CAUSES OF DEATH AND FAILURES TO REPRODUCE, LIMITING RESOURCES, ECOLOGICAL DYNAMICS, AND SELECTION: HOW TO EVOLVE A LOW PREDATION GUPPY, AND CAUSE A TROPHIC CASCADE

Tim Coulson

Department of Zoology, University of Oxford, The Zoology Research and Administration Building, 11a Mansfield Road, Oxford, OX1 3SZ, UK

Email: tim.coulson@zoo.ox.ac.uk

Abstract

What happens when a species is introduced to, or removed from, a community? In many cases such biotic perturbations can result in a change to the dominant causes of death, or failures to reproduce, in the species that are most significantly impacted. These changes can also alter the limiting resource types (be they food, mates, enemy free-space, nutrients, mineral, water or shelter) and consequently the factors that limit the population. A change in the limiting resource can relax selection on some traits, while imposing novel selection pressures for phenotypic traits associated with the new limiting resource. These traits may then evolve in response to the novel selection regime. Phenotypic evolution can feedback to further alter resource distributions, and consequently species interactions, generating cascading effects throughout the ecological network describing the community. I propose a framework that links ecology and evolution among interacting species via the dynamics of the dominant causes of mortality and failures to reproduce, and the limiting resources that determine these causes. I argue that structured modeling approaches are now sufficiently mature that predictive models can now be constructed.

Introduction

In the lowland streams of Northern Trinidad guppies (*Poecilia reticulata*) live in complex communities. They predominantly feed on high-quality stream invertebrates that are also food for a number of other fish species. Guppies themselves are prey for predatory fishes, such as wolf fish (*Hoplias malabaricus*) and pike cichlids (*Crenicichla frenata*). Predation is the primary cause of death for a guppy living in these communities, such that the populations are limited by predation. A consequence of this is that enemy-free space is the limiting resource. In response to this limitation, guppies have evolved streamlined bauplans, live in shoals, have high maximum swimming speeds, fast metabolic rates, are rather dully colored, have small sizes and low ages of sexual maturity, large litters of small offspring, and fast life histories (Reznick and Endler 1982, Reznick and Bryga 1987, Reznick and Yang 1993, Reznick et al. 2001, Travis et al. 2014).

Above waterfalls, on the same streams where high predation guppies are found, predatory fish are largely absent. Guppies in these streams live in simpler communities, with only one major competitor, Hart's killifish (*Rivulus hartii*), which is also an intra-guild predator. The guppy populations are food-limited, the limiting resource is therefore food availability, and the likely primary cause of death and failure to breed is starvation. A consequence of this is that their diet contains a high percentage of low-quality food, such as algae and bacteria, that are largely absent from the diets of high-predation guppies. Guppies adapted to these low predation environments have evolved a greater age and larger size at sexual maturity, brighter male coloration, a less-streamlined bauplan, reduced shoaling behaviour, a lower metabolic rate, smaller litter sizes, larger offspring and a slower life history. Because these changes are repeated across streams, they are predictable in that we know what will happen when guppies are released from predation (Reznick and Endler 1982, Reznick and Bryga 1987, Reznick and Yang 1993, Reznick et al. 2001, Travis et al. 2014).

The guppies provide a well-characterised example of a general phenomenon: the primary causes of death or failures to reproduce within a population reflect the resources that are limiting and the factors that limit the population's growth (Schindler 1974, Sibly and Hone 2002, Rohr et al. 2003). In addition, the limiting resources determine which phenotypic traits are under selection, and the expected course of evolution (Walters and Juanes 1993, Fishman and Willis 2008). These observations suggest a general framework that may help explain ecological dynamics, evolution, and eco-evolutionary feedbacks. In this paper I develop such a framework. I do this by first considering the dynamics of birth and death in a population that is limited by some process. Second, I consider the causes of death and failures to reproduce that can limit populations, and their connection to limiting resources. Third, I consider how different types of limiting resources can influence selection. Fourth, I consider how selection can result in evolution in environments that are limited by the availability of a key resource, be it food, enemy-free space, mates, territories, minerals, nutrients, or water. Fifth, I bring the various threads together to consider eco-evolutionary feedbacks, before sixth, considering approaches to capture the processes described above. Seventh, I place these dynamics in a community setting. I finish with two thought experiments to demonstrate the potential utility of the framework I propose.

The Dynamics of Birth and Death

I start by considering the dynamics of a population at a numerical, stochastic, equilibrium in a natural setting. I assume that the population dynamics generate a stationary distribution of population sizes such that population size fluctuates with time but shows no persistent temporal trend. The assumption is supported by the observation that natural populations are frequently limited by the availability or accessibility of biotic or abiotic resources, and that if these drivers are themselves stationary yet fluctuate with time, they will generate variation in population size (Berryman 2002, Turchin 2003, Royama 2012).

Given this assumption, the long-run stochastic population growth rate $a = 0$, where

$$a = \frac{1}{T}\sum_{t=1}^{T}\log(\lambda_t)$$

with T being time. The arithmetic average $\bar{\lambda}_t$ of the per time step population growth rates $\lambda_t = \frac{N_{t+1}}{N_t}$ where N is population size, are greater than unity when $a = 0$ and the population fluctuates over time (Figures 1(a, b)) (Cohen 1979, Tuljapurkar and Orzack 1980). The long-run stochastic population growth rate can consequently be approximated as $a \approx \log(\bar{\lambda}_t) - V_E$ where V_E is a measure of the variance in the logged λ_ts (Tuljapurkar and Orzack 1980, Tuljapurkar 1990b, Lande et al. 2003). The variance can be attributed to contributions from variation in environmental drivers and demographic stochasticity (Lande et al. 2003). Obviously, when $a = 0$, $\bar{\lambda}_t \approx V_E$. But how does a relate to the fundamental biological processes of birth and death?

The per time step population growth rate $\lambda_t = \frac{N_{t+1}}{N_t}$ can also be expressed as $\lambda_t = S_t + R_t$ where S_t is the annual per capita survival rate, and R_t is the annual per capita recruitment rate (defined as the per capita production of offspring that are born between times t and $t + 1$ that survive to enter the population at time $t + 1$) (Coulson et al. 2006). The mean of the sum of temporally varying annual survival and annual recruitment rates will consequently also be greater than unity when $a = 0$ and the population shows no temporal trend. Exactly how much greater this average needs to be for the long-run stochastic growth rate to equal zero depends upon the temporal variation in S_t and R_t and how it contributes to V_E. As temporal variation in S_t and R_t increases so too does the arithmetic mean of $S_t + R_t$ that is required for a long-run stochastic population growth rate to equal 0 in a population at a stochastic numerical equilibrium (Figure 1(a, b)).

Per capita survival and recruitment rates vary with individual attributes including age and sex (Leslie 1945, Caswell 2001). The values of S_t and R_t at any particular time t consequently depend upon the population's age and sex structure, as well as with other aspects of the environment (Coulson et al. 2001). The ways that survival and recruitment vary with age are captured by survivorship and fertility schedules (Figure 1(c, d)) (Stearns 1976). Survivorship from birth (age 0) to age x, L_x, and age-specific fertility rates, M_x, can be combined to estimate moments of the distributions of generation length, life expectancy at each age x, and lifetime reproductive success (Caswell 2001). These quantities define the average life history of individuals within the population. R_0 is mean lifetime reproductive success and describes the rate of population growth per generation. In fluctuating environments where $a = 0$, a value of $\log(\bar{R}_0) > 0$ will be observed. An analogous quantity to the long-run stochastic growth rate for R_0 exists (termed a_0), where temporal variance within and across the L_x and M_x schedules will contribute to the life history equivalent of V_E (termed $V_{0,E}$). We can consequently write $a_0 \approx \bar{R}_0 - V_{0,E}$ (Tuljapurkar et al. 2009).

S_t, R_t, λ_t, a, V_E, L_x, M_x, R_0, a_0, and $V_{0,E}$ can all vary among genotypes, phenotypes or strategies within a population (Lande 1982, Charlesworth 1994, Coulson et al. 2011). I now assume clonal strategies that are passed between parents and their offspring without error. In stochastic, density-independent environments, two different strategies can have different long-run stochastic growth rates. The strategy with the highest value of a will grow to dominate the population at the expense of strategies with lower values (Orzack and Tuljapurkar 1989, Metz et al. 1992). In other words, if one strategy has a value of $a = 0$ any strategy that can invade would have to have a value of $a' > 0$. There are two routes via which a strategy can have higher fitness than another. First, it may have higher expected values of S_t, or R_t (and consequently $\bar{\lambda}_t$) by having a higher value of R_0 via altered L_x and M_x schedules, but an equivalent value of V_E. Second, it might have identical values for $\bar{\lambda}_t$ via identical L_x and M_x

schedules but a lower value of $V_{0,E}$ and consequently V_E (Orzack and Tuljapurkar 1989, Orzack 1993, Tuljapurkar et al. 2003, Lande et al. 2009). For example, the red life history in Figures 1(c, d) would invade the grey one if they were to compete in a density-independent environment with all other things being equal. Evolution can consequently proceed by either increasing expected survivorship of fertility at any age, or by reducing temporal variation in survivorship or fertility at any given age as either route can increase the long-run stochastic growth rate a.

Causes of death and failures to reproduce

The L_x and M_x schedules of a population are determined by the environment and the distribution of causes of death or failures to reproduce at each age x (Figure 1(e)) (Stearns 1976, Gaillard et al. 2000). Different ages can have different survival and reproductive rates because they experience different primary causes of death or failures to reproduce. For example, the primary cause of death in prime-aged female elk (*Cervus canadensis*) in Yellowstone is likely accidents and survival rates are high. In contrast, senescent female elk die at a much faster rate due to their vulnerability to wolf (*Canis lupus*) predation (Wright et al. 2006). In a stationary population, the causes of death and the failures to reproduce balance across ages to maintain a long-run stochastic growth rate of $a = 0$. and $a_0 = 0$.

At each age x at time t, different individuals can have different expected values of L_x and M_x as a function of their phenotypic trait values (Coulson et al. 2010, Steiner et al. 2014). For example, if the primary cause of death is predation, then individuals with more effective armour or effective predatory avoidance behaviours will have higher age-specific survival rates than their less effectively endowed conspecifics. A consequence of this is that environmentally-determined causes of death or failures to reproduce generate phenotypic selection pressures that can result in evolution. This means that observation of particular phenotypic traits within

a population can inform about the processes that have historically determined survivorship and reproductive schedules through their roles as key causes of death or failures to reproduce (Dawkins and Krebs 1979). The streamlined bauplans and high maximum swimming speeds in Trinidadian guppies provide evidence that high-predation guppies have evolved to avoid predation (Reznick and Endler 1982). Many the phenotypic traits we have adaptive explanations for tend to to be associated with elevated values of age-specific survival and reproduction rather than with reductions in V_E (but see below) (Krebs and Davies 1997).

