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Journal

Journal of Experimental Biology, 215(6)

ISSN

0022-0949

Author

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Publication Date

2012-03-15

DOI

10.1242/jeb.059022

Peer reviewed

The Journal of Experimental Biology 215, 948-961

doi:10.1242/jeb.059022

REVIEW

Integrating mechanistic organism–environment interactions into the basic theory of community and evolutionary ecology

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Accepted 22 August 2011

Summary

This paper presents an overview of how mechanistic knowledge of organism–environment interactions, including biomechanical interactions of heat, mass and momentum transfer, can be integrated into basic theoretical population biology through mechanistic functional responses that quantitatively describe how organisms respond to their physical environment. Integrating such functional responses into simple community and microevolutionary models allows scaling up of the organism-level understanding from biomechanics both ecologically and temporally. For community models, Holling-type functional responses for predator–prey interactions provide a classic example of the functional response affecting qualitative model dynamics, and recent efforts are expanding analogous models to incorporate environmental influences such as temperature. For evolutionary models, mechanistic functional responses dependent on the environment can serve as fitness functions in both quantitative genetic and game theoretic frameworks, especially those concerning function-valued traits. I present a novel comparison of a mechanistic fitness function based on thermal performance curves to a commonly used generic fitness function, which quantitatively differ in their predictions for response to environmental change. A variety of examples illustrate how mechanistic functional responses enhance model connections to biologically relevant traits and processes as well as environmental conditions and therefore have the potential to link theoretical and empirical studies. Sensitivity analysis of such models can provide biologically relevant insight into which parameters and processes are important to community and evolutionary responses to environmental change such as climate change, which can inform conservation management aimed at protecting response capacity. Overall, the distillation of detailed knowledge or organism–environment interactions into mechanistic functional responses in simple population biology models provides a framework for integrating biomechanics and ecology that allows both tractability and generality.

Key words: mechanistic models, species interactions, quantitative genetics, game theory, functional response, fitness function.

Introduction

As described by Denny and Helmuth (Denny and Helmuth, 2009), integrating biomechanics and ecology represents a scaling up from the biomechanics of individual organisms and their interaction with their environment to the ecology of populations and communities. A full population biology understanding requires extension of biomechanics in two dimensions. In the ecological dimension, scaling up from populations to interacting species provides a community- and ecosystem-level understanding rooted in mechanistic knowledge. In the temporal dimension, scaling up from ecological to evolutionary time provides an evolutionary understanding also rooted in mechanistic knowledge of organisms' interactions with their environment.

This pursuit of understanding the interaction between patterns across multiple scales is central to the field of ecology and evolutionary biology and its application to management, and theory and models play a key role in cross-scale extrapolation (Levin, 1992). Models serve a number of purposes in population biology (and in general), with the goal of a model determining whether it is constructed as a general, phenomenological or detailed mechanistic model. Levins translates the range of possible model structures into a trade-off between generality, realism and precision (Levins, 1966). Here, the distinction between precision and realism is akin to that between precision and accuracy: quantitatively exact and consistent values *versus* closeness to the actual (real-world) value. Within this trade-off, Levins suggests that one of the three properties is typically sacrificed in favor of the other two, and the two focal properties dictate the potential model contribution (Levins, 1966). In particular, (1) focusing on realism and precision at the expense of generality leads to the potential for testable predictions for a specific situation, (2) focusing on generality and realism at the expense of precision leads to the capacity for qualitative predictions about different possible outcomes, and (3) focusing on generality and precision at the expense of realism leads to null-type models that allow exploration of how the incremental addition of realism affects overall dynamics.

Much of the basic theory of population biology focuses on the generality side of this trade-off, where the goal might be to formalize a hypothesis about the factors driving a given outcome in a mathematical framework to rigorously test and quantify the underlying logical expectations (i.e. mathematics as 'a way of thinking clearly') (May, 2004). In contrast, biomechanics, including that integrated with ecology, is rooted in a bottom-up approach focusing on detailed mechanisms (Denny and Helmuth, 2009) and therefore gravitates towards the realism side of this trade-off. A number of modeling frameworks, described elsewhere in this special issue of *The Journal of Experimental Biology*, follow the biomechanics approach of scaling up from mechanistic

physiological [e.g. dynamic energy budget models (Nisbet et al., 2012)] or individual-level [e.g. scale-transition and complexity theory (Benedetti-Cecchi et al., 2012; Van de Koppel et al., 2012)] dynamics to patterns on ecological scales.

In addition to building up directly from the mechanistic knowledge itself and therefore starting with realism, another approach to integrating biomechanics or analogous mechanistic knowledge with theoretical population biology is to start with more general models from basic theory and incrementally add realism based on the mechanistic understanding. Specifically, one approach is to distill mechanistic understanding of organism–environment interactions into a functional response that can fit into the framework of more generic and tractable models of theoretical population biology. Mechanistic functional responses, in particular those that quantitatively describe how an organism or population responds to an environmental variable, represent a key component to the scaling-up process inherent to integrating biomechanics and ecology (Denny and Benedetti-Cecchi, in review). This approach takes advantage of the biological realism that stems from biomechanics and allows integration of that mechanistic, detailed understanding into a tractable framework where central dynamics and drivers can more easily be discerned.

Here, I describe how mechanistic functional responses that describe organism–environment interactions can be integrated into basic community and evolutionary ecological models. While others have proposed the use of mechanistic functional responses in models of community and evolutionary ecology (typically treated separately) [(e.g. Arnold, 1983; Schoener, 1986; Denny and Benedetti-Cecchi, in review) see also additional citations throughout this manuscript], the goal of this manuscript is to provide an accessible outline of how to approach this task in both subdisciplines. Therefore, this manuscript serves as an overview rather than a comprehensive review. In addition, a specific focus here is on incorporating mechanistic knowledge pertaining to how organisms interact with their environment, which includes but is not limited to the biomechanical interactions of heat, momentum and mass transfer (Denny and Helmuth, 2009).

This functional response-based approach can apply to additional modeling frameworks from basic theoretical population biology not covered here such as non-evolutionary single-population dynamics, including stage- or physiologically structured dynamics. Also, scaling up in space receives only superficial attention here. The focus here on community and evolutionary ecology is intended to provide an illustration of the general approach centered on the scaling up of biomechanics ecologically and temporally. In addition, it complements the population models presented elsewhere in this special issue, such as the structured population models of Nisbet et al. and Madin and Connolly (Nisbet et al., 2012; Madin and Connolly, 2012) as well as the models focused on scaling up community dynamics in space discussed by Benedetti-Cecchi et al. and Van de Koppel et al. (Benedetti-Cecchi et al., 2012; Van de Koppel et al., 2012).

In order to start with the simplest possible models within community and evolutionary ecology, below I describe the potential integration of biomechanics into basic non-evolutionary multi-species models and microevolutionary single-species models through mechanistic functional responses, defined more specifically in each context. For each I provide the general frameworks and illustrative examples, from a classic example for community ecology to a novel example for evolutionary ecology. Throughout I highlight how this approach mechanistically adds an environmental dimension to population dynamics to provide insight into when mechanism matters to the qualitative or quantitative outcome, connect models to biologically relevant, empirically measurable parameters, and allow exploration of dynamical responses to different environmental conditions. Finally, I outline the potential for this understanding to inform conservation biology through a mechanistic understanding of the central drivers of ecological and evolutionary response to environmental change.

Community ecology

In this section, I first describe a classic, textbook example of how the shape of the functional response affects the dynamics of interacting species in a basic predator–prey model. Then I discuss ways that mechanistic knowledge of organism–environment interactions can factor into the functional forms of predator–prey models, with a Boltzmann factor-based approach of incorporating temperature effects on predator–prey dynamics as an example. Finally, I discuss how this approach might apply more broadly to general models of interacting species and highlight connections to the frameworks of trait-mediated interactions and ecosystem engineering.

