{\partial \phi_2(y_i, \tau)}{\partial \tau} \approx r_2 \phi_2(y_i, \tau) \left(1 - \frac{\alpha_{21} \int C_{21}(y_i, z) \phi_1(z, \tau) dz + \int C_{22}(y_i, z) \phi_2(z, \tau) dz}{\kappa_2(y_i)} \right). \quad (\text{A4b})$$

Here, the integrals are over $[-\lambda, \lambda]$ and $1 \leq i \leq N$. Finally, the integrals can be computed using trapezoidal rule over the same partition and the derivatives can be approximated using the Euler forward method. This leads to the iterative equations:

$$\begin{aligned} \phi_{1,\tau+\Delta}(x_i) \approx \phi_{1,\tau}(x_i) & \left(1 + r_1 \Delta - \frac{r_1 l \Delta}{2\kappa_2(x_i)} \sum_{k=1}^{N-1} \left(C_{11}(x_i, z_k) \phi_{1,\tau}(z_k) + C_{11}(x_i, z_{k+1}) \phi_{1,\tau}(z_{k+1}) \right. \right. \\ & \left. \left. + \alpha_{12} \left(C_{12}(x_i, z_k) \phi_{2,\tau}(z_k) + C_{12}(x_i, z_{k+1}) \phi_{2,\tau}(z_{k+1}) \right) \right) \right), \end{aligned} \quad (\text{A5a})$$

$$\begin{aligned} \phi_{2,\tau+\Delta}(y_i) \approx \phi_{2,\tau}(y_i) & \left(1 + r_2 \Delta - \frac{r_2 l \Delta}{2\kappa_2(y_i)} \sum_{k=1}^{N-1} \left(\alpha_{21} \left(C_{21}(y_i, z_k) \phi_{1,\tau}(z_k) + C_{21}(y_i, z_{k+1}) \phi_{1,\tau}(z_{k+1}) \right) \right. \right. \\ & \left. \left. + C_{22}(y_i, z_k) \phi_{2,\tau}(z_k) + C_{22}(y_i, z_{k+1}) \phi_{2,\tau}(z_{k+1}) \right) \right). \end{aligned} \quad (\text{A5b})$$

For small Δ and l , these equations converge. Numerical convergence can be made faster by using adaptive time steps. This is achieved by halving the time steps and reiterating one step whenever population densities becomes negative. Time steps can also be occasionally doubled if population densities remains positive over several time steps.

Setting $\alpha_{12} = \alpha_{21} = 0$ reduces the two-species competition model to two independent single-species model (equation (2.2)). We then confirmed that the equilibrium phenotypic distribution we obtain using the numerical method matches the analytical solution of the single-species model for different parameter choices.

This same method was applied to the exploiter-victim and mutualism models.

A.3 Effects of evolving trait distributions on the equilibrium mean trait values

When the optimal trait values of the two coevolving species are same, then at equilibrium the mean traits are predicted to be at the optimum regardless of whether we model the trait variances as constant (as it is done in mean-centered models) or changing through time (as

it is done here). However if the optimum values differ between the species, the two methods can give different predictions for the means. For example, in the model of victim-exploiter interactions subject to Gaussian stabilizing selection considered in Gavrillets (1997), if the phenotypic variance in the exploiter is larger than that in the victim, the system evolves to an equilibrium. At this equilibrium, the mean value of the exploiter is at the optimum for the victim while that of the victim is displaced from its optimum by the value equal to the difference between the two optima, i.e. $\theta_1 - \theta_2$. Let us set the exploiter's optimum $\theta_2 = 0$ without loss of generality. Then in Gavrillets (1997)'s model, the predicted trait values at equilibrium as a function of the victim's optimum θ_1 are given by the dashed lines in Figure A1. Under comparable parameter values, the corresponding equilibria in our model are shown by filled circles. One can see that with evolving variances the victim is at its optimum while the exploiter is very close to the victim.