Population limitation

I will now return to the initial case of a stationary population where $a = 0$. It is stationary because some process is preventing it from growing: no population can continue to increase indefinitely. Population limitation is determined by some form of resource limitation, where resources can include food, minerals, nutrients, territory, mates, shelter, and enemy-free space (Sibly and Hone 2002, Turchin 2003). Prey species that are predator-limited are limited by the availability of enemy-free space, while consumers in a food-limited population are limited by energy availability (Jeffries and Lawton 1984, Royama 2012).

A change in limiting regime will be a consequence of a change in the dominant processes that determine death or failures to reproduce (e.g. (Smith et al. 2003)). For example, if a population shifts from being predation-limited to food-limited, the primary cause in death may shift from predation to starvation. Prior to the removal of predators, enemy-free space was likely the limiting resource, while once predators have been removed, food becomes limiting (Travis et al. 2014).

What happens when a change in limiting factor occurs, and a population shifts from an initial stationary distribution where $a = 0$ to another, new equilibrium where $a = 0$ also? The shift between equilibria will likely not last more than a small number of generations. The two

equilibria may have different distributions of λ_t, but because something will be limiting the population in each regime, they will have long-run stochastic growth rates of $a = 0$ once stationarity is achieved (Hastings and Wysham 2010). Any differences between the distributions of λ_t reflect differences in the distributions of S_t and R_t, which in turn are highly likely to represent differences in the population's L_x and M_x schedules. In turn, these differences may result in a change in the moments of the generation length, lifetime reproductive success, and life expectancy at age x distributions (Coulson et al. 2011).

Although both regimes have long run stochastic growth rates of $a = 0$, they will likely have different distributions of population sizes, N_t (Hastings and Wysham 2010). For example, when guppies are released from predation, their average population size increases, with stationary high and low predation populations also having different variances and higher moments of N_t (Travis et al. 2014). Stationary populations where $a = 0$ will exhibit evidence of statistical density-dependence regardless of the process that is limiting them, as the per time-step population growth rate, λ_t, will tend to decline as population size N_t increases (Turchin 2003).

A change in limiting resource and consequently the factor limiting the population, and the resulting change in population dynamics regime, will also likely result in a change in selection pressure that could ultimately result in evolutionary change (Roughgarden 1971, Metz et al. 1995, Doebeli 1997, Coulson et al. 2017). Selection always favours individuals that are best equipped to detect, acquire and utilise limiting resources, whatever those resources may be, but exactly how selection plays out, and how phenotypes adaptively evolve, depends upon whether resources are defensible, the manner in which resources are depleted, and the rate at which they renew (Figure 2). For example, nesting sites for hole-nesting birds are a fairly constant resource over time. If nest sites are limiting and constant in number, we might expect to see evolution for strategies that aid in nest-site defence. In contrast, in food-limited environments where food

cannot be defended (e.g. grass in a food-limited sheep population), evolution is expected to favour strategies that can exploit resources as soon as they renew, or that allow them to survive on resources that they have driven down to low levels of availability (Roughgarden 1971). In order to understand evolution in response to key causes of death, failures to breed, and resource limitation, and how it might influence population dynamics, it is helpful to start by considering the structure of selection differentials, before focusing on patterns of inheritance.

Selection

Selection operates on phenotypic traits (Falconer 1960). Most phenotypic traits that are under selection are associated with the detection, acquisition or utilisation of resources, be those resources food, water, minerals, nutrients, mates, shelter, or enemy-free space. For example, the senses are associated with the detection of resources, many behaviors and some aspects of morphology are associated with the acquisition of resources, while respiration, metabolism, physiology and the production of offspring are associated with the utilisation of resources.

A change in limiting resource is likely to result in a change in selection pressures (Roughgarden 1971). For example, the loss of predators from a predator-limited system could result in a switch from enemy-free space being the limiting resource to food becoming limiting. The associated change in the cause of death and failures to reproduce from predation to starvation will likely result in relaxed selection on phenotypic traits or strategies associated with the detection or avoidance of predators or the successful utilizing of enemy-free space, but would result in novel selection pressures on phenotypic traits or strategies associated with the detection, acquisition and utilization of food (Reznick and Endler 1982, Reznick and Bryga 1987, Travis et al. 2014). Given sufficient time, the population will adapt to the new environmental regime, and will express traits to demonstrate this. Given this, can we predict how the population will evolve in response to a change in key limiting factor? I start by

considering evolution that acts to directly increase L_x or M_x at a given age x rather than to reduce V_E. Initially I do not consider resource limitation.

Evolution is defined as a change in allele frequencies (Charlesworth 1994), the mean value of a heritable phenotypic trait (Falconer 1960), or the frequency of a strategy (Maynard Smith 1982). But evolution is also sometimes expected to result in a change in the population dynamics. Adaptive evolution is determined by selection, a process which operates on the phenotype or strategy and which can change allele frequencies, which is described with a selection differential (Price 1970). A selection differential for a phenotypic trait at time t can be written as

$$\phi_t(z) = \frac{\frac{1}{N_t}\sum_{i=1}^{N_t}\left(z_{i,t} - \bar{z}_t\right)\left(w_{i,t} - \bar{w}_t\right)}{\bar{w}_t} \qquad 1$$

where z_i and w_i are respectively the phenotypic trait and absolute fitness of individual i. Over a single time-step, individual absolute fitness $w_{i,t}$ is the number of offspring produced by an individual between times t and $t+1$ that survive to enter the population at time $t+1$, scaled by the relatedness value between parents and offspring, plus one if the individual itself survives (Fisher 1930). Individual absolute fitness $w_{i,t}$ consequently consists of a survival and reproductive success component. The mean of $w_{i,t}$ across all N_t individuals within the population is equal to the per time step population growth rate such that $\bar{w}_t = \lambda_t$ and the sum $\sum_{i=1}^{N_t} w_{i,t} = N_{t+1}$. The means of the survival and reproductive components of $w_{i,t}$ across all individuals within the population are S_t and R_t. The selection differential at time t is determined by the association between the trait and individual absolute fitness that is usually statistically estimated via regression (Falconer 1960, Price 1970).

In a constant, density-independent environment, a constant selection differential of zero implies no evolution of the phenotypic trait or mean fitness, and the population will grow at a

constant, exponential rate. In contrast, a persistent non-zero selection differential resulting from a constant statistical relationship between the phenotype and individual absolute fitness that is not influenced by any other variables (Figure 3(a)), implies that an increase in the mean $\bar{Z}$ of the phenotype as a result of selection (Figure 3(a)) will also result in an increase in mean fitness (the per time step population growth rate) (Figure 3(b)). If we assume the trait is heritable, a consequence of this is the population will grow hyper-exponentially as the mean phenotype evolves (Witting 2000a, b). As this happens, the covariance between the phenotype and fitness will also likely change with time (Figure 3(c)), as will the selection differential (Figure 3(d)) (Coulson et al. 2011, Coulson et al. 2017, Coulson et al. 2018).

No population can increase indefinitely at an exponential or hyper-exponential rate. In time, one or more resources becomes limiting, and population increases cease (Malthus 1798). I now relax the assumption that there is no limiting resource and assume the population cannot grow indefinitely. The check on population growth (e.g. Figure 3(b)) does not mean that phenotypic evolution necessarily grounds to a halt (Haldane 1932, Wright 1969, Matsuda and Abrams 1994, Rankin and López-Sepulcre 2005). Persistent nom-zero selection differentials can occur in a population of constant (or even declining) size if evolution results in a temporal change to the association between a phenotype and individual absolute fitness, as well as in change in the mean phenotype (Figure 4) (Coulson et al. 2017).

When a limiting resource such as the availability of nesting sites for hole-nesting birds remains constant over time, where a hole is required for reproduction, evolution can occur while mean fitness (the population growth rate) remains constant (Figure 4). To understand how this happens, it is helpful to consider how a selection differential over a single time step can be calculated from the distribution of a phenotypic trait $N_t(Z)$ and a fitness function $w_t(Z)$ (Coulson et al. 2010, Wallace et al. 2013). First, calculate the means of the trait before, and after, selection,

$$\bar{Z}_t = \frac{\int Z\, N_t(Z) dz}{\int N_t(Z) dz}$$

$$\bar{Z'}_t = \frac{\int w(Z)\, Z N_t(Z) dz}{\int w(Z) N_t(Z) dz}$$

before calculating their difference,

$$\phi_t(z) = \bar{Z'}_t - \bar{Z}_t = \frac{\frac{1}{N_t}\sum_{i=1}^{N_t}(z_{i,t} - \bar{z}_t)(w_{i,t} - \bar{w}_t)}{\bar{w}_t}$$

We can also calculate the per time step population growth rate over the time step as,

$$\lambda_t = \bar{w}_t = \frac{\int w(Z) N_t(Z) dz}{\int N_t(Z) dz}.$$

Assume that the fitness function $w(Z)$ is not influenced by any other terms than the phenotype Z, and is of the form

$$w(Z) = \beta_0 + \beta_1 Z \text{ with } \beta_1 > 0.$$

It is now apparent that $\phi_t(z) > 0$ and, assuming the trait is heritable, then $\bar{Z}_{t+1} > \bar{Z}_t$ and $\lambda_{t+1} > \lambda_t$. In contrast, if $\phi_t(z) > 0$, the trait is heritable, and $\lambda_{t+1} = \lambda_t$, then the fitness function must vary with time such that the intercept β_0 must change (Figure 4). Such change occurs when the improvement in fitness of selected individuals comes via a reduction in the fitness of competitors (Wright 1969, Matsuda and Abrams 1994). For example, if a large bird gains a nest site, it does so at the expense of a smaller individual. More generally, in such cases some heritable phenotypes will have long-run stochastic population growth rates that are greater than 0, others will necessarily have rates that are less than zero, but the average long-run stochastic growth rates across competing phenotypes will be 0.

In some cases, it is possible for evolution to result in a decrease in population size (e.g. Figure 2) (Haldane 1932). For example, if a food resource can be defended, and the ability to defend

a resource scales with body size, then evolution to larger sizes will result in greater energy demands per individual. In turn, assuming the resource renewal rates and availability remain constant, evolution will result in fewer, larger, individuals exploiting the resource. At the limit, this could result in evolutionary suicide (Rankin and López-Sepulcre 2005).