Classic functional response example in predator–prey interactions

Perhaps the most common use of the term ‘functional response’ in ecology is in predator–prey models. The basic model structure of this classic example follows a specialist predator and its prey, whose densities are denoted P and H, respectively. Here, the functional response is the per-predator rate of prey capture $f(H)$ as it depends on prey population density H. In addition to experiencing predation at a total rate $f(H)P$, the prey have a constant population growth rate r and experience density-dependent mortality with carrying capacity K. The predators convert the predation into reproduction with an efficiency factor of c and experience density-independent mortality at a rate k. The overall dynamics are:

$$\frac{\mathrm{d}H}{\mathrm{d}t} = rH\left(1-\frac{H}{K}\right) - f(H)P\ , \tag{1}$$

$$\frac{\mathrm{d}P}{\mathrm{d}t} = cf(H)P - kP\ . \tag{2}$$

Holling (Holling, 1959) posited three possible functional responses for the predation rate $f(H)$. Type I predation, the simplest possible representation, employs a linear rate:

$$f(H) = bH\ , \tag{3}$$

(Fig. 1A), which can be considered the mass-action case where the rate of prey capture increases in direct proportion to the prey density (Berryman, 1992). Type II predation takes a slightly more mechanistic approach by dividing the predation time into the proportion of time spent searching S as compared to handling the prey. If the rate of prey capture per unit search time is bH, then the overall capture rate per unit time is bHS. In addition, if the handling time per capture is β, then the total time spent handling is βbHS. For time to be appropriately scaled (i.e. S represents a proportion of one unit of time), then one unit of time is the sum of the search time S and handling time βbHS, or $1=S+\beta bHS$. Rearranging this in terms of the time spent searching $S=(1+\beta bH)^{-1}$ and defining $d=\beta b$, the overall capture rate per unit time bHS is the type II functional response:

$$f(H) = \frac{bH}{1+dH}\ , \tag{4}$$

where predation saturates for high prey density (Fig. 1B). Incorporating handling time is one of many possible mechanistic

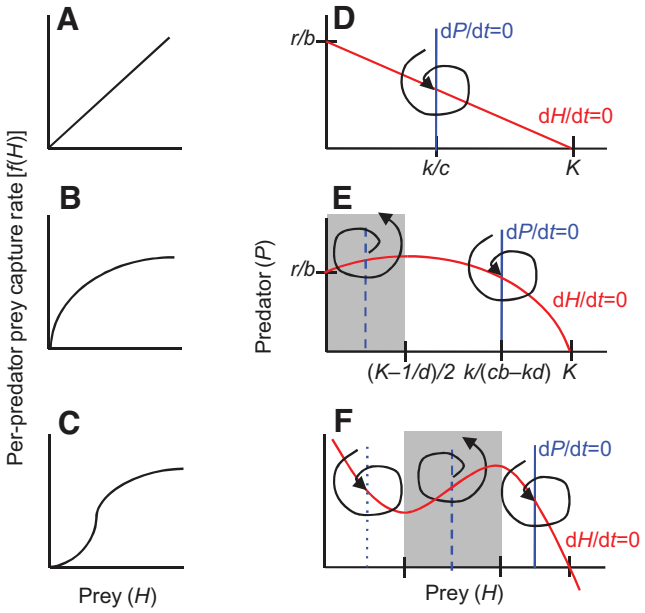

Fig. 1. The three Holling functional responses describing predation (A–C, illustrating Eqns 3–5) and the resulting possible outcomes for predator–prey dynamics based on phase plane analysis (D–F) (Hastings, 1997). A and D, type I; B and E, type II; F and G, type III. D–F illustrate the isoclines (dynamics in Eqns 1 and 2 set to zero, or no change in time), with the prey isocline ($1/H\times dH/dt=0$) in red and the predator isocline ($1/P\times dP/dt=0$) in blue. In E and F the dashed and solid blue lines indicate different predator isoclines for the different possible outcomes depending on the parameter values. Intersection points indicate equilibrium predator and prey densities, and spiraling arrows indicate dynamics in the neighborhood of those points. In addition, gray shaded areas indicate regions of instability, and non-shaded areas indicate regions of stability for the internal (predator and prey at non-zero values) equilibrium. Note that the predator isocline depends only on prey density (i.e. $dP/dt=0$ with $P\neq 0$ depends only on H in Eqn 2); therefore, this line (the blue lines in D–F) defines the equilibrium prey density.

approaches for a saturating functional response (Spalinger and Hobbs, 1992). Finally, type III predation expands on this saturation with a slower increase in predation at low prey densities, as might occur with a limited number of hiding places, such that:

$$f(H)=\frac{bH^2}{1+dH^2} \quad (5)$$

(Fig. 1C).

These three functional responses lead to qualitatively different model outcomes. For type I, the system is always stable (predators and prey haven damped oscillations towards a fixed point; Fig. 1D). For type II, either unstable or stable dynamics is possible depending on the parameter values, with stabilization at parameter values that yield higher equilibrium prey densities (Fig. 1E). For type III, again either unstable or stable dynamics is possible depending on parameter values, but in this case stabilization occurs at parameter values that yield lower or higher equilibrium prey densities with unstable dynamics (e.g. limit cycles) occurring at intermediate prey densities (Fig. 1E).

Adding mechanistic functional responses to predator–prey models and Boltzmann factor example

While the example of the three Holling functional responses demonstrates the potential importance of the function shape, the type I–III functional responses are relatively phenomenological (i.e. have a general functional form that resembles empirical patterns, as opposed to the functional form being mechanistically derived from first principles). Mechanistic knowledge can factor into determining which predator–prey functional response might apply among these three or the variety of other possibilities such as those based on predator interference (e.g. Skalski and Gilliam, 2001), herbivore grazing dynamics (e.g. Spalinger and Hobbs, 1992) and ratio (P/H) dependence (e.g. Berryman, 1992). While the saturating type II functional response is the most commonly used in basic community models, data from predator–prey dynamics more often have a better fit to functional responses that are based on predator interference, where the functional response depends on predator as well as prey density (Spalinger and Hobbs, 1992).

In addition, Holling-type functional responses are narrowly concerned with the effects of prey density on the rate of prey capture. Mechanistic consideration of organism–environment interactions, including those relevant to biomechanics, requires extension of this framework to incorporate a broader class of mechanistic response functions in which the independent variable can be any aspect of the environment (either biotic or abiotic). For example, Vasseur and McCann incorporate temperature (a key environmental characteristic) into a predator–prey model with Holling type II (i.e. saturating) predation rate based on first principles of thermodynamics (Vasseur and McCann, 2005). According to a relationship first defined by Boltzmann, the rate of any chemical reaction is governed by the ratio of its activation energy, E_i, to the energy available from molecular collisions, κT, where κ is Boltzmann's constant and T is absolute temperature. Specifically, the rate is proportional to $e^{-E_i/(\kappa T)}$; the higher the temperature, the faster the rate. This mechanistically determined proportionality is the basis for the Q_{10} effects traditionally measured by physiologists.

The Boltzmann relationship provides a basis for Vasseur and McCann (Vasseur and McCann, 2005) to scale the rates of prey production r, predator biomass gain from ingestion $(1-\delta)J$, and predator respiration k:

$$r=f_r a_r(T_0)m_H^{-0.25}e^{E_r(T-T_0)/(\kappa TT_0)}, \quad (6)$$

$$(1-\delta)J=f_J a_J(T_0)m_P^{-0.25}e^{E_J(T-T_0)/(\kappa TT_0)}, \quad (7)$$

$$k=a_k(T_0)m_P^{-0.25}e^{E_k(T-T_0)/(\kappa TT_0)}, \quad (8)$$

where r and k factor into the prey and predator dynamics as in Eqns 1 and 2. Here a_r, a_J and a_k are the maximum sustainable rates of each process (measured at representative body temperature T_0), and f_r and f_J are the fractions of these rates realized in nature. m_H and m_P are the masses of individual predators and prey, respectively. Note that in each case, rate depends on $m_i^{-0.25}$, a scaling commonly found in nature (Schmidt-Nielsen, 1984) and much debated in the physiological literature (Glazier, 2005). δ is the fraction of biomass lost during ingestion and digestion. With these temperature-dependent rates defined, Vasseur and McCann (Vasseur and McCann, 2005) frame the predator–prey functional response $f(H)$ in terms of the maximum prey ingestion rate J, the predation half-saturation constant H_0, and f_e, the fraction of captured prey biomass that the predator actually ingests:

$$f(H)=\frac{JH}{f_e(H+H_0)}. \quad (9)$$

In addition, the authors express the predator efficiency factor in Eqn 2 as $c=f_e(1-\delta)$. While this formulation adds many new

parameters, including some that could be consolidated if symbolic mathematical analysis were the only consideration, the mechanistic basis allows for more straightforward biological interpretation of the parameters and parameterization from empirical data. In addition, the model structure maintains enough simplicity to remain tractable, in particular allowing analytical expressions for the dynamical change in each of the prey and predator with temperature (dH/dT and dP/dT).

Because the basic dynamical structure of the model remains the same, no new dynamics emerge from the inclusion of this temperature dependence, unlike the Holling-type functional responses described above. Rather, it adds a new dimension to the model by allowing exploration of how temperature will affect which of the possible dynamical outcomes (stable node or cyclical behavior as illustrated in Fig. 1E) occurs. The mechanistic basis of this added dimension provides a concrete foundation for exploring the interactive effects of temperature dependence in three different aspects of the predator–prey dynamics (prey production, predator ingestion and predator respiration), the complexity of which would limit any phenomenological consideration. In particular, from their analysis of the model, Vasseur and McCann find that increasing temperatures destabilize predator–prey dynamics, lead to decreases in prey biomass, and lead to greater changes in predator than prey biomass (Vasseur and McCann, 2005).

