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Putting evolutionary biology back in the ecological theatre: a demographic framework mapping genes to communities

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ABSTRACT

Question: How can we link genotypic, phenotypic, individual, population, and community levels of organization so as to illuminate general ecological and evolutionary processes and provide a framework for a quantitative, integrative evolutionary biology?

Framework: We introduce an evolutionary framework that maps different levels of biological diversity onto one another. We provide (1) an overview of maps linking levels of biological organization and (2) a guideline of how to analyse the complexity of relationships from genes to population growth.

Method: We specify the appropriate levels of biological organization for responses to selection, for opportunities for selection, and for selection itself. We map between them and embed these maps into an ecological setting.

Keywords: demography, dynamics, evolutionary change, response to selection, selection.

INTRODUCTION

Evolutionary biology is unusual among the sciences in that its inception occurred with the publication of an underlying unifying theory – evolution by natural selection (Darwin, 1859) – that has remained pivotal ever since. This theory has proved astonishingly successful in explaining the generation and maintenance of biological diversity across levels of biological organization from genes (Fisher, 1930), through phenotypes and strategies (Houston and McNamara, 1999) to populations (Lande *et al.*, 2003) and communities (Hubbell, 2001). Given the success of the theory in identifying processes within population and quantitative genetics, and behavioural, population, and community ecology, it is perhaps surprising how disparate

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these fields have become. One reason for this incongruence is that there is no analytical framework linking multiple levels of biological diversity (Singh and Uyenoyama, 2004).

Different levels of biological organization must logically be linked: genes play a major role in determining the phenotype (a trait measurable at the individual level), while the performance of different phenotypes – in terms of survival and reproduction – can vary with both the biotic and abiotic environment they experience (Schluter, 2000). An integrative framework of evolution should be based on a framework that links these levels.

Our aims in this paper are to (1) provide a synthetic review of how evolutionary change can be characterized as change at different levels of biological organization, (2) review how these levels of biological organization can be linked together with maps, (3) describe how each map could be parameterized from data, and (4) suggest uses of the linked maps. We do not attempt to construct a general formal theoretical model. We refer to this approach to evolutionary biology as the 'demographic framework'.

The basis for the framework was developed by Lande and the Chicago school in the early 1980s (Lande, 1982; Lande and Arnold, 1983; Arnold and Wade, 1984), but until recent advances in characterizing the genotype–phenotype map (Giot *et al.*, 2003) and the development of demographic models of community ecology it was impossible to expand Lande's framework to other levels of biological organization. Technical information on gene expression provided by systems biology (Wiley *et al.*, 2003; Thomas and Klaper, 2004) and the demography-based theories of community ecology now permit the development of an integrative framework for ecology and evolutionary biology.

THE DEMOGRAPHIC FRAMEWORK

Because evolutionary change is measured as differences in the distributions of genotypes and phenotypes either between populations or within a population over time, any framework should be population based (Lande, 1982). Changes to these population-level distributions occur as a result of the births and deaths of individuals within the population (demography), so the framework has to incorporate individuals as well as the genotypes and phenotypes that define them. Structuring the framework around demography allows it to extend to include population and community level dynamics because inter- and intra-specific interactions can influence demography where selection acts (Hubbell, 2001; Lande *et al.*, 2003).

There is an important distinction between phenotypic traits such as testes size and body size which are subject to selection, and demographic rates such as age-specific survival probability and expected fecundity which are the outcomes of the interaction of the trait and the environment. Growth is often considered by population ecologists to be a demographic rate. Size, however, is a trait and we consider growth as developmental change of that trait and not a demographic rate as such.

The demographic framework works by linking genes to traits to demography and by examining how each of these links influences ecological and evolutionary change measured as changes in the frequency distributions of alleles, phenotypes, and demographic rates. Non-adaptive changes in allele frequency can occur as the result of drift caused by demographic stochasticity (including the distribution of alleles in offspring in sexually reproducing species) and adaptive changes in allele frequency can arise as a result of natural selection (Fisher, 1930). Conceptually, we view fitness (w) as a function of the genotype