I now consider the second way that evolution can proceed. Selection can also act to reduce V_E (Hirshfield and Tinkle 1975, Orzack and Tuljapurkar 1989). When this happens, instead of selection acting to increase fitness at a particular age, it acts to reduce the expected variance around the survivorship and fertility schedules. Evolution achieves this via the evolution of traits associated with an ability to be resilient to variation in the environment (Hirshfield and Tinkle 1975)(e.g. red versus grey life histories in Figure 1(c, d)). Traits associated with reducing V_E may include body size, the ability to store fat reserves, the ability to migrate to new locations, or even the ability to hibernate when times are bad.

Exploration of evolution via a reduction in V_E requires comparing the fitness of competing life history strategies, each with a different temporal variation in L_{x} and M_{x} schedules (Tuljapurkar et al. 2009). If we assume that two strategies have identical expected (mean) schedules, then the fittest strategy will have lower variation than its competitor across the entirety of its schedules – I.e. lower $V_{0,E}$. This does not mean it will have less expected variation in L_{x} and/or M_{x} at every age x, but it will do across the entirety of both schedules.

Over a single time-step, individual fitness is *always* the number of offspring produced by an individual between times t and $t+1$ that survive to enter the population, weighted by the ploidy of the species, plus one if the focal individual survives (Fisher 1930). Similarly, mean fitness over a time step is *always* λ_t. In contrast, longer-term individual absolute fitness, and mean fitness, over multiple time steps cannot be universally defined with a single scalar quantity (Sæther and Engen 2015). The reason for this is that the definition of fitness is context-

dependent (Grafen 1988). In particular, if the fitness function changes with time because the fitness gains of selected individuals are achieved via the reduction in fitness of others through successful exploitation of a resource, then the definition of fitness needs to capture the way that the fitness function changes as evolution proceeds (Matsuda and Abrams 1994, Travis et al. 2013, Coulson et al. 2017). In a number of simple cases of population limitation fitness has been shown to be carrying capacity, with the fittest strategy having the higher carrying capacity (Charlesworth 1994, Lande et al. 2009, 2017). However, this will not always be the case as can easily be seen by considering cases of evolutionary suicide where population size evolves to be smaller with time (Haldane 1932). The only reliable way to measure the fitness of different life history strategies in many circumstances is via competing them against one another in a simulation and examining which one wins (Maynard Smith 1982). This is most easily achieved by assuming that competing strategies are clonally inherited with (near) perfect fidelity (Metz et al. 1995). For sexually reproducing species this assumption is violated.

For phenotypic traits that are selected by increasing survival or fertility at a point in time, we can measure the strength of selection at that single point in time. In contrast, selection on phenotypic traits that are selected by reducing the variance around survivorship and fertility schedules, must be measured across the entire history (Orzack and Tuljapurkar 1989, Lande et al. 2009). However, even for phenotypic traits that acts to increase survival or fertility at a particular point in time, a dynamical understanding of their evolution requires information on how the phenotypic trait develops. In order to understand why an individual expresses a particular phenotypic trait value at a point in time it is necessary to understand the genotype-phenotype map. The map can be significantly impacted by resource availability and the abilities of individuals at accessing those limiting resources.

Genotype-phenotype maps

Selection acts on phenotypes, not genotypes, but selection can change genotype and allele frequencies. Exactly how selection on a phenotype changes genotype and allele frequencies depends upon details of the genotype-phenotype map (Falconer 1960). When the map is one-to-one, where one genotype always produces the same unique phenotype, evolutionary change is relatively straightforward to predict using methods from population genetics (Crow and Kimura 2009).

For many phenotypic traits, the map between genotype and phenotype is more complex than one-to-one. Biologists can characterize more complex maps by modifying levels of gene expression, or knocking out particular genes entirely, and examining effects on the phenotype (or fitness). The approach has been used across a range of environments in some model organisms (Bouché and Bouchez 2001, Van Opijnen et al. 2009). Gene networks have been identified for a number of key phenotypic traits in a number of species, but such work has not been applied in the field, except in very limited cases, and I do not attempt to review the extensive literature on gene-knockouts here.

In most cases where the genotype-phenotype is more complex than the one-to-one maps widely assumed in population genetics, details of the map are not known. In these cases, assumptions from quantitative genetics are relied upon (Falconer 1960). These assumptions derive from insights from population genetics (Lynch and Walsh 1998).

A quantitative trait, such as height, is a phenotypic trait that can be numerically scored. An individual's quantitative trait value Z_i consists of numerical additive genetic A_i, non-additive genetic B_i, and environmental components E_i, that combine to give the individual's trait value: e.g. $Z_i = A_i + B_i + E_i$. The additive genetic component A_i is referred to as a breeding value (Lynch and Walsh 1998). The value of the environmental component of the phenotype E_i can be determined by the environment and developmental noise: e.g. $E_i = f(N_t, \varepsilon)$ where f is a function and ε is a Gaussian distribution with a mean of zero and a variance that describes

developmental noise (Falconer 1960). If the environmental component of the phenotype varies with the biotic or abiotic environment (e.g. population size N_t), any particular breeding value can express a different phenotypic trait value in different environments: a process termed phenotypic plasticity (Pigliucci et al. 2006).

The contribution a breeding value makes to a phenotype is usually assumed to remain constant both throughout life and across generations. However, in some cases, breeding values may vary with the environment: e.g. $\mathrm{A}_i = g(N_t)$ where g is a function (Wilson et al. 2006). Such scenarios can occur if a genotype's contribution to a phenotypic trait value is determined by the level of gene expression, and environmental drivers determine gene expression.

When breeding values change with the environment (or with age) they need to be specified as environment- and/or age-specific. This assumes that a phenotypic trait such as body size of individual i in a cold environment is a different trait compared to body size of individual i in a warm environment, or metabolic rate at age x is a different trait to metabolic rate at age $x + 1$ (Wilson et al. 2006). What is inherited under this scenario, is a function describing how a breeding value changes with age, with the environment, or with both. In the simplest case, the function for each parent is a vector of values describing each parent's breeding value at each age and/or in each environment (Lande and Arnold 1983). In more complex cases, a smooth function specified with individual-specific parameters describes each parent's breeding value at each age and in each environment, and the parameters are inherited (Schaeffer 2004). Functions may capture genetic covariation across ages or environments (Wilson et al. 2006).

Regardless of the complexity of the genotype-phenotype map, or the way it is characterized or assumed, or how it varies with age or environment, selection on a phenotypic trait can result in change to allele frequencies or breeding values, and this change can then be passed across generations. However, much as with the dynamics of individual and mean fitness, the influence

of the environment on phenotypic expression can be complex. One example of this complexity arises in resource-limited environments.

Many phenotypic traits, and particularly those that develop with age, are partly determined by the quantity of resources an individual has invested into the environmental component of the phenotype (Schlichting and Pigliucci 1998). For example, large body size may reflect a large breeding value, but it also likely reflects past successes at detecting, acquiring, and efficiently utilizing food.

Assume that an individual with a large breeding value for a phenotypic trait associated with detecting, acquiring or utilizing limiting resources in a particular environment accesses more resources, or more effectively uses those resources that it acquires, than individuals with smaller breeding values (Houle 1991). Because fitness is determined by access to resources and their efficient use, the phenotypic trait will be under positive selection. As large phenotypic trait values infer higher fitness, it will be adaptive to invest some resources that are acquired into the environmental component of the phenotype. Because individuals with a large value of A_i are better at detecting, acquiring or utilizing resources, they are also expected to have more resources available to invest in the environmental component of the phenotype. Assuming they do this, they will large values of the environmental component of the phenotype E_i. Under this scenario, breeding values and the environmental component of the phenotype will positively covary – e.g. $\mathrm{cov}(A_i, E_i) > 0$.

Let us now assume that food is the limiting resource. Under such a scenario, as evolution proceeds and the average individual evolves to be genetically better at detecting and acquiring food, then food will become scarcer. If these resources impact development of the environmental component of the phenotype as argued above, then this could generate a negative temporal trend in the mean value of the environmental component of the phenotype that counters any increase in the mean breeding value with time. Such a temporal negative

covariance between the mean breeding value and the mean of the environmental component of the phenotype has been referred to as cryptic evolution as the mean phenotype may not itself evolve, but the mean breeding value does change (Hadfield et al. 2011).

The process described above may play a role in determining evolutionary dynamics in the wild (Merilä et al. 2001a). It could help explain the success of quantitative methods at predicting evolution in constant environments where resources are not limiting (Lynch and Walsh 1998), but their frequent failure in food-limited natural ones (Merilä et al. 2001b).

In artificial environments, where resources are not limiting and are easily detected and acquired regardless of an individual's phenotype, artificial selection primarily operates on the utilization of resources, and in particular on how they are invested in desirable phenotypic traits. A consequence of this is there is little competition over resources, and opportunities for covariances between breeding values and the environmental component of the phenotype to arise are lacking. Statistical approaches used in quantitative genetics are not designed to detect such types of covariances, and in their absence the methods work well. In contrast, in free-living populations, predictions of evolutionary change rarely tally with observation, particularly for traits like body size that are likely determined by the past successes of individuals in accessing and utilizing resources (Merilä et al. 2001b). One untested hypothesis for this is that when resources are limiting and their detection, acquisition and utilisation is intensely competed for, covariances between breeding values and the environment component of the phenotype are likely to arise and could hinder statistical characterisation of evolution.

I now turn to consider genetic inheritance – a process that is not influenced by resource availability, but is rather a straightforward inevitability.

Inheritance — genetic and otherwise

As already defined, evolution is a change in allele (or breeding value) frequencies. Allele frequency change requires genotype frequencies to change within a generation. However, a change in genotype frequencies within a generation does not always result in a change in allele frequencies. For example, in a population at genetic equilibrium where heterozygote advantage operates at a locus (i.e. the heterozygote genotype has a higher per time step fitness than either homozygote), genotype frequencies change within a generation, but allele frequencies do not (Charlesworth 1994, Crow and Kimura 2009). Allele frequency change within a generation is additive genetic change. Evolution requires additive genetic variation between individuals, and adaptive evolution requires change in allele frequencies to be attributable to selection.

In diploid species, an offspring inherits one allele at each locus from each of its parents. Its genotype is consequently expected to be midway between that of its parents. However, because its parents each have two alleles at each locus, this does not mean that all offspring born to the same parents are going to have identical genotypes. The variation in genotypes at a locus among offspring born to the same parent is caused by meiotic segregation and the random selection of gametes at conception, and is termed the segregation variance (Slatkin and Lande 1994).