Rall and colleagues employ a similar Boltzmann factor-based approach of exploring the effect of temperature changes on the predator–prey model in Eqns 1 and 2, but with a linear, type I Holling functional response for the predation rate and temperature dependence in the prey carrying capacity K as well as the prey growth and the predator ingestion rates (Rall et al., 2010). Again, the overall equilibrium dynamics remain the same (stable node as in Fig. 1D), but the inclusion of complex temperature effects on multiple processes allows exploration of this new dimension and reveals that increasing temperature leads to decreasing equilibrium interaction strength between the predator and prey. Both examples illustrate the potential for the inclusion of mechanistic functional responses that describe organism–environment interactions in relatively simple dynamical models to elucidate how environmental change might affect the dynamics of the interacting species. In addition, both studies exemplify the potential for connection to empirical data given mechanistically based, biologically relevant parameters: Rall and colleagues parameterize their model based on terrestrial arthropod experiments (Rall et al., 2010), and Vasseur and McCann provide a variety of parameterizations based on vertebrate ectotherm and marine invertebrate systems (Vasseur and McCann, 2005).

Community models more generally

Beyond predator–prey dynamics, a generic representation of two interacting species N_1 and N_2 given per-capita growth rates F_1 and F_2 is:

$$\frac{dN_1}{dt} = N_1 F_1(N_1, N_2) , \quad (10)$$

$$\frac{dN_2}{dt} = N_2 F_2(N_1, N_2) . \quad (11)$$

Species interactions imply that the per-capita growth rate for a given species (F_i) will depend on the population density of the other species (N_j); it is typically possible to break down the population growth into the intraspecific term $f_i(N_i)$ and the interspecific interaction term $g_i(N_i,N_j)$, where $F_i(N_i,N_j)=f_i(N_i)+g_j(N_i,N_j)$. The simplest possible (Lotka–Volterra) formulation for the species interaction functional response is linear with each species, i.e. the interaction term is $g_i(N_j)=a_{ij}N_j$, where the sign of the constant a_{ij} for each species depends on whether the interaction is predation (opposite signs for a_{12} and a_{21}), competition (both negative) or mutualism (both positive). The field of theoretical ecology has continually added biological realism to this structure in order to better understand community dynamics, including extension to 3–6 species to explore 'community modules' (e.g. trophic chains, apparent competition, intraguild predation) (Holt, 1997). While limiting the number of interacting species to a tractable set will always limit realism, it can provide insight into a key, strongly interacting subset of species or guilds (Holt, 1997).

As with the predator–prey case, a mechanistic understanding can inform the functional form of both interspecific [$g_i(N_i,N_j)$] and intraspecific [$f_i(N_i)$] dynamics. The general idea of building mechanistic functional responses for interspecific interactions overlaps with the framework of trait-mediated interactions, which extends the typical consideration of population density-dependent species interactions to also include the role of individual traits, and the traits that influence species interactions can be morphological, behavioral or life history (reviewed by Bolker et al., 2003). The theory of trait-mediated interactions indicates the importance of the shape of functional responses to population dynamics, but such shapes are rarely measured to construct empirically driven models (Bolker et al., 2003).

Mechanistic functional responses hold the potential to fill this crucial knowledge gap to connect theory and data as well as extend this theory to incorporate the interaction between organisms and their environment. As mentioned above, mechanistic functional responses that incorporate dependence on the abiotic environment add another dimension to allow understanding of how environmental drivers affect the dynamical outcome. For example, the Boltzmann factor framework described in the context of predator–prey models above (e.g. Vasseur and McCann, 2005) has the potential to provide a general approach for incorporating temperature dependence into the dynamics of interacting species. In addition, dispersal kernels as they depend on abiotic conditions (e.g. wind or currents, discussed in more detail in 'Game theory and dispersal evolution example' below) represent a type of functional response based on organism–environment interactions that can provide insight into the co-existence of competing species (e.g. Berkley et al., 2010).

In addition to the abiotic environment, mechanistic functional responses can depend on the biotic environment, in which case the potential for novel dynamics is possible and indicates when mechanism matters to the qualitative outcome. For example, in the Rietkerk and van de Koppel plant resource model (Rietkerk and van de Koppel, 1997), incorporating the response of resource dynamics to plant density due to indirect environmental effects changes the model outcome. Specifically, they (Rietkerk and van de Koppel, 1997) model plant P and resource R density dynamics using a chemostat-type model, where the resource input rate is $R_{in}(P)$, the resource uptake by plants is $c(R)$, the resource loss (outflow) rate is $r(P)$ and the plant growth rate is $g(R)$, such that:

$$\frac{dR}{dt} = R_{in}(P) - c(R)P - r(P)R , \quad (12)$$

$$\frac{dP}{dt} = g(R)P . \quad (13)$$

If the resource outflow $r(P)$ is a constant independent of plant density P (Fig. 2A), as is typical in the basic model formulation, the model has one possible stable state for a given set of parameters (Fig. 2C). However, if the resource outflow $r(P)$ monotonically

declines with plant density P at a decreasing rate (Fig. 2B), a biotic environmental response function supported from empirical data that occurs due to plant density-dependent runoff and erosion, then two alternative stable states are possible for some parameter values (Fig. 2D). This possibility of alternative stable states leads to hysteresis, where the path a system follows under environmental change depends on the direction of the environmental change (black arrows in Fig. 2D). Such dynamical outcomes have profound implications for the management of a system (e.g. in the case of plant resource dynamics, management of grazing or nutrient runoff) due to the potential for a small change in the environment (e.g. increased herbivory) to lead to a subsequent change in state (e.g. plant collapse) that cannot be reversed with a reversion to the original environmental condition.

More generally, consideration of dynamical resource dependence on the biotic environment falls within the framework of ecosystem engineering (Jones et al., 1994). In other words, the functional form of the response of the abiotic environment to the density of an ecosystem engineer can be considered a type of environmental functional response. The theory of ecosystem engineering indicates that explicit consideration of organism-dependent resource dynamics, and therefore biotic environmental functional responses for resources, is most important when the organism–resource interaction dynamics occur on larger spatial and temporal scales than the intra- and/or inter-specific dynamics (Hastings et al., 2007). Models that incorporate this type of environmental functional response demonstrate the importance of ecosystem engineering to ecological dynamics such as population persistence, invasive species spread and spatial dynamics (reviewed by Hastings et al., 2007).

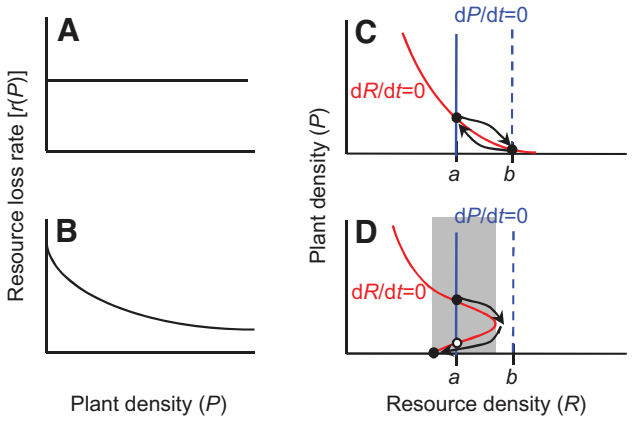

Fig. 2. Chemostat model that ignores (A, plant-independent outflow) or accounts for (B, plant-dependent outflow) resource loss rate dependence on plant density, and the resulting plant resource dynamics (C and D, respectively). (C,D) The isoclines (dynamics in Eqns 12 and 13 set to zero, or no change in time), with the resource isocline (dR/dt=0) in red and the plant isocline (dP/dt=0) in blue. Filled circles indicate locally stable equilibria and open circles indicate locally unstable equilibria, and the shaded region in D indicates the region of resource density with alternative stable states. Black arrows illustrate the outcome for an environment shifting between points a (equilibrium resource density at the blue solid line) and b (equilibrium resource density at the blue dashed line): given plant-independent outflow, if the system starts at a, shifts to b, and returns to a, the system state (resource and plant densities) will shift between the indicated equilibria points, returning to the original state (C). However, given plant-dependent outflow as illustrated in B, if the system starts at a within the region with alternative stable states and the internal equilibrium point (solid point with P>0 and R>0 in D) then shifts to b, the plant and resource densities can pass beyond a threshold such that the system shifts to the resource-only equilibrium point (solid point with P=0 and R>0) and a return to the conditions in a will not lead to a return to the original state. [Adapted from Rietkerk and Van de Koppel (Rietkerk and Van de Koppel, 1997) fig. 1A, fig. 2B and fig. 3B.]