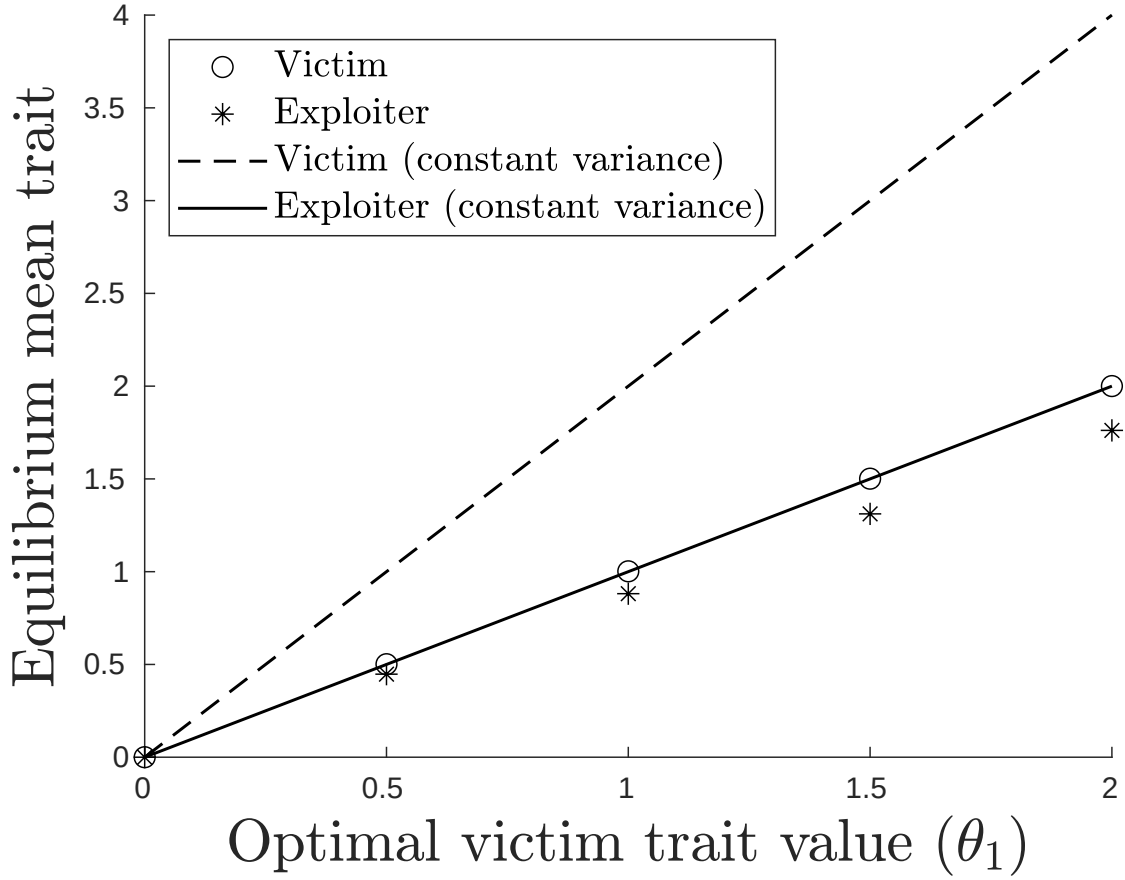


Figure A1: The effects of evolving trait distributions on the equilibrium mean traits in the exploiter-victim system. The dashed and solid lines corresponds to equilibrium values of in [Gavrilets \(1997\)](#)'s model which assumed constant variances. Note that the solid line (the diagonal) is also the optimum value for the victim. The points correspond to equilibria in our model. The initial conditions for numerical simulations used the same phenotypic distribution $\phi(z) = 0.1 \exp(-10z^2)$ for both species. Other parameters: $\theta_2 = 0, \alpha = 1, \beta = 1, \zeta = 1, \gamma = 1, \sigma_{c1} = 0.5, \sigma_{c2} = 0.5, \sigma_{s1} = 0.71, \sigma_{s2} = 0.71, K_1 = 1, K_2 = 1$.

Vita

Athmanathan was born in Tiruchirappali, Tamil Nadu, India and attended school in Chennai until graduating from higher secondary education. He moved to Bengaluru in 2011 for his undergraduate education at the Indian Institute of Science (IISc). He graduated with a Bachelor of Science degree in 2015 with a major in Mathematics. During his undergraduate studies he was part of the Theoretical Ecology and Evolution (TEE) lab at the Center for Ecological Sciences, IISc. He also studied formal logic as a summer student in Chennai Mathematical Institute. In the fall of 2015, he moved to The University of Tennessee at Knoxville, USA to attend graduate school in Ecology and Evolutionary Biology. While in graduate school he obtained a Master of Science degree in Mathematics. He completed his PhD in Ecology with the mentorship of Dr. Sergey Gavrilets in the summer of 2021.