Regardless of the segregation variance, in large populations the distribution of allele frequencies is not expected to change between individuals selected to be parents and their offspring. Adaptive evolution consequently proceeds with selection generating change in allele frequencies within a generation, before this change is represented in the allele frequency distribution of offspring at birth (Falconer 1960, Crow and Kimura 2009). The mating system determines how offspring genotypes are formed from the allele frequencies of parents, with assortative, dis-assortative and random mating each generating different offspring genotype distributions from identical parental allele frequency distributions (Nunney 1993). If different

genotypes have different fitnesses, either via additive or non-additive genetic processes, patterns of mate choice can be selected for and can influence population dynamics.

When the genotype-phenotype map is not known, and allele frequency change due to selection within a generation is not explicitly monitored, evolution is inferred from change in the mean phenotype. The phenotypic gambit, that assumes phenotypic changes represents underlying evolution, requires a number of assumptions (Hadfield et al. 2007). Widely used quantitative genetic theory assumes that an individual's breeding value for a particular trait consists of very small additive contributions from genotypes at a large (technically infinite) number of unlinked loci. This assumption means that an offspring's breeding value can be specified as the mid-point of the parents' breeding values plus a random number drawn from a Gaussian distribution with a mean of zero and a variance specified to capture meiotic segregation (and potentially mutations) (Lynch and Walsh 1998). If it is a function that is being inherited, offspring parameter values will be the midpoint of parental parameter values plus a random deviation.

Recombination can generate novel genotypes in each generation as can mutation. Mutations come in a wide range of forms such as single base pair substations, inversions, the formation of de novo genes, insertion and deletions (Stenson et al. 2009). In addition, in some species new genetic variation can be generated via horizontal gene transfer such as via the incorporation of retroviruses into host genomes (Gifford and Tristem 2003). Such genes can contribute to significant evolutionary innovation including, for example, the development of the placenta (Dunlap et al. 2006). Current research is providing significant insight into the dynamics and evolutionary consequences of mutation, but is not discussed further here.

Parents and their offspring genetically resemble one another. However, recent research has revealed that the environmental components of their phenotype can also resemble one another via epigenetic inheritance (Jablonka and Raz 2009). Parents can influence the environmental component of their offspring's phenotype by manipulating resource levels or composition of

the developing young in eggs or in utero, which in turn influences patterns of gene expression and phenotypic development. In species showing parental care, further manipulation can occur via milk or food provisioning. Parents in good condition can potentially produce offspring in better condition that their poorer-condition conspecifics by investing more heavily in them, generating a covariance between the environmental components of parent and offspring phenotypes (Wu and Suzuki 2006). Research into the role of epigenetic inheritance in ecological and evolutionary dynamics is still in its infancy, but it is clear that non-genetic inheritance has the potential to alter selection and consequently population and community dynamics (Coulson et al. 2017).

The sections above demonstrate that competition for limiting resources determines population dynamics, selection, development, and evolution. Resources are consequently central to biological understanding. For consumer species, many resources such as food, shelter, enemy-free space and (of course) mates are biotic. Species interactions are consequently primarily about access to resources, stopping other individuals (be they of your own species or another) accessing them, or avoiding becoming a resource for something else.

Resources, species interactions and trophic cascades

In the same way that the population dynamics and survivorship and fertility schedules can be influenced by evolution, so too can species interactions (Doebeli 1997, Thompson 1999). To understand how, it is useful to start with standard treatments of interacting species.

In simple models, the effect of one species on the population growth of another is captured with interaction coefficients that are multiplied by the population sizes of interacting species (Allen 1975). Interaction coefficients capture processes such as functional responses and the conversion rates of resources into offspring by consumers. Interaction coefficients typically

capture the mean fitness (e.g. population growth) impacts of one individual of an interacting species on an individual of a focal species (Berlow et al. 2004).

In more complicated models, interaction coefficients can be replaced with functions, and the population size of interacting species replaced with information on population structure (Inouye 2001, McCoy and Bolker 2008, Bassar et al. 2017). Such formulations are useful as empirical observations have revealed that the outcomes of interactions between two individuals are often dependent upon their phenotypic trait values (Bolnick et al. 2011). For example, whether an individual of a predator species succeeds in capturing an individual of a prey species can depend upon phenotypic traits associated with prey capture in the predator, and those associated with predator avoidance in the prey (Dawkins and Krebs 1979). If the predator population is food-limited, and the prey population is limited by the availability of enemy-free space, then we might expect traits in each species to co-evolve. However, if the predator population does not evolve in response to evolution in the prey, it is expected to decline in numbers, and the primary cause of mortality or failure to reproduce in the prey may change from predation as a consequence of the evolution of anti-predator phenotypes.

There are a wide range of possible outcomes from a change in key limiting resource (Figure 2). The dynamics that are observed depend upon whether the limiting resource can be defended or not, the rate at which the resource renews (or not), and whether coevolution occurs between the consumer and the producer. However, answers to these questions provide insights into how a change in limiting resource may result in relaxed selection on some traits, and novel selection regimes on others.

When the outcomes of species interactions are a function of the phenotypic traits of interacting individuals, species interactions themselves can change as phenotypic traits evolve (Doebeli 1997). The strength of an interaction between two individuals is typically specified in models as a measure of distance between the phenotypic traits of the interacting individuals (Werner

and Gilliam 1984, Bassar et al. 2017). Interactions are usually considered to be stronger when the distance between the traits is small. Under this assumption, we may expect selection for traits in consumer species to reduce the distance to traits in resource species they utilize. In contrast, phenotypic traits in resource species may evolve to be further away from those in the species that consume them. Antagonistic coevolutionary arms races consequently involve evolution trying to maximize the distance between traits from the perspective of one species, while trying to minimize it from the perspective of the other. Such arms races can ultimately result in the evolution of specialization (Roughgarden 1972).

Fit individuals are those that are successful at detecting, acquiring or utilising resources. In a competitive environment, selection will favour phenotypes that are able to access resources that conspecifics are not utilising, be those resources food, enemy-free space, mates, shelter, nutrients, minerals or water. Many populations consist of individuals that specialise on only a part of the niche that the entire population utilises, with different individuals expressing different phenotypic traits (including behaviours) (Bolnick et al. 2002). Having a novel phenotypic trait compared to others in the population may increase an individual's fitness if it aids access to a novel resource by minimising the distance to traits in the resource species such that it is able to effectively exploit them. Such specialisation can result in frequency-dependent selection, a process that maintains additive genetic variation (Roughgarden 1972). For example, if two groups of individuals specialise on different resources that each renew at constant rates, then when each group becomes rare via a stochastic process, there will be more resources per capita for the individuals that use that resource, resulting in an expected increase in fitness and an increase in population size of that group. When individual specialisation is determined by phenotypic traits, this can result in competitive interactions between individuals varying as a function of the distance between their trait values. Such trait-mediated interactions – either within or between species – can have significant impacts on the diversity of resources

that are utilised by a species, patterns of resource use, community dynamics, selection, and evolution (Roughgarden 1972, Bassar et al. 2017).

In some extreme cases, evolutionary change may be able to impact species interactions to such an extent that they result in trophic cascades. However, most empirical work on trophic cascades focuses on ecological causes (Polis et al. 2000). Nonetheless, because ecological change can occur as a function of evolution, it is worth hypothesizing on the circumstance when evolution might result in a trophic cascade.

The most pronounced trophic cascades occur when the composition of the dominant producer species changes. For example, in Yellowstone, it is argued that the decline in elk numbers due to increased wolf predation has led to forests replacing grasslands (Fortin et al. 2005). The effect is currently localized to small areas as two decades is not sufficient time for a forest to displace a grassland. However, if the effects observed on small spatial scales are eventually repeated on a larger scale, the reintroduction of wolves will be responsible for a pronounced trophic cascade as forests replace grasslands on the Northern range.

Less pronounced trophic cascades can occur when only a small part of a food web is affected, with impacts localized to a few species. For example, a decline in the density of a predatory bird may influence the dynamics of its passerine prey and the insects on which they feed, but effects on the species structure of the primary producers is likely to be minimal (Borer et al. 2005). Those species directly impacted may experience altered causes of death or failures to reproduce, and novel selection pressures, but community structure and ecosystem function is unlikely to be significantly impacted to a sufficient extent to generate a pronounced trophic cascade.

When might evolution result in a pronounced trophic cascade where the composition of the dominant producer species changes altering the structure of an ecosystem? Imagine the

evolution of a pathogen that kills the dominant tree-species in a species-poor forest. The loss of trees has the potential to impact the majority of species within the community, and as the forest disappears and is replaced by scrub or grassland, many tree-dependent species will disappear to be replaced eventually with specialists on the new dominant producer species. Similarly, the evolution of anti-predator defenses in a large herbivore has the potential to result in its population growing in size and reducing, or eliminating, tree recruitment in forests. Over time, such an evolutionary adaptation could result in a pronounced trophic cascade as the system switches from forest to grassland.

When evolution results in a pronounced trophic cascade, it will have done so because evolution in one species altered the dominant causes of death or failure to reproduce in dominant producer species within a community. The dominant species was unable to evolve a response to the new cause of death or failure to reproduce prior to extinction. Such dynamics demonstrate an extreme case of a process where the biotic environment imposes a constraint that organisms struggle to adapt to. However, such constraints will only result in extinction in extreme cases. In other cases, such constraints limit options for species to adapt to a particular limiting resource. Guppies in high-predation environments face such a constraint.

The wolf fish and pike cichlid predators are orders of magnitudes larger than their guppy prey. Regardless of the size of an individual guppy, it is vulnerable to predation from these predator species (Reznick and Endler 1982). There seems little realistic chance of guppies evolving to a sufficiently large size to escape predation in high-predation environments, as they would need to evolve to be at least an order of magnitude larger than they currently are. In response to the ever-constant limitation of enemy-free space, and instead of evolving to be large enough to escape predation, guppies have evolved small adult sizes and a fast life history: they live their lives quickly, aiming to successfully reproduce before an inevitable death in the jaws of a

predator (Reznick and Bryga 1987). This leads us to the evolution of body size – one of the most studied, and important, phenotypic traits of all.

The case of body size evolution

In the absence of processes that select for an increase in body size, body size should evolve to be small (Kutschera and Niklas 2015). The reason for this is that key traits associated with resource utilization are metabolic. Metabolic rate allometrically scales with body size and other traits that strongly correlate with it such as life expectancy and generation length. The metabolic theory of ecology provides the reason why species should evolve to be small, all other things being equal.