Overall, different functions for intraspecific and interspecific dynamics readily affect the outcome of community ecology models, and a mechanistic approach to developing such functions can both connect the parameters to biologically relevant and measurable processes and provide an indication of the appropriate functional response for a given system (Schoener, 1986). Focusing on functional traits as they affect performance under different environmental conditions has the potential to enhance the quantitative and predictive content of community ecology theory (McGill et al., 2006). Furthermore, the incorporation of a more bottom-up, mechanistic approach in multispecies models is in line with recent suggestions to build community ecology up from the processes that drive community-level patterns (rather than the top-down approach of starting with the patterns themselves), which Roughgarden (Roughgarden, 2009) and Vellend (Vellend, 2010) posit has a greater potential to lead to a general theory of community ecology.

Evolutionary ecology

In the community models described above, mechanistic knowledge about organisms' interactions with their environment could factor into an environment-dependent functional response for a variety of dynamics, from intraspecific dynamics such as population growth rate to interaction dynamics such as predation rate. For a single-species model of evolutionary change, it is the mechanistic functional response in terms of the environment-dependent population growth rate (i.e. function that describes how the population growth rate responds to the environment) that holds particular relevance as it provides a metric of fitness. If this function appropriately describes both population growth and fitness, then it can serve to couple ecological and evolutionary dynamics.

Below, I describe two broad frameworks for integrating such a mechanistic functional response into evolutionary dynamics: quantitative genetics (with two modeling approaches described) and game theory. This is not intended to be an exhaustive accounting of all models relevant to these frameworks but rather an example illustration of a handful of commonly used models. The focus here is on the microevolutionary dynamics of changing gene frequencies within a population rather than the macroevolutionary processes of speciation and extinction because microevolution has greater ecological relevance and is therefore more relevant to this special issue. In addition, for simplicity, this section focuses on single-species models; for an integrative review of both population genetic and game theoretic frameworks in the context of co-evolutionary questions of evolutionary ecology, see Abrams (Abrams, 2001) and Day (Day, 2005).

In addition to describing the generic mathematical formulations with an indication of which term can be an environment-dependent functional response in all cases, for the first quantitative genetics modeling approach I discuss existing theoretical constructs (specifically, the phenotype–performance–fitness framework and function-valued traits) that are particularly relevant to the integration of biomechanics. For the second quantitative genetics modeling approach I analyze an example based on the evolution of thermal tolerance in a changing environment in order to illustrate the mechanistic functional response approach. Finally, for the game theory approach I use a discussion of dispersal, a topic often explored in both game theory and biomechanics, to exemplify how a

biomechanics-based understanding might shift the focus of evolutionary models and allow better integration of models with data.

Quantitative genetics

Population genetic models follow gene frequencies over time as they depend on relative fitness, which encompasses both survivorship and reproductive success. While the simplest possible model construction of density-independent fitness leads to genetic dynamics independent of population dynamics (i.e. gene frequency dynamics do not depend on population size), the average population fitness (relative frequency of each genotype multiplied by its fitness, then summed over all possible genotypes) could be considered a metric of the population growth rate, thus coupling the population (ecological) dynamics to the genetic (evolutionary) dynamics. In addition, consideration of density-dependent influences on fitness inevitably leads to population size-dependent genotype fitness and therefore necessitates fully coupled population and genetic dynamics (Day, 2005).

A mechanistic understanding of organism–environment interactions can underlie the quantification of fitness in population genetic models. For example, a physiological understanding of organism response to the physical environment and how that affects investment in growth, maintenance and reproduction can allow measurement of the fitness for the traits relevant to that process. This measurement of fitness for different traits could directly provide the parameters for a Mendelian (i.e. discrete phenotypes) model of population genetics, such as a one-locus, two-allele model where fitness is defined separately for each genotype as a distinct parameter.

However, traits relevant to mechanistic knowledge (including biomechanics-based knowledge), such as morphological and physiological traits, are typically quantitative genetic traits: they can take on a continuum of values that depend on multiple contributing genes as well as the environment. One approach to modeling such traits is to follow the continuous probability density function $p_z(z)$ of phenotype z [i.e. $p_z(z)\mathrm{d}z$ describes the proportion of the population with phenotypes between z and $z+\mathrm{d}z$] and/or the probability density $p_y(y)$ of genotype y, where both the genotypes and the phenotypes are continuous variables (the mathematical description of the link between the two is described in more detail below; the genetic probability density can also be considered the 'breeding value' distribution). Then the fitness function $W_z(z)$ that drives adaptive dynamics is also a continuous function that describes how fitness depends on phenotype z; note that here and throughout I use y and z subscripts to the probability density and fitness functions to indicate whether they are functions of genotypes or phenotypes, respectively. Often, fitness functions have phenomenological functional forms for generality and tractability (e.g. general representations of stabilizing, disruptive or directional selection) (Lande and Arnold, 1983) or occasionally are determined from fits to empirical data (e.g. Schluter, 1988), but they can also be built up from mechanistic knowledge.

As a demonstration of where fitness functions integrate into models of coupled population and genetic dynamics, including example fitness functions to provide a more concrete illustration, I use two different models: a discrete-time, diploid model with sexual reproduction and a continuous-time, haploid model with asexual reproduction. Both are built on the 'infinitesimal alleles' assumption that a large number of unlinked loci contribute additively to the overall genotype y. Among the many possible population genetic frameworks [see, for example, Denny and Dowd in this issue for another quantitative genetic modeling framework (Denny and Dowd, 2012)], the two described here hold appeal for integrating biomechanics-based knowledge because they provide flexibility for the definition of the fitness function.

Discrete-time sexual model, the phenotype performance–fitness model, and function-valued traits

The discrete-time, diploid, sexual model follows the genotype probability density $p_t(y)$ at each point in time t assuming non-overlapping generations. The model dynamics consist of (1) applying selection and (2) mating and inheritance of genotypes, in either order. Selection occurs on the phenotype (z), but the genotype (y) is what is inherited in the 'transmission function' that describes the transmission of parental genotypes to the offspring.

Determining how selection affects the genotype distribution requires multiplying the genotype probability density $p_t(y)$ by the probability of having a particular phenotype given a particular genotype $q(z|y)$, then multiplying by the phenotype-dependent fitness $W_z(z)$ and integrating over all phenotypes. In other words, the genotype-dependent fitness is $W_y(y)=\int W_z(z)q(z|y)\mathrm{d}z$ (assuming one selection event per generation). For phenotypes randomly distributed around the genotypes with environmental variance V_E, the phenotype–genotype probability function is the Gaussian function $q(z|y)=\exp[-(z-y)^2/(2V_E)]/\sqrt{(2\pi V_E)}$ (i.e. given each genotype y, the probability density of phenotype z around that genotype is normal with a mean y and variance V_E). Applying selection then requires multiplying the fitness by the genotype probability density $[W_y(y)p_t(y)]$ and normalizing such that the resulting distribution remains a probability density function. The normalization factor, the fitness of each genotype multiplied by its frequency integrated over all genotypes $[\int W_y(y)p_t(y)\mathrm{d}y]$, is also the mean fitness of the population W_t.

Under random mating, the probability that two individuals with genotypes y_1 and y_2 mate is their encounter probability as described by the product of their frequencies in the population $p_t(y_1)p_t(y_2)$. For inheritance, assuming each offspring genotype is the average of the genotypes it inherits from its parents, the distribution of possible offspring genotypes has an expected value of the parental genetic mean $(y_1+y_2)/2$ and a variance of half of the parental genetic variance $V_G/2$. The half factor comes in because of the averaging:

$$\mathrm{Var}\left(\frac{Y_1+Y_2}{2}\right)=\frac{1}{4}\mathrm{Var}(Y_1+Y_2)=\frac{1}{2}\mathrm{Var}(Y)\,, \qquad (14)$$

for independently and identically distributed Y_1 and Y_2; note that this half factor appears in the transmission function, not the final offspring genetic distribution. Therefore, for inheritance under the infinitesimal model, the parent–offspring genotype transmission function is:

$$L(y\,|\,y_1,y_2)=\frac{1}{\sqrt{\pi V_G}}\exp(-(y-(y_1+y_2)/2)^2/V_G)\,; \qquad (15)$$

if random mutation is expected to increase the genetic variance in each generation, then one approach to account for this is to add the amount by which that happens V_M to the variance of the transmission function. The determination of the genetic variance V_G in this function is discussed in more detail below.