Central to the metabolic theory of ecology is the observation that larger animals require more energy than those that are smaller, but that smaller animals have a higher metabolic rate per unit mass (termed relative metabolic rate). There are a number of reasons proposed to explain why larger animals have a lower relative metabolic rate than those that are larger, ranging from the challenges of dissipating heat generated by metabolism (an elephant with the metabolic rate of a shrew would spontaneously combust) or of distributing resources to all areas of the body (West et al. 1997), and the fact that energy absorption occurs across a surface but body weight is a volume (Kooijman and Kooijman 2010). These arguments predict that total metabolic rate should scale with body mass as a power law with an exponent of either 2/3 or 3/4. The precise value does not matter for my argument. What is key is that this scaling exponent is less than unity.

Because evolution is about the rate at which alleles spread through populations (Tuljapurkar 1990a, Crow and Kimura 2009), if all else is equal, strategies with small body sizes that can rapidly turn resources into copies of themselves should outcompete strategies with larger body sizes that have slower life histories (Kutschera and Niklas 2015). In the absence of some

process selecting for individuals to be large, all species should consequently be small. So what processes can select for large body size? There are a number of possible drivers of an increase in body size, ranging from competition for resources such as food and mates, through to evolutionary arms races.

Body size may evolve as a way to defend a resource. For example, island populations of the passerine bird, the silvereye (*Zosterops lateralis*), tend to live at higher density than their mainland ancestors, are limited by food availability, and have evolved larger body sizes to allow them to defend limited food (Clegg and Owens 2002, Robinson-Wolrath and Owens 2003). In contrast, in cases where resources are not defensible, body size is not selected to increase. Food-limited populations of ungulates on islands tend to evolve smaller body sizes than their island counterparts (Van Valen 1973). The silvereyes and ungulates provide examples of aspects of the island rule, where large mammals and birds tend to evolve to smaller sizes, while smaller ones tend to evolve to larger sizes (Lomolino 1985). An untested hypothesis that could explain the island rule is that when food is limiting and can be defended, selection for large size occurs, but when it is limiting and cannot be defended, selection occurs for a reduction in size.

Body size may also evolve in response to predation. For example, the large size of some herbivorous and carnivorous Jurassic dinosaurs has been proposed to have arisen as a consequence of an arms race (Sander et al. 2011). Large adult sauropods may have been immune from predation for times during their evolution, but as ever larger carnivorous dinosaurs evolved, they became vulnerable, increasing selection for larger size.

A third route to large body size is the ability to access resources that may be unavailable to smaller individuals. Obvious examples include evolve increased height to reach resources unavailable to those individuals that are shorter, but a more nuanced example comes from life in fluctuating environments (Orzack and Tuljapurkar 1989, Tuljapurkar 1990a). In many

systems in seasonal environments, resources are limiting during only part of the year. For example, in food-limited ungulates such as Soay sheep (*Ovis aries*) living in seasonal environments, grass is abundant during spring and summer but rare or unavailable during the winter months. A large body size may well have evolved to cope with this period of resource limitation, as body size determines the likelihood of an individual surviving over-winter when food is scarce (Pelletier et al. 2007). Larger individuals are able to store more fat reserves than those that are smaller, and have lower relative thermoregulatory costs than their smaller conspecifics. In support of this argument, as winters have shortened, smaller adult sheep are able to survive through the winter, and this resulted in a reduction in average body size (Ozgul et al. 2009). Body size evolution to survive a period of resource scarcity is an example of a phenotype evolved in response to variable environments by reducing the impact of V_E on a.

There are consequently a number of plausible ways to select for larger body sizes, of which a few are listed above. As soon as processes selecting for large body size are removed, we would expect evolution to smaller body sizes given the allometric relationships between metabolism and size. Such characterisation of energy budgets is useful as it provides mechanistic links between energy availability and the evolution of size-related traits. However, energy is only one of multiple possible limiting resources. Organisms consist a large number of phenotypic traits, each of which may have evolved in response to a different limiting resource and causes of death or failures to reproduce – body size can evolve in response to competition for mates, for example. As the biotic composition of a community an organism is embedded within changes, selection on trait combinations will also vary. Coupled with variation in the abiotic environment that may determine periods of resource availability for some resource types, it would be a surprise if species interactions have not significantly contributed to the enormous diversity of life we observe.

Understanding which resources are limiting, and how these relate to population limitation and the primary causes of death and failures to reproduce is clearly central to understanding the dynamics of populations, communities, fitness and evolution. The complex feedbacks between evolution and species interaction are a form of eco-evolutionary dynamic – a field that has becoming increasing popular over the past decade.

Eco-evolutionary feedbacks

Eco-evolutionary feedbacks are currently a hot topic in biology (Hendry 2016). However, they are not always well-defined, and for this reason, compelling empirical evidence for them is relatively sparse. Evolution is defined as a change in allele frequencies (Charlesworth 1994). Ecology is less well-defined, but is frequently assumed to be measured by the dynamics of populations, communities or ecosystems. If we stick with this definition, we can define ecological change as being a change in the population, community or ecosystem dynamics.

Given this definition, an eco-evolutionary feedback consequently requires change in allele frequencies to result in a change to the dynamics of a population, community or ecosystem which in turn results in a change in selection pressure that alters the course of evolution and so on *ad infinitum* (Hendry 2016). Such dynamics occur in one of the simplest possible models of ecology and evolution.

As I have already shown, if a statistical relationship between a trait and fitness remains constant over time (Figure 3(a)), generating a persistent non-zero selection differential, evolution will result in hyper-exponential growth (Figure 3(b)) – in other words the population growth rate (and consequently the population dynamics) change in response to selection and evolution (Witting 2000a, b). This change feeds back to impact the strength of selection (Figure 3(d)) by altering the denominator of the selection differential in equation 1 (e.g. the population growth rate), and also possibly altering the numerator too (Figure 3(c)). Whether the numerator

changes will depend upon how evolution alters the distribution of fitness and the trait, which in turn depends upon patterns of inheritance (Coulson et al. 2017). What all this means is that for a constant fitness function and no resource limitation, evolution alters the population growth rate, which in turn alters selection and so on *ad infinitum*. I am not convinced that most people would consider this as an eco-evolutionary feedback *per se* as it does not capture the complexity often invoked in tomes about eco-evolutionary feedbacks (Hendry 2016). However, if this definition is accepted, then standard density-independent models of evolution are a form of eco-evolutionary feedback.

An alternative definition of eco-evolutionary feedback would involve evolution altering the statistical association between the trait and fitness (Figure 2). In order to do this, evolution must alter intra- or interspecific interactions in such a way that they feedback to impact selection on the focal trait (Figure 5). Such a process will occur in systems where resources are limiting, where density- and frequency-dependent selection operates, or where coevolution is observed (Roughgarden 1971, Thompson 1999). In other words, we might expect such dynamics in systems where the fitness function includes the size or structure of the focal population, or the population of an interacting species. For example, consider a density-dependent fitness function,

$$w(Z) = (\beta_0 + \beta_N N_t) + \beta_Z Z$$

where the βs are parameters and inheritance is clonal. We start with a Gaussian distribution of Zs, $N_{t=1}(Z)$ with a mean of $\bar{Z}_{t=1}$, and assume that $\beta_Z > 0$ and $\beta_N < 0$. Because $\beta_Z > 0$ individuals with larger values of Z_i will have higher fitness and clonal inheritance means the location of the distribution of phenotypes will change location such that $\bar{Z}_{t=2} > \bar{Z}_{t=1}$ (Figure 3(a)). The distribution of individual fitness $w(Z)$ will also change such that mean fitness $\overline{w}_t = \lambda_t$ will also increase with time, as will population size N_t. However, because $\beta_N < 0$, as

evolution proceeds the term $\beta_0 + \beta_N N_t$ becomes increasingly less positive, and the statistical association between the trait and fitness changes with time. In other words, if I were to estimate a regression at each point in time between the trait and fitness as evolution proceeds, I would observe the intercept of the regression becoming increasing less positive with time (Coulson et al. 2017).

Fitness functions that include the population size and/or structure of the focal species or an interacting one, or a resource, are common, and this suggests that eco-evolutionary feedbacks defined in this manner are widespread. However, they may be quite hard to detect as they will likely require significant amounts of data. Nonetheless, in some cases eco-evolutionary feedbacks have the potential to generate pronounced ecological and evolutionary dynamics.

Tying the strands together

The sections above lead to two conclusions:

1. In order to understand ecological and evolutionary dynamics, it is necessary to understand, (i) the mating system, and in particular how alleles combine to form genotypes, (ii) mutation, (iii) how genotypes and resources determine phenotypic development, (iv) how phenotypes determine expected survival and reproduction in different environments, and (v) how the biotic and abiotic environment determines each of these processes. As evolution proceeds, each of these processes can change generating a wide range of ecological and evolutionary dynamics and eco-evolutionary feedbacks.
2. Within any population, dynamics are determined by the key causes of death and failures to reproduce. These determine what processes limit the population, and are a function of the resources that are most limiting. Specific phenotypic traits evolve in response to the key limiting resources, and evolution can eventually result in a switch in the key

causes of death or failures to reproduce and the dominant limiting resources. In many cases, limiting resources are biotic, with access to them frequently determined by both the biotic and abiotic environment. Evolution has the potential to generate change in species interactions, community dynamics, and even pronounced trophic cascades under some scenarios.

I summarize the links between different levels of biological organization described above in a diagram (Figure 5). The figure reveals the way that the biotic and abiotic environment can loop back to influence the fundamental processes that link alleles, genotypes and phenotypes, and how the dynamics of these quantities can generate population, community and ecosystem dynamics. Much of the text above considers the dynamics of these linkages.

We can formally model these linkages and feedbacks. To do so requires modeling the dynamics of a distribution of components of the phenotype that combine to form phenotypes within each population of interacting species (Barfield et al. 2011, Coulson et al. 2011, Childs et al. 2016, Coulson et al. 2017). The distribution consists of the number of individuals within a population with each combination of components of the phenotype. The distribution can be multivariate. For example, if a phenotype is made of genetic and environmental components, the bivariate distribution of these two components is modeled (Coulson et al. 2017). Similarly, if two phenotypes within a population are modeled, and each consists of a genetic and environmental component, we would construct a multivariate distribution with four dimensions – one for each of the two genetic components of the phenotypes and one each for the two environmental components. The components can be specified to covary or be independent. The distribution of components of the phenotype can be further classified by additional variables such as age, sex, or location (Schindler et al. 2013, Ellner et al. 2016).