Combining mating and inheritance, the offspring genotype probability density is the product of the mating probability of parents y_1 and y_2 and the transmission function, integrating over all mating pairs. With selection [to arrive at the post-selection distribution $p_t^*(y)$] then mating [applied to the selected population

to arrive at the next generation's distribution $p_{t+1}(y)$], the full dynamics are:

$$p_t^*(y) = \frac{W_y(y)\,p_t(y)}{\int W_y(y)\,p_t(y)\,\mathrm{d}y}\ , \tag{16}$$

$$p_{t+1}(y) = \frac{1}{\sqrt{\pi V_G}} \iint p_t^*(y_1)\,p_t^*(y_2)\exp\left(-\frac{(y-(y_1+y_2)/2)^2}{V_G}\right)\mathrm{d}y_1\mathrm{d}y_2 \tag{17}$$

(Slatkin, 1970; Turelli and Barton, 1994). Assuming fitness is measured in terms of population growth and is density independent, these genetic dynamics can be coupled with population dynamics where the population size N_t changes according to a per-generation, per-capita growth factor derived from mean fitness $\overline{W}_t=\int W_y(y)p_t(y)\mathrm{d}y$ and any density dependence $F(N_t)$:

$$N_{t+1} = \overline{W}_t N_t F(N_t) \tag{18}$$

(Slatkin, 1980). In cases where density-dependent or other demographic factors might affect the genetic as well as the population dynamics, one approach to determining the coupled population and genetic dynamics is to start with the dynamics of the population density distribution $n_t(y)$, where $n_t(y)\mathrm{d}y$ is the number of individuals with genotypes between y and $y+\mathrm{d}y$. Then the genetic distribution dynamics can be derived from $p_t(y)=n_t(y)/\int n_t(y)\mathrm{d}y$ and the population dynamics from $N_t=\int n_t(y)\mathrm{d}y$. Under the assumptions of weak selection, a Gaussian genetic distribution $\{p_t(y)=\exp[-(y-y_t)^2]/(2V_G)]/\sqrt{(2\pi V_E)}\}$, and a constant genetic variance (V_G), Eqns 16 and 17 reduce to the dynamics of the change in mean phenotype as the product of the genetic variance and the selection gradient:

$$\overline{z}_{t+1} - \overline{z}_t = \frac{V_G}{\overline{W}_t}\frac{\partial \overline{W}_t}{\partial \overline{z}_t} \tag{19}$$

(Lande, 1976). This framework can be extended to multiple co-evolving traits given the genetic variance–covariance matrix for the suite of traits (Lande, 1979). However, evolutionary ecologists might be interested in strong selection and evolving genetic variance. If the assumption of a normal genetic distribution still applies, one way to simplify Eqns 16 and 17 is to follow the genetic and phenotypic means, variances and covariances rather than the full distribution (e.g. Cavalli-Sforza and Feldman, 1976). This requires a fitness function that allows maintenance of the normal distribution, such as stabilizing selection for the optimal trait θ given fitness function width S as an inverse measure of selection strength: $W_z(z)=\exp[-(z-\theta)^2/2S]$. Also, using the parental genetic variance $V_{G,t}$ directly in the transmission function assumes no drift and no linkage (i.e. ignores recombination), which can affect model predictions (see Feldman and Cavalli-Sforza, 1981; Felsenstein, 1981). For alternative approaches to accounting for genetic variance evolution that employ the genetic variance at linkage equilibrium as the V_G term in the transmission function in Eqn 17, see Turelli and Barton (Turelli and Barton, 1994). For a more mechanistic fitness function that leads to departures from normality, Turelli and Barton (Turelli and Barton, 1994) provide computational tools for evaluating the full distribution dynamics in Eqns·16 and 17 (they also find that a normal distribution can provide a good approximation in a number of cases).

One approach to empirically measuring the selection gradient:

$$\frac{1}{\overline{W}_t}\frac{\partial \overline{W}_t}{\partial \overline{z}_t}\ , \tag{20}$$

for structural, physiological or behavioral traits, as originally detailed for directional selection by Arnold (Arnold, 1983) and extended to different types of selection functions (Arnold, 2003), is to break it down into two components: (1) the performance function that translates phenotype to performance and can be measured in the lab, and (2) the fitness function that translates performance to fitness and can be measured in the field. This phenotype–performance–fitness framework has provided a quantitative framework for measuring adaptation and its necessary components in the context of evolutionary physiology (Feder et al., 2000; Garland and Carter, 1994). The use of this performance-based approach in the context of multiple co-evolving traits allows exploration of evolutionary trade-offs between morphological, physiological and life history traits (e.g. trade-offs between growth, reproduction and escape performance in fish) (Ghalambor et al., 2003). Therefore, the phenotype–performance–fitness framework can provide an empirical basis to construct fitness functions and explore the evolution of physiological or biomechanics-related traits (e.g. those related to energetic investment and transfer). The advantage of using empirically derived fitness functions is the addition of biological realism (at the potential cost of analytic tractability) to determine quantitative expectations for evolution.

Another approach to determining more realistic fitness functions is to build up the functional form from a mechanistic understanding. Many fitness-optimization models, including those rooted in energy budgets and their constraints (Weiner, 1992), provide such a functional form. While these models typically focus on determining the optimal trait, using such a fitness function in the framework of Eqns 16–18 provides a dynamical understanding of the trajectory of evolutionary change, which will depend both on the selection strength (determined by the shape of the fitness function) and population traits such as genetic variance. The qualitative outcome of evolution towards the selected trait at a rate dependent on selection strength and heritable variation will not differ from phenomenological fitness functions; however, a mechanistic fitness function's basis in empirically measurable, biologically relevant parameters allows quantitative understanding of how specific biological processes affect the evolutionary trajectory and outcome.

In particular, the discrete time construct of Eqns 16–18 with density-independent and frequency-independent selection can employ a discrete time-based fitness metric such as the population growth factor λ or, if generation time is constant independent of evolutionary change, the net reproductive value R_0 [the expected lifetime reproductive output per individual (Kozlowski, 1993; Stearns, 1992)]. These metrics depend on the survivorship l_x and fecundity m_x at each age x: λ is the solution to the Euler equation:

$$1 = \sum_{x=0}^{X} l_x m_x \lambda^{-x}\ , \tag{21}$$

and

$$R_0 = \sum_{x=0}^{X} l_x m_x\ , \tag{22}$$

– or, for continuous age structure:

$$1 = \int_0^X l(x)m(x)\lambda^{-x}\,\mathrm{d}x\ , \tag{23}$$

and

$$R_0 = \int_0^X l(x)m(x)\,\mathrm{d}x\ , \tag{24}$$

given maximum age X (Kozlowski, 1993; Stearns, 1992). Then the survivorship and fecundity functions can depend on physiological trade-offs of energetic investment in growth, maintenance and reproduction (Chown and Gaston, 1997; Perrin and Sibly, 1993; Sibly and Calow, 1986; Stearns, 1992). Many existing fitness-maximizing models take this construct to predict optimal life history traits such as age or size at maturity, which then allows a mechanistic understanding of the biological drivers of the optimal trait (Kozlowski, 1992; Stearns, 1992). Integrating such fitness functions into Eqns 16–18 would similarly allow a mechanistic understanding of the biological drivers of the rate of evolutionary change as well as the expected evolutionary outcome. Note that a number of modeling studies integrate age-specific survivorship and fecundity into quantitative genetic models to explore the joint evolution of multiple co-evolving life history traits that trade off in their effects on survivorship and fecundity (e.g. Charlesworth, 1990; Lande, 1982), while the approach suggested here focuses on the evolution of a single trait as it depends on how it affects trade-offs in energetic investment in processes related to survivorship and fecundity [see also Coulson for a fully age-structured approach (Coulson et al., 2010)].

For example, Roff (Roff, 1984) presents an optimization model, applied to teleost fish, that separately considers survivorship and growth costs to investment in reproduction, which depends on size. The simplest possible case is that of semelparity: both growth and survivorship cease after reproduction, thus the maximum age is the age at maturity A. Fecundity depends on length-at-age L_x given fecundity coefficient a according to $m_x=aL_x^3$, and length depends on age according to the von Bertalanffy saturating growth function with asymptotic length L_∞ and growth exponent k: $L_x=L_\infty(1-e^{-kx})$. Given larval survivorship probability p and natural mortality rate M, the survivorship for a given age is $l_x=pe^{-Mx}$. Then two possible fitness metrics are the lifetime reproductive output:

$$R_0 = e^{-MA}paL_\infty^3(1-e^{-kA})^3\ , \tag{25}$$

and the population growth factor:

$$\lambda = (e^{-MA}paL_\infty^3(1-e^{-kA})^3)^{\frac{1}{A}}\ . \tag{26}$$

Roff uses these equations and analogous ones for iteroparity to describe how optimal age at maturity A (A that maximizes the fitness metric) depends on the additional life history parameters that describe fecundity, growth and survivorship (Roff, 1984), all of which, except p, are readily available for many teleost fish (note that, given the change in generation time with change in A, λ provides the more appropriate metric in this case). Similarly, using these fitness metrics as functions of age at maturity A in the framework of Eqns·16–18 (i.e. the phenotype z=A) can describe how the fecundity parameter a, growth parameters L_∞ and k, and survivorship parameters p and M affect dynamical evolutionary outcomes such as the rate of evolutionary change and the effect of mutation–selection balance. This simple, non-environmentally driven example illustrates how a fitness function built up from biological processes can allow insight into the dependence of evolutionary change on biologically relevant, empirically measurable parameters; a more biomechanical approach can similarly build a fitness function up from principles of energetic transfer between the processes relevant to growth, maintenance and fecundity and incorporate organism–environment interactions.