The univariate distribution of a phenotype can be constructed from the multivariate distribution of its components using a genotype-phenotype function. For example, if the phenotype is

determined by a sum of genetic and environmental components then $Z = f(A, E) = A + E$. Functions are also specified to project the distribution of phenotypic components being modeled from time t to time $t + 1$. Two functions describe how the distribution of components of the phenotype shrink and grow with time as a function of survival and reproduction, and two other functions describe how individuals transition between different parts of the distribution as a function of phenotypic development among survivors, and the construction of phenotypic components of offspring as a function of those of their parents (Easterling et al. 2000, Coulson 2012). Each of these functions can be specific to each additional classifying variable such as age, sex or location.

The survival function describes how the phenotype influences the probability of survival; it acts to remove individuals from the distribution. Key causes of mortality or limiting resources such as the risk of predation or food availability can be included into the function as additive effects, or in interaction with the phenotype (Ellner and Rees 2006, Coulson 2012). Trait-mediated effects can also be included in the function (Bassar et al. 2016, Bassar et al. 2017). In models capturing eco-evolutionary feedbacks, the survival function might include effects of the size and/or structure of the population of the focal species or an interacting one.

The reproduction function acts to add individuals to the distribution. In two sex models, the phenotypes of parents determine patterns of mate choice and the number of offspring produced by each mating pair (Schindler et al. 2013). Environmental drivers, including the size and phenotypic structure of the populations of the focal species or of interacting ones can also be included in the function.

The development function describes how the environmental component of the phenotype develops among surviving individuals, with potentially different genetic components contributing to the phenotype at different ages (Coulson et al. 2017). The function can contain descriptors of the biotic and abiotic environment, such as resource availability. The

development function is a probability function and it does not directly alter the size of the distribution being modeled (Easterling et al. 2000).

The inheritance function describes the construction of the distribution of components of the phenotypes from the distribution of mating pairs formed by the reproduction function. For example, the genetic component of the offspring phenotype is specified to be the midpoint of parental values with some segregation (and potentially mutational) variance added (Childs et al. 2016). The environmental component of the offspring phenotype can be independent of parental components, or a function of them (Coulson et al. 2017). As with all other functions, the abiotic or biotic environment can influence construction of the environmental component of the offspring phenotype.

These functions can be combined into a model in two frameworks: discrete-time structured population models and in particular integral projection models (IPMs) (Ellner et al. 2016), and individual- (or agent-) based models (IBMs) (Grimm and Railsback 2013). Both class of model can track distributions of alleles, genotypes, and phenotypes within one population, or multiple populations of interacting species. The models can describe how the mating system determines the way that alleles combine to form genotypes, how genotypes develop into phenotypes, and how phenotypes determine which individuals die, reproduce, and with whom. Fluctuations in the size, locations and shapes of the allele, genotype, and phenotype distribution determine population dynamics of each species and evolution, and the population dynamics of all the modeled species determine community and ecosystem dynamics. There is a significant literature on both frameworks.

The survival and reproduction functions determine the population growth rate over a time step as a function of the distribution of phenotypic traits and other drivers (Ellner and Rees 2006). In addition, these functions determine selection at each point on the trait (Coulson et al. 2010, Wallace et al. 2013). Both functions can also change the distribution of the components of the

phenotypic traits by removing, or adding, individuals at variable rates in different parts of the distribution (Coulson 2012). The development and inheritance functions act to rearrange the distribution of components of the phenotypic traits as individuals develop or pass aspects of the phenotype onto their offspring (Coulson et al. 2017). These functions do not directly contribute to selection or the population growth rate over a time step, but they indirectly effect both quantities by impacting the distribution of phenotypic traits for selection to operate on (Wallace et al. 2013). All four functions combine to determine the life history (Steiner et al. 2014), and hence evolution of traits that act to reduce V_E, the long-run stochastic growth rate, and distributions of population sizes. The contribution of each function, or parameters in each function, to each of these quantities to evolution and phenotypic trait, population and community dynamics, and species interactions, can be examined using perturbation analyses (Ellner et al. 2016).

To date, a single model that captures all the levels of organization and feedbacks in figure 5 is yet to be constructed. However, the underlying theory required to make each link has been developed. For example, within the IPM framework, Adler *et al.* (Adler et al. 2010) and Bassar *et al.* (Bassar et al. 2017) have developed models of interacting species, Coulson *et al.* developed models that include single locus genotype-phenotype maps (Coulson et al. 2011), Childs *et al*. (Childs et al. 2016) and Coulson *et al*. (Coulson et al. 2017) incorporated quantitative genetic genotype-phenotype maps into models, Schindler *et al*. developed two sex models that incorporated flexible breeding systems and patterns of mate choice (Schindler et al. 2013), a number of authors have incorporated density-dependence, environmental stochasticity and age-structure into models (Childs et al. 2004, Ellner and Rees 2006, Coulson et al. 2010, Coulson et al. 2011, Coulson 2012, Simmonds and Coulson 2015, Ellner et al. 2016), Bassar et al. included trait-mediated interactions (Bassar et al. 2016), Vindenes added demographic stochasticity (Vindenes and Langangen 2015), and Smallegange incorporated

mechanistic growth rules as a function of energy budgets (Smallegange et al. 2017). In addition to adding in the various feedbacks and links in figure 5, a number of authors have shown how numerous quantities of interest to biologists can be calculated from models, and the dynamics of these quantities explored using sensitivity and elasticity analysis. For example, Tuljapurkar has developed methods to calculate and analyze distributions of life history quantities such as generation length, life expectancy and lifetime reproductive success (Tuljapurkar et al. 2009, Steiner et al. 2014), while Ellner *et al.* and Tuljapurkar *et al.* have also focused on population dynamics, calculating the deterministic population growth rate, the long-run stochastic growth rate, and distributions of population size (Tuljapurkar et al. 2003, Ellner and Rees 2006, Plard et al. 2016). Coulson *et al*. and Wallace *et al*. have shown how selection differentials can be estimated and analyzed (Coulson et al. 2010, Wallace et al. 2013), Coulson *et al*. explored the dynamics of allele and genotype frequencies (Coulson et al. 2011) building on seminal work by Charlesworth using matrix models (Charlesworth 1994), Childs *et al*. (Childs et al. 2016) and Coulson *et al.* (Coulson et al. 2017) calculated additive genetic variances and heritabilities from models, Childs *et al.* competing strategies against one another in a game theoretic framework (Childs et al. 2004), while Simmonds *et al.* examined how climate change might influence phenotypic and population dynamics in Soay sheep (Simmonds and Coulson 2015). Caswell et al. show how mutation can be included into matrix models in a manner that could be used for IPMs (De Vries and Caswell In press).

IPMs can be used as skeletons to construct IBMs, with IBMs and IPMs of the same structure providing equivalent results (Ellner et al. 2016). The difference between IBMs and IPMs is that IPMs track the distribution of all possible allele, genotype and phenotype combinations regardless of whether any individuals are alive with each combination at any point in time, while IBMs only simulate the fate of those individuals that are born. IBMs are consequently

easier to construct when there are multiple genotypes or phenotypes being modeled in each interacting species than IPMs, while IPMs often easier to analyze.

Work is required to take the existing theory and to combine it into a single model, but once this is done, it will be possible to simultaneously explore how altering the primary causes of death or failure to reproduce can generate the types of ecological and evolutionary dynamics described above. The challenge will be developing simulation models that run sufficiently quickly they can be analyzed to provide useful insight.

The strengths and weaknesses of such models

The potential feedbacks in Figure 5 are numerous, complex, and can play out over a range of timescales. They reveal how evolution can feedback to generate ecological change and *vice versa*, and how change in one species can impact dynamics in other species, potentially feeding back once again to impact the focal species. The complexities captured in the figure help explain the numerous empirical observations of the myriad ways that species can interact via mate choice, development, and demography. Graphical representation of the feedbacks can also be useful in posing hypotheses as how feedbacks via particular processes might generate specific patterns. However, is modeling such complexity desirable?

At one level it is. Being able to construct models to capture all the feedbacks allows construction of realistic simulations of how particular processes can influence evolution and the dynamics of phenotypic traits, life histories, populations, species interactions, and communities. However, the downside is that the complexity of the models will likely mean that analytical solutions are unachievable. This means that the insights gained from a particular model may not be general, but may rather be specific to the particular parameterization used. Nonetheless, I argue that such insights are extremely useful.

The feedbacks captured in Figure 5 are general, although the dynamics they produce will inevitably be dependent upon the abiotic environment and the collection of coexisting species within a community. The key questions are under what conditions are particular types of dynamics most likely to be observed? For example, when will particular feedbacks select for particular phenotypic traits, life histories or types of species interactions? When will pronounced trophic cascades happen, and when will they not? When will the introduction or removal of species from communities result in pronounced ecological and evolutionary change, and when will it not? Why do we see repeated patterns of evolution such as the island rule, but in other cases evolution appears unpredictable? These are the questions that many empiricists are asking, that have spurred on the modeling approaches I have been involved in developing, and underpin construction of the framework that is summarized in this paper. I believe that models within the framework, although frequently not analytically tractable, can usefully help address key questions in biology.

The construction of simplified models within the framework have already provided useful insight. IPMs and IBMs do not need to capture all the feedbacks in figure 5 explicitly. For example, structured models that finesse evolution have provided accurate insight into life history adaptation and the likely evolutionary consequences of trophy hunting (Traill et al. 2014), while purely ecological models have been used to successfully address questions about optimal allocation of resources to offspring (Schindler et al. 2015), to explain when males can ignored from population dynamic models and when they will impact ecological dynamics (Schindler et al. 2013), to identify optimal size-structured life histories across a range of stochastic, resource-limited environments (Childs et al. 2004), to explain why sheep are shrinking on Scottish islands (Ozgul et al. 2009) but marmots are getting larger in the Rocky Mountains (Ozgul et al. 2010) in the face of climate change, and to explore how trait-mediated

dynamics can generate dynamics that diverge from purely density-dependent ones (Bassar et al. 2017).

I believe that the recent development of models capturing the feedbacks in Figure 5 to capture the types of dynamics described in the earlier section of this paper provide enormous potential to gain further insights into ecological and evolutionary dynamics. However, not everyone is a fan of models, or of the particular class of model that I champion here, or able to construct models themselves (Janeiro et al. 2017). So, what, if anything, can be inferred without recourse to modeling the feedbacks in figure 5?