A mechanistic underpinning to the fitness function more readily allows insight into the evolutionary response to different environmental conditions (Arnold, 2003), especially novel environmental conditions where direct empirical measurements of selection or fitness are not available. For such an exploration, the fitness function will depend on both the phenotype and the environment. A natural extension of the phenotype–performance–fitness framework mentioned above is to include the influence of the biotic and abiotic environment (i.e. habitat) on phenotype and performance (Garland and Losos, 1994). For example, Kingsolver and Gomulkiewicz use thermal performance functions to explore the effect of environmental variation on the evolution of performance (Kingsolver and Gomulkiewicz, 2003). Their framework, readily connected to experimental caterpillar data, provides unique insight into whether selection acts on total growth, integrated performance or growth rates in particular environments. This approach is an example of following the evolution of 'function-valued traits', or traits that are a continuous function of an organism's status (e.g. age) or an environmental parameter (e.g. temperature); for the latter, following the evolution of the function is akin to following the evolution of phenotypic plasticity (Kingsolver et al., 2001). Modeling the evolution of function-valued traits can involve direct extension of the above-described approach to quantitative genetics (Kingsolver et al., 2001). In addition, new genetic and genomic tools to mechanistically explore fitness and plasticity (see Whitehead, 2012) are increasing the capacity to integrate this theory with data. An example of a model that incorporates such organism–environment interactions is provided in the next section on continuous-time asexual models.

Continuous-time asexual model and thermal tolerance example

For the analogous model of a clonal, asexual population in continuous time, the genotype distribution $p(y,t)$ is a continuous function of both genotype y and time t. Given continuous time, the phenotype (z)-dependent fitness $r_z(z)$ can be considered the growth rate (in comparison to the interpreting fitness $W_z(z)$ as a per-generation growth factor to link genetic and demographic dynamics in the discrete-time, diploid model above). As in the diploid model above, considering the phenotype-dependent fitness $r_z(z)$ and the probability of a phenotype z given a genotype y, $q(z|y)$ {e.g. $q(z|y)=\exp[-(z-y)^2/(2V_\mathrm{E})]/\sqrt{(2\pi V_\mathrm{E})}$ given phenotypes normally distributed around the genotypes with a random environmental variance V_E}, the genotype-dependent fitness is the product of these integrated overall phenotypes $r_y(y)=\int r_z(z)q(z|y)\mathrm{d}z$. In addition, as above, the mean fitness is the product of the genotype-dependent fitness and the genotype distribution integrated over all genotypes $\bar{r}(t)=\int r_y(y)p(y,t)\mathrm{d}y$. Given these definitions, the dynamics of the genotype distribution $p(y,t)$ depend on the difference between the rate of growth for a given genotype $r_y(y)$ and the overall population growth rate $\bar{r}(t)=\int r_y(y)p(y,t)\mathrm{d}y$:

$$\frac{\mathrm{d}p(y,t)}{\mathrm{d}t} = p(y,t)(r_y(y)-\bar{r}(t))\ . \tag{27}$$

The above model has the intuitive interpretation that genotypes with a fitness greater than the mean population fitness will increase in frequency and genotypes with a fitness less than the mean will

decrease in frequency; see Crow and Kimura for a detailed derivation (Crow and Kimura, 1970).

If fitness is defined as the asymptotic per-capita population growth rate, these genetic dynamics can connect to population $N(t)$ dynamics according to:

$$\frac{\mathrm{d}N(t)}{\mathrm{d}t} = \bar{r}(t)N(t) - F(N(t)) , \quad (28)$$

given any density-dependent mortality $F[N(t)]$, where $F[N(t)]$ is subtracted rather than multiplied here because of the potential for $\bar{r}(t)$ to be negative in a maladapted population. Under particular fitness functions that allow the maintenance of normality in the genetic distribution [e.g. given optimal trait θ, growth at that trait r_θ, and fitness function width S, $r_z(z)=r_\theta-(z-\theta)^2/2S$], it is straightforward to calculate the dynamics of the genetic mean $\bar{y}=\int yp(y)\mathrm{d}y$ and variance $V_G=\int(y-\bar{y})^2p(y)\mathrm{d}y$ (with the rate at which random mutation increases genetic variance V_M possibly added in) from Eqn 27, thus simplifying the model analysis (Lynch et al., 1991). However, a more mechanistic, empirically driven fitness function might not readily lend itself to such a simplification.

One extension to this model, relevant to the goal of integrating biomechanics and ecology to provide mechanistic predictions for population responses to novel environments (Denny and Helmuth, 2009), is to allow the selection to change in time, i.e. the fitness function becomes a function of time as well as phenotype $r_z(z,t)$. For example, Lynch et al. (Lynch et al., 1991) investigate this model with a changing optimal trait $\theta(t)$ in the fitness function (Fig. 3A):

$$r_z(z,t) = r_\theta - \frac{(z-\theta(t))^2}{2S} , \quad (29)$$

to determine the amount of environmental change that a population can keep up with [e.g. if the optimal trait is increasing linearly in time, or $\theta(t)=\theta_0+kt$, determining the threshold rate of change k beyond which mean population growth rate $\bar{r}(t)$ will eventually go negative]. This framework has been extended in a number of ways, such as accounting for sexual reproduction (Lynch and Lande, 1993), finite population size (Lynch and Lande, 1993) and phenotypic plasticity (Chevin et al., 2010).

Underlying these modeling efforts is often the more specific question of the rate of climate change a population can keep up with, in which case the changing environment typically can be considered in terms of changing temperature. For example, Huey and Kingsolver (Huey and Kingsolver, 1993) and Bonebrake and Mastrandrea (Bonebrake and Mastrandrea, 2010) apply the more generic models of Lynch and colleagues (Lynch and Lande, 1993; Lynch and Gabriel, 1987) to the evolution of thermal tolerance. These models retain symmetric fitness functions, such as the one specified above, for tractability. In reality, thermal performance curves tend to take a more asymmetrical form with a faster drop-off in population growth at higher temperatures than the temperature of peak performance, compared with lower temperatures than the peak (Angilletta et al., 2003).

Employing a more realistic thermal performance curve as the fitness function for dynamical explorations of adaptive responses to climate change exemplifies the incorporation of a mechanistic functional response that describes organism–environment interactions. One possible mathematical description of such thermal performance curves maintains θ as the optimal trait (temperature of peak performance) and S as the function width but adds a temperature-dependent factor with constants a and b to scale population growth by temperature (Fig. 3B):

$$r_z(z,t) = \left(1 - \frac{(\theta(t)-z)^2}{2S}\right) ae^{b\theta(t)} \quad (30)$$

[see, for example, Norberg's work (Norberg, 2004) based on an empirical motivation from phytoplankton thermal performance curves; but see Gilchrist (Gilchrist, 1995) for an alternative formulation of a thermal tolerance envelope-based fitness function that includes maximum and minimum temperature tolerance]. While multiple aspects of this curve might be considered to be under evolutionary pressure (Angilletta et al., 2002; Huey and Kingsolver, 1993), the above formulation considers the temperature