What can we say without models?

There are cases where a model may not be needed to make predictions, or to explain observations. In this section I return to the example of the guppies and consider how we can explain the observation in terms of change in the key causes of death. However, the guppies are unusual in that they are very well studied. The second example considers emus. I choose these birds because my father-in-law once rescued an abandoned email chick that grew into the worst pet you could possibly imagine (Figure 6). Also, relatively little is known about them – presumably because no sane person would ever consider having such wonderful, but irascible, beasts as study organisms.

Why does the loss of predators generate repeated evolution in guppies?

In high predation guppy populations, the primary cause of death is predation, the population is predator-limited, and the limiting resource is enemy-free space. The age and size at sexual maturity is low, and there are relatively few failures to reproduce. In response to this environment, guppies have evolved a number of traits, including a rapid maximum swimming speed, a high metabolic rate, and a streamlined bauplan that allows them to minimize the amount of time spent away from enemy-free space when foraging for food. They are typically

found in shoals, a behavioral adaptation to predation. In addition, they have a fast life history that has evolved in order to allow the rapid production of offspring before an eventual, almost inevitable, death due to predation (Reznick and Bryga 1987, Reznick et al. 2001, Travis et al. 2014).

In contrast, in low predation environments, the primary cause of death is thought to be starvation, a key failure to reproduce is females not being in appropriate condition, the population is food-limited, and food is the limiting resource. Guppies adapted to live in this environment have evolved traits that allow them to survive on a broader diet with increased levels of individual specialization (Montiglio et al. 2013), to expend less energy via a lowered metabolic rate (Auer et al. 2018), and a large body size that may help with being able to survive the wet season when competition for limited food is intense and many guppies likely experience a negative energy balance (Reznick et al. 1990). Relaxed selection on traits associated with predator avoidance, including a high maximum swimming speed, results in a less-streamlined morphology and a low maximum swimming speed (Ghalambor et al. 2004). Reduced predation pressure means guppies are no longer constrained to be camouflaged, which results in sexual selection for brighter colors in males (Kodric-Brown 1985).

The guppies reveal that some of the differences in phenotypes between low and high predation environments are due to relaxed selection from the loss of predation, while others are a consequence of novel selection pressure for traits associated with the detection, acquisition and utilization of limiting food. A comprehensive understanding of the eco-evolutionary feedbacks for guppies consequently requires understanding what the causes of death and failures to reproduce are in each environment, which resources are limiting, and how each phenotypic trait is associated with the detection, acquisition and utilization of different types of resources. An exciting experiment would be to make predictions on which phenotypic will evolve, and how, given artificially imposed causes of mortality and failures to reproduce.

How to evolve an emu

Emus have a number of unusual features. They are large for a bird species, they are flightless, they develop their legs extremely quickly (Castanet et al. 2000), they can run fast, they can move vast distances over relatively short periods (Davies et al. 1971), and they can survive in arid environments. We also know from phylogenetic work, that as well as being related to other flightless ratites such as the cassowary and the rhea, they are also related to tinamous, a flighted group of birds found in Central and South America (Phillips et al. 2009).

Emus evolved from a flighted ancestor that was much smaller than they are. They presumably lost flight because it was energetically expensive and was a redundant feature. Many ground feeding birds fly to escape predators, so it is plausible that emus lost the ability to fly during a period when predation was not a significant cause of mortality. Having lost the ability to fly, they increased in body size. There are a number of possible reasons for this. First, the arrival or evolution of predators such the marsupial lion or marsupial wolf may have started an arms race driving an increase in development rate and body size, and an ability to run quickly (Johnson 2006). Alternatively, as the climate became more arid, a rapid development rate and large body size may have allowed emus to develop quickly when resources are abundant, to survive through periods of food scarcity, and to travel large distances in search of food. The ability to run may have helped them move significant distances to find food, water, or mates, or to escape predators.

The primary cause of mortality of adult emus is not known, although humans have hunted them for at least 65,000 years and has likely been a significant cause in recent millennia (Bowler et al. 1970). However, their young are susceptible to predation by dingoes and wild dogs, which likely limits recruitment. The old may die from starvation or thirst if they are unable to source water or food and are not predated. Climate change is predicted to make the arid and semi-arid environment in which they live even more arid, which is expected to result in them being

selected to move even larger distances in search of resources, or to be better able to preserve water.

We can try a though experiment. What would happen if we were to move emus to a tropical, non-seasonal environment with no predators? I hypothesize an evolutionary decline in body size, a shortening of the life history, and the loss of the ability to run fast and move large distances. Unfortunately, such a hypothesis is beyond the length of a standard grant or research career. Perhaps useful insights can be gained from looking at relatives of the ratites, the kakapo, or even the dwarf emus that once inhabited Tasmania, Kangaroo Island and King Island off the Southern coast of Australia (Thomson et al. 2018).

Conclusions

In this paper I propose an approach to predict how a change in limiting factor can generate ecological and evolutionary dynamics. I do this by arguing that the key factors limiting populations are determined by the key limiting resources, and that these determine the phenotypic traits that are subject to selection, and the types of dynamics that are likely to be observed. Structured modeling approaches are sufficiently well-developed that the approach I propose can now be implemented. The next step is to apply the framework to a number of systems where the limiting factor has changed to explain and predict changing ecological, evolutionary and eco-evolutionary dynamics.

Acknowledgements

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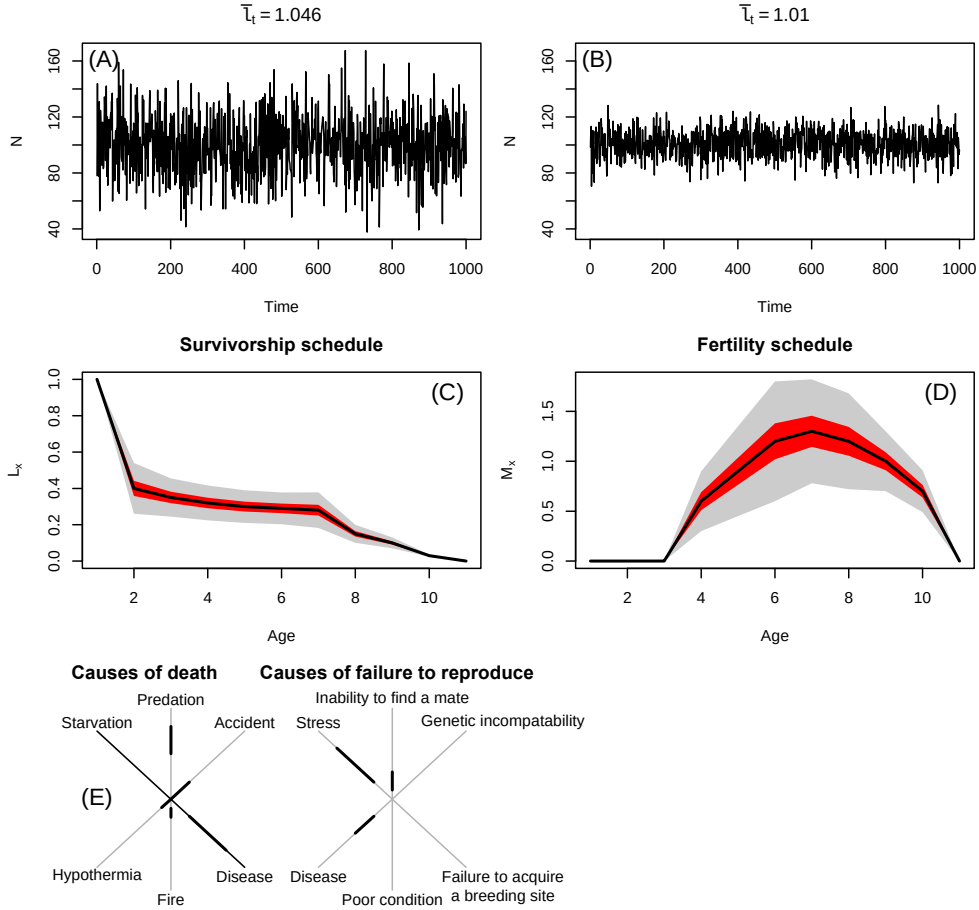

Figure 1. Population dynamics, life history, and causes of death. The population dynamics (A) and (B) or two different life history strategies (C, D), one which has high V_E and one low V_E. The population dynamics (A) of the gray survivorship (C) and fertility (D) schedules are more variable than those (B) of the red survivorship (C) and fertility schedules (D) even though the mean survivorship and fertility schedules are identical for the two strategies (black lines). In (E) an example is provided of the numbers of death or failures to reproduce attributable to different causes in a hypothetical population over a period of time (black lines). If these causes of death or failures to reproduce were to change, the life history and population dynamics would be expected to change too.

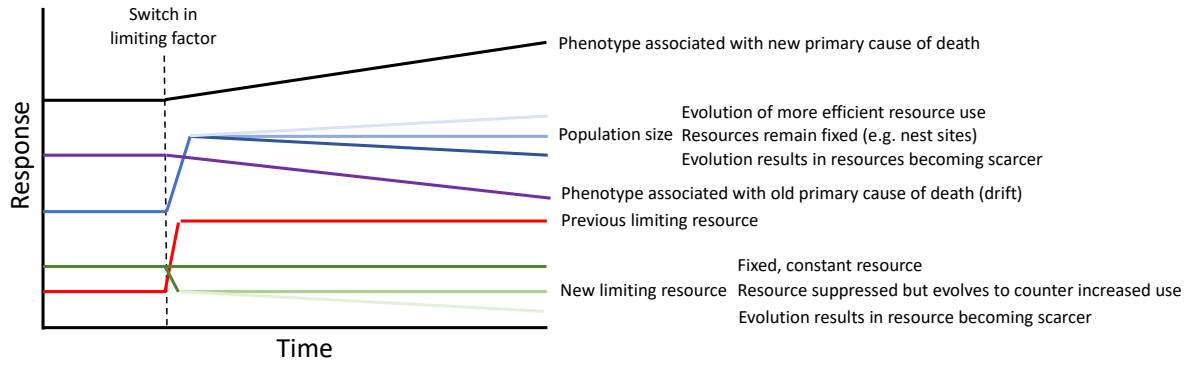

Figure 2. A range of possible dynamics of population size, two phenotypic traits, and limiting resources before and after a switch in key limiting factor. These examples are illustrative only, and do not cover the complete range of possibilities. Prior to a switch in limiting factor (dashed vertical line), the population is at a constant size (blue line), phenotypic traits are at an optimum value for that environment (purple and black lines), and the limiting resource at a low level (red line). A second resource (green line) is also present but is not limiting. Following a switch in limiting factor, the initial limiting resource is no longer limiting and increases in abundance (red line), and the phenotype associated with the initial limiting resource drifts to a lower value following relaxed selection (purple line). The second phenotypic trait that is subject to a new selective regime following the switch in limiting resource evolves to increase in value (black line). The population dynamics also evolve (blue lines) evolve but as a function of the dynamics of the new limiting resource (green lines) and in particular whether evolution negatively impacts the resources, whether the resource evolves in response to becoming limiting, or whether the resource is constant and unchanging.