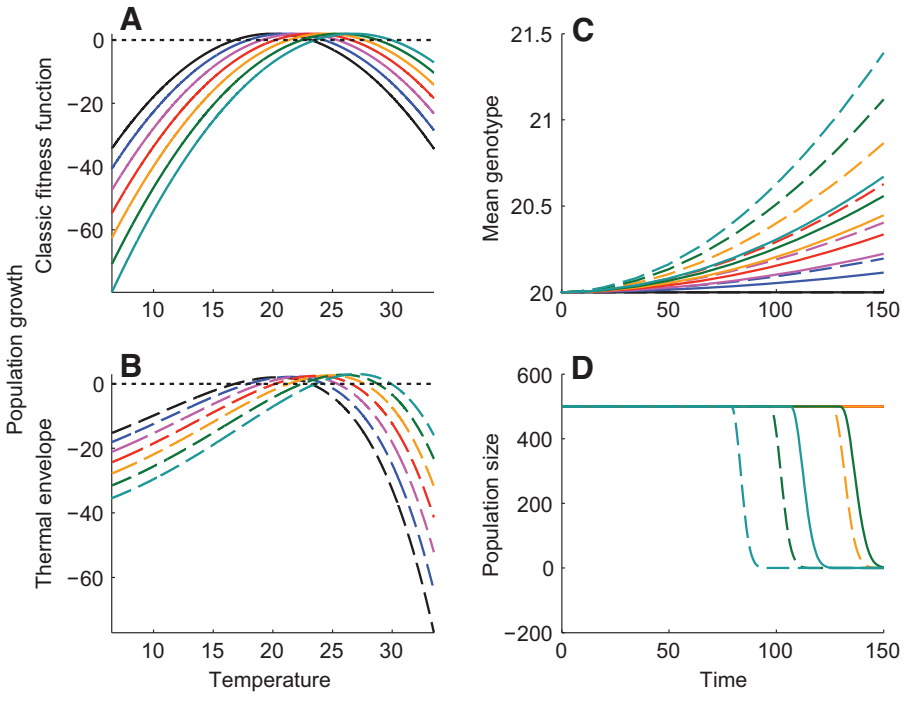

Fig. 3. Comparison of evolutionary change for a haploid population in continuous time with a traditional fitness function *versus* a physiologically based fitness function. (A,B) The two types of thermal response curves for an optimally adapted individual at the endpoint of each simulation, where different colors indicate simulations with different rates of environmental change in all panels (the black curve illustrates zero environmental change, and therefore the starting point for all curves). The physiologically based function (B, Eqn 30) is a thermal tolerance envelope, which accounts for increases in maximum population growth with increasing temperature and a faster drop-off in population growth with higher as compared with lower temperatures; these components are not in a more commonly used symmetric fitness function (A, Eqn 29). (C,D) The mean genotype and population size, respectively, over time with dashed lines indicating simulation using the physiologically based fitness function (Eqns 31–34) and solid lines indicating simulations with the traditionally used phenomenological fitness function. Parameter values: a=0.6, b=0.06, mutation variance $V_M=10^{-5}$, environmental variance V_E=0.005, fitness function width S=5, carrying capacity K=500, optimal trait θ_0=20, and rate of environmental change k ranges from 0 to 0.05 in steps of 0.0075.

of peak performance as the evolving trait for illustration (Baskett et al., 2009; Huey and Kingsolver, 1993) [but see Denny and Dowd, this volume, for an alternative approach (Denny and Dowd, 2012)]. This curve can represent a variety of trade-offs (Angilletta et al., 2003); for example, the peak can be considered the temperature for which an organism is optimally adapted, with the faster drop-off at higher temperatures due to thermal stress and the slower drop-off at lower temperatures due to over-investment of resources in thermal tolerance. The relatively phenomenological construct of Eqn 30 illustrates the approach of distilling detail-oriented mechanistic knowledge, in this case a physiological understanding of temperature-dependent dynamics, into a simple function that maintains model tractability but adds some elements of biological realism. In other words, as described in the Introduction, this approach adds incremental realism to the model (Eqns 27 and 28) in order to maintain the generality and precision that are the goals of basic population theory but also to provide insight into when mechanism matters and quantify how. Specifically, in addition to accounting for the asymmetry of the thermal performance curve, Eqn 30 accounts for the potential for faster growth at higher temperatures (as compared with a more traditional fitness function, Eqn 29, that does not include the $ae^{b\theta}$ factor). These two aspects of added realism have opposite effects on the potential for evolutionary response to increasing temperatures (θ): the faster drop-off at temperatures to the right of the peak will mean faster declines in population growth for maladapted populations and therefore greater extinction potential, while the increasing peak population growth rates with increasing temperatures can increase the rate at which evolution occurs.

To explore how the difference between the thermal performance functional response (Eqn 30) and the traditional fitness function (Eqn 29) affects evolutionary dynamics, I compare the outcome of Eqns 27 and 28 with these two fitness functions. Using the thermal performance function (Eqn 30) for fitness in Eqns 27 and 28 leads to the dynamics for the genetic moments (mean $\bar{y}$ and variance V_G):

$$\frac{\mathrm{d}\bar{y}}{\mathrm{d}t} = \frac{V_G(\theta-\bar{y})}{S} ae^{b\theta} , \tag{31}$$

$$\frac{\mathrm{d}V_G}{\mathrm{d}t} = V_M - \frac{V_G^2}{S} ae^{b\theta} , \tag{32}$$

given mutation increases genetic variance by a constant rate V_M. The overall population growth rate over time is then:

$$\bar{r}(t) = \left(1 - \frac{V_G + V_E + (\bar{y}-\theta)^2}{2S}\right) ae^{b\theta} \tag{33}$$

[see Appendix A of Baskett et al. (Baskett et al., 2009) for a derivation of Eqns 31–33]. Coupling these genetic dynamics with population dynamics based on this growth rate, with a cap to the population size at carrying capacity K, yields:

$$\begin{aligned} &\frac{\mathrm{d}N}{\mathrm{d}t} = 0 \text{ for } N > K \text{ and } \bar{r}(t) > 0 , \\ &\frac{\mathrm{d}N}{\mathrm{d}t} = \bar{r}(t)N \text{ otherwise} . \end{aligned} \tag{34}$$

Here the modification from the traditional fitness function (Eqn 29) based on the physiological understanding of population responses to changes in temperature is the factor $ae^{b\theta}$ multiplied by the population growth rate, which then reappears throughout the dynamics; see Lynch et al. (Lynch et al., 1991) for the equivalent dynamics with the traditional fitness function.

As in the study by Lynch and colleagues (Lynch et al., 1991), I investigate the capacity for a population to adapt to a linearly changing environment, i.e. $\theta(t)=\theta_0+kt$ for different rates of environmental change k (Fig. 3). In the simulations that use the thermal performance curves, the potential for faster population growth rates with increasing temperatures does lead to faster evolutionary change (Fig. 3C, comparing dashed lines with solid lines of the same color). However, the faster decline in population growth at higher temperatures leads to earlier and greater likelihood of extinction rather than adaptation (Fig. 3D, again comparing dashed lines to solid lines of the same color). This example illustrates how a mechanistic fitness function can affect the quantitative predictions for adaptation to environmental change in ways that are difficult to predict.

Game theory and dispersal evolution example

Another approach to modeling phenotypic evolution is game theory. Instead of following gene frequencies, game theory predicts the evolutionary outcome based on whether strategies can invade and be invaded by other strategies. This approach explicitly accounts for frequency dependence, i.e. the potential for the fitness of a given phenotype to depend on the phenotype distribution in the population (accounting for frequency dependence in population genetics models is feasible but would require extension of the types of equations described above, much like accounting for density dependence) (Day, 2005). Specifically, the fitness function (or pay-off function) describes the per-capita population growth factor $\lambda(z,\hat{z})$ of a rare invader with phenotype z in a population with resident phenotype $\hat{z}$, where the population dynamics of the invader are:

$$N_{t+1} = \lambda(z,\hat{z})N_t \tag{35}$$

[or, in continuous time, $\mathrm{d}N/\mathrm{d}t=r(z,\hat{z})N$, where the per-capita population growth rate r and λ are not equivalent; $r=\ln(\lambda)$ in the density-independent and frequency-independent case].

The classic evolutionarily stable strategy (ESS) (*sensu* Maynard Smith and Price, 1973), or a strategy that cannot be invaded by any other strategy, is z^* such that $\lambda(z,z^*)\leq\lambda(z^*,z^*)$ for all z in this representation. Note that if λ is independent of $\hat{z}$ [i.e. no frequency dependence such that $\lambda(z,\hat{z})=\lambda(z)$], this reduces to a question of fitness optimization [z that maximizes $\lambda(z)$]. In addition to the ability to withstand invasion, another dimension important to predicting the evolutionary outcome is the ability to invade when rare, here formalized as z^* such that $\lambda(z^*,z)\geq\lambda(z,z)$ for all z [termed the neighborhood invader strategy, or NIS, when considering nearby strategies (Apaloo, 1997)]. Plotting successful *versus* unsuccessful invasion for the full set of all pairwise combinations of invaders and residents in a 'pairwise invasibility plot' indicates the predicted evolutionary outcome [see Geritz et al. (Geritz et al., 1998) and Levin and Muller-Landau (Levin and Muller-Landau, 2000) for a full classification of evolutionary outcomes]. Expanding on this theory to understand the process of evolutionary branching (often interpreted as speciation) is central to the related framework of adaptive dynamics (Day, 2005; Geritz et al., 1998).

As with the previous evolutionary frameworks, integrating mechanistic knowledge (including, but not limited to, biomechanics-relevant knowledge) of organisms' interactions with their environment into this framework would involve extending the fitness function λ. In this case, λ becomes a function of the

environment as well as the invader and resident traits. As with quantitative genetics, the theoretical framework for function-valued traits, described for adaptive dynamics by Dieckmann et al. (Dieckmann et al., 2006), provides a possible construct for modeling such traits.

For an illustration of how incorporating mechanistic knowledge of organism–environment interactions might provide different insight from game theoretic models, consider the evolution of dispersal. Models of the evolution of dispersal have a long history in game theory (reviewed by Johnson and Gaines, 1990; Ronce, 2007). The evolving trait in such models typically concerns whether or not dispersal occurs (e.g. proportion of offspring dispersing), with any dispersal occurring in a generic way such as all offspring having an equal likelihood of landing in any site (global dispersal). Recent models have begun to incorporate the evolution of dispersal kernels (functions that describe the probability density of dispersing offspring in space) (e.g. Hovestadt et al., 2001; Rousset and Gandon, 2002) and therefore allow exploration of the evolution of dispersal distance or kernel shape. In contrast, non-evolutionary models of dispersal have a long history of incorporating mechanism, including the influence of the physical environment, to determine the expected shape of the dispersal kernels, especially for wind-dispersed seeds (reviewed by Levin et al., 2003).

A similar development to seed dispersal models, both ecological and evolutionary, has occurred in models of dispersal for marine organisms with both planktonic and benthic stages. Models of the evolution of dispersal for such marine organisms have typically focused on the question of the evolution of feeding (planktotrophic) and non-feeding (lecithotrophic) larvae given life history trade-offs, often based on a fitness optimization approach (where the planktonic stage might serve more as a migration to increase feeding opportunities or decrease predation risk than a dispersal event) (reviewed by Strathmann, 1985). More recent evolutionary models connect the expected offspring size that results from such trade-offs to the average dispersal distance of a dispersal kernel based on phenomenological expectations of the energetic requirements for development to metamorphosis (e.g. Baskett et al., 2007). Most recently, non-evolutionary mechanistic models of the dispersal kernel for marine spores as it depends on currents have recently been built on biomechanics-based knowledge (Gaylord et al., 2006; Gaylord et al., 2012).

The recent extension of evolutionary models to incorporate dispersal kernels and the existence of mechanistic descriptions of dispersal kernels as they connect to the physical environment (the functional response of this example) for wind-dispersed seeds and marine spores provides an opportunity to develop more mechanistic evolutionary models. Incorporating the mechanistic dispersal kernels into evolutionary models would shift the focus of such models from the question of whether or not dispersal (or planktotrophy) evolves to the evolution of a trait that drives dispersal as it depends on the environment. Therefore, such models would allow deeper insight into the evolution of the shape of the dispersal kernel, which is key to a variety of ecological processes from range expansion to community-level diversity (Levin et al., 2003).

Furthermore, even for ecological or evolutionary questions where the exact shape of the dispersal kernel does not have a major impact [e.g. rules of thumb for marine reserve design (Lockwood et al., 2002)], following the evolution of a biomechanics-based dispersal kernel can provide insight into how the expected outcome depends on local or changing environmental conditions [e.g. marine currents (Gaylord et al., 2006)]. The connection between the physical environment and a biologically relevant trait creates the potential to mechanistically consider constraints on dispersal evolution as well. Finally, the focus on biologically relevant traits, in the case of dispersal kernels likely a morphological trait in the dispersal stage, might allow better connection of models to data such as heritability estimates. For example, one crucial gap between theory and data in the context of dispersal evolution is that theoretical predictions and model-based hypotheses about the amount of dispersal are difficult to test against empirical data because dispersal itself is difficult to measure (Ronce, 2007); theoretical predictions about dispersal-related traits under different environmental conditions might provide more opportunities for relevant empirical measurement and tests. This speculative example suggests the general potential for more mechanistic evolutionary models to provide ecological insight, allow greater understanding of evolution under local or changing environmental conditions, and better connect theory and data.

Conservation applications

Both ecological and evolutionary processes occur in the context of human-driven global change (Palumbi, 2001; Vitousek et al., 1997). One proposed strength of a more mechanistic approach to ecological and evolutionary modeling, especially that related to biomechanics, is the enhanced predictive power under novel environmental conditions, such as physiological understanding informing predicted responses to climate change (Denny and Helmuth, 2009; Helmuth et al., 2005; Hoffmann and Sgro, 2011; Hofmann and Todgham, 2010; Kearney et al., 2012; McGill et al., 2006; Norberg, 2004). However, the types of models described here, even with added realism from mechanistic functional responses, remain at the general end of the modeling trade-off described in the Introduction and therefore tend not to have the level of both realism and precision necessary for predictions (Levins, 1966). In addition, the key challenge for ecologists and conservation biologists is not just predicting what will happen under climate change, but informing local science-based management decisions (e.g. reserve design, invasive species control) under a changing climate (Dawson et al., 2011; Heller and Zavaleta, 2009). Vulnerability estimated from predictive models can provide a first step towards conservation management under a changing climate (Rowland et al., 2011), but insight into relative vulnerability and sensitivity to different processes can stem from the more general models described here as well.

One possible component of local management under climate change is to protect the capacity of natural systems to respond to the global changes (Heller and Zavaleta, 2009). On the evolutionary level, protecting response capacity means protecting the potential for genetic adaptation as it depends on properties such as genetic variance, population size and the level of gene flow (Hoffmann and Sgro, 2011; Sgro et al., 2011) where genetic adaptation is one aspect of the population-level response to climate change, along with movement and acclimation (Parmesan, 2006). On the community level, protecting the response capacity involves protecting community-level resistance, resilience and robustness to environmental change as it depends on properties and processes such as diversity, modularity, redundancy and feedback loops (Levin and Lubchenco, 2008). Sensitivity of basic models to different assumptions and parameters can provide insight into which processes and properties are most important to the overall response and therefore inform this aspect of management under climate change.

Sensitivity analysis is an important tool for prioritizing conservation management efforts [e.g. Crouse and colleagues (Crouse et al., 1987) provide a quintessential example of this approach]. For an example related to the question of physiological response to climate change, we constructed a model, rooted in the thermal tolerance fitness function described above, of coral reef ecological and evolutionary response to the future thermal stress expected with climate change (Baskett et al., 2010). Sensitivity analysis of a variety of model constructs and of all model parameters provided an integrative and quantitative comparison of existing recommendations (previously considered qualitatively and occasionally contradictory) for local protection of coral reef resistance, resilience and adaptive capacity in a changing climate.

Generally, incorporating mechanistic functional responses into basic models allows greater connection of model parameters to biologically relevant characteristics (Schoener, 1986), and therefore increases the potential to connect model outcomes to an empirical understanding of important traits or physiological processes. Furthermore, mechanistic functional responses that include interactions with the physical environment will also indicate sensitivity to empirically relevant environmental conditions. Such insights can provide a sense both of which patterns or processes might best allow response capacity and of which local stressors are more likely to interact synergistically with global change, which can suggest prioritization in terms of which locations and processes to protect and which local stressors to protect against. This enhanced realism and biological relevance in the context of basic models could help add much-needed specificity to general recommendations, allowing them to strike the difficult-to-achieve balance between broad applicability and concreteness for recommendations for management under a changing climate (Heller and Zavaleta, 2009).

Conclusions: a trait-based approach

In summary, mechanistic functional responses can readily fit into existing frameworks for modeling species interactions and, in the guise of fitness functions, evolutionary dynamics. Furthermore, mechanistic functional responses that incorporate organisms' interactions with their environment can extend these frameworks to explore environmental influences, including multispecies or adaptive responses to changing environments and the evolution of phenotypic plasticity. For both community and evolutionary models, this approach ties into existing theoretical frameworks such as trait-mediated interactions (Bolker et al., 2003) and the evolution of function-valued traits (Dieckmann et al., 2006; Kingsolver and Gomulkiewicz, 2003).

These connections highlight how integrating mechanistic functional responses into basic models represents a trait-based approach, where physiological or morphological traits mediate the organism–environment interaction. Therefore, a key model-building decision will be the careful choice of what trait(s) dictate the model system response to the environment (Chevin et al., 2010; Naeem and Wright, 2003; Norberg, 2004). For community models, such traits must be relevant to organism or population performance (McGill et al., 2006; Naeem and Wright, 2003), and for evolutionary models, such traits must have a heritable component as well as be ecologically relevant (Chevin et al., 2010).

Overall, integrating mechanistic functional responses into community and evolutionary models provides a mechanistic underpinning that more readily connects these models to empirical data and environmental conditions [at the cost of some generality (Schoener, 1986)], thus advancing theoretical population biology. In addition, using the aggregated functional responses allows a tractable exploration that draws from existing tools and frameworks in theoretical population biology, thus advancing efforts to integrate biomechanics with ecology in terms of scaling up biomechanics-based knowledge both ecologically and temporally. This can complement more detail-oriented theoretical frameworks that directly model the mechanisms by providing general insight into when mechanism affects population dynamics. The examples discussed here (the classic example of Holling functional responses, recent efforts to incorporate temperature dependence into predator–prey dynamics, the plant resource example of ecosystem engineering, the phenotype–performance–fitness framework, the use of the thermal tolerance envelope as a fitness function in quantitative genetic models, and the potential for integration of mechanistic dispersal kernels in game theoretic frameworks of dispersal evolution) illustrate how mechanistic functional responses can affect quantitative and qualitative dynamics and allow a better connection of theory and data.

Acknowledgements

I am indebted to Mark Denny for the invitation to participate in the 2011 *Journal of Experimental Biology* Symposium on Integrating Biomechanics and Ecology in addition to valuable feedback on these ideas and previous versions of the manuscript. Insightful conversations with and feedback from Brian Gaylord and Roger Nisbet also contributed to the ideas presented here. Finally, Johan van de Koppel and an anonymous reviewer provided helpful feedback on a previous version of this manuscript.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

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