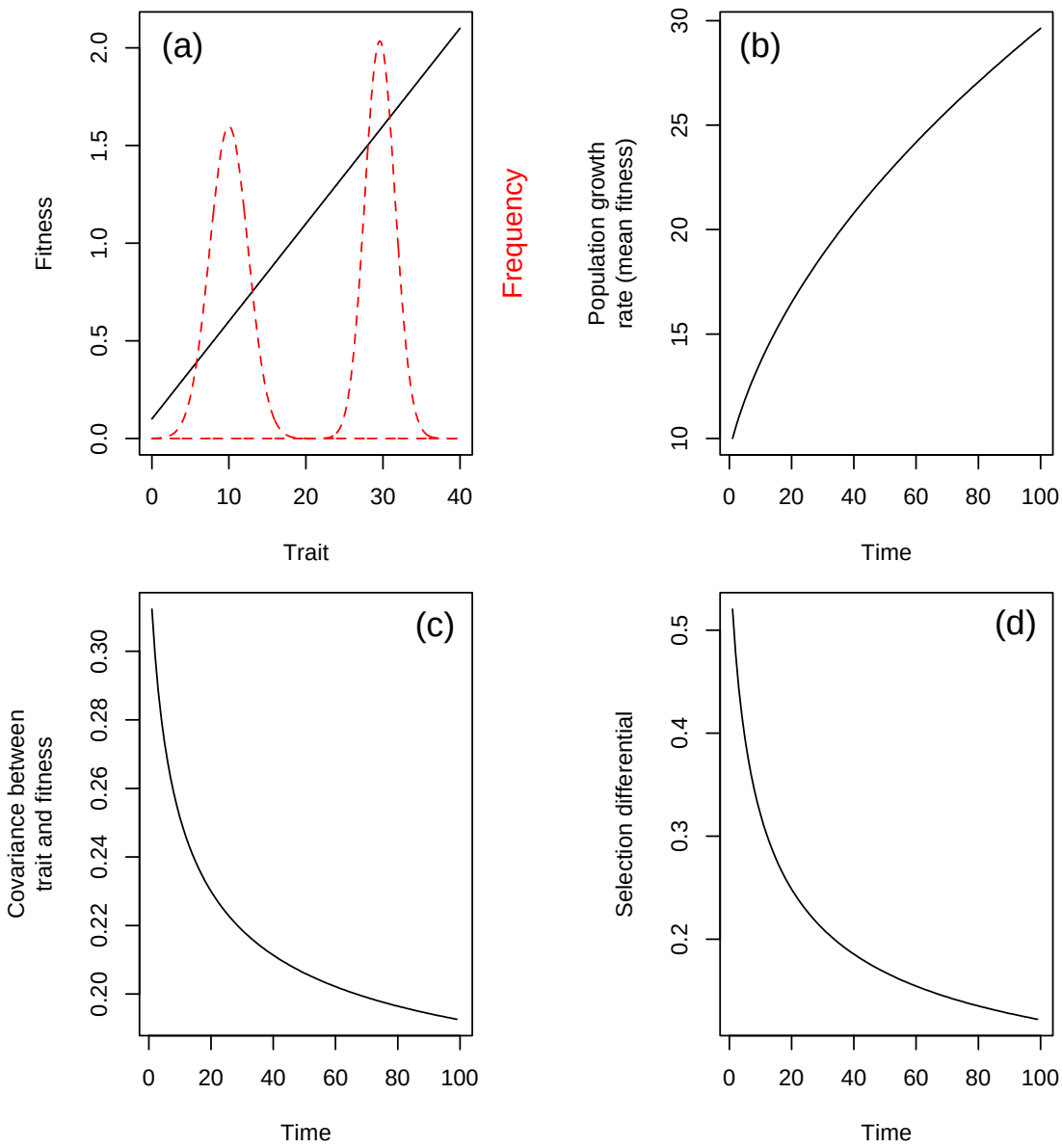

Figure 3. Results from a simple model with a (a) constant statistical association between a phenotypic trait and fitness (solid line), an initial frequency distribution of the phenotypic trait (left hand red-dashed distribution), and clonal inheritance. By the end of the simulation, the frequency distribution of the phenotypic trait has evolved location and shape (right hand red-dashed distribution). This change occurs simultaneously with evolution of (b) the population growth rate, (c) the covariance between the trait and fitness (the numerator of the selection differential), and (d) the selection differential itself.

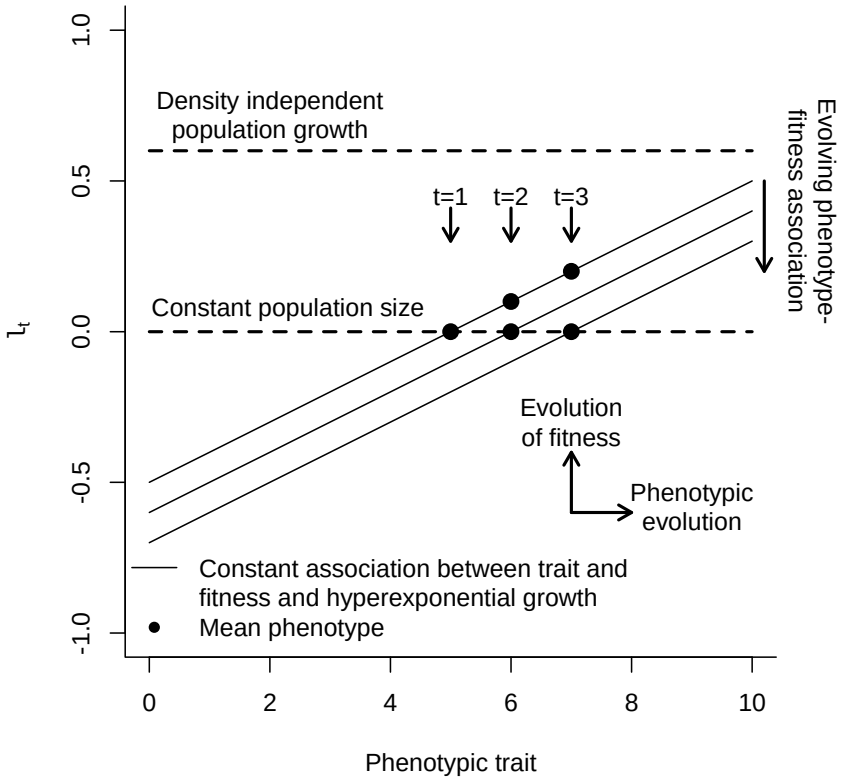

Figure 4. Associations between evolution of the phenotypic trait and the population growth rate λ_t (also mean fitness across a single time step) under different scenarios. In the absence of selection, and for a value of $\lambda_t > 0$, the population growth rate remains constant with time (upper dotted line) and population size increases exponentially. When $\lambda_t = 0$ population size remains constant with time (lower dotted line). For a constant statistical association between a trait and fitness, both the mean phenotype and λ_t increases with time and the population grows hyper-exponentially (upper solid line with points shown at times *t*=1, 2 and 3). When phenotypic evolution results in a change to the statistical association between the trait and fitness, (parallel solid lines for *t*=1, 2 and 3) mean fitness does not necessarily evolve with the mean phenotype.

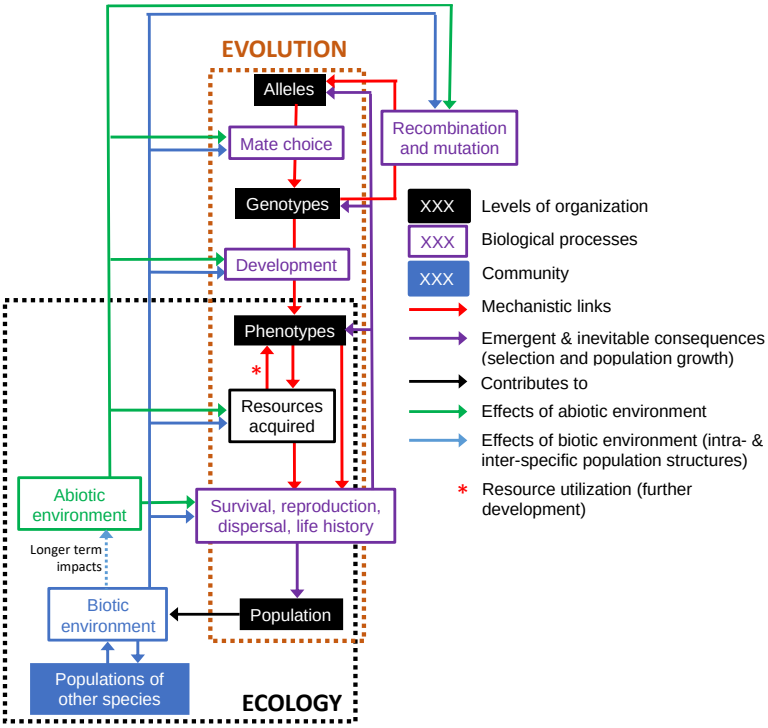

Figure 5. Complex eco-evolutionary feedbacks can arise from the dynamics of distributions of alleles, genotypes and phenotypes (levels of biological organization). These levels are mechanistically linked by key biological processes. Mate choice determines how alleles form genotypes in offspring, with development determining how phenotypes develop from genotypes. Phenotypes impact which resources individuals acquire and can influence survival, reproduction, dispersal and life history (demography). Acquired resources, such as a territory, can also influence demography directly, while also feeding back to impact further phenotypic development (including state-dependent behaviours). Selection and population dynamics are emergent consequences of which phenotypes survive and reproduce. The size and structure of the focal species and those species with which it interacts, constitute the biotic environment. The biotic environment, along with the abiotic environment, can directly influence the distribution of resources, but also biological processes in both the focal species and other species within the community. Recombination and mutation generate genetic variation and can be influenced by the environment.

Figure 6. Emma-Steve, the irascible emu.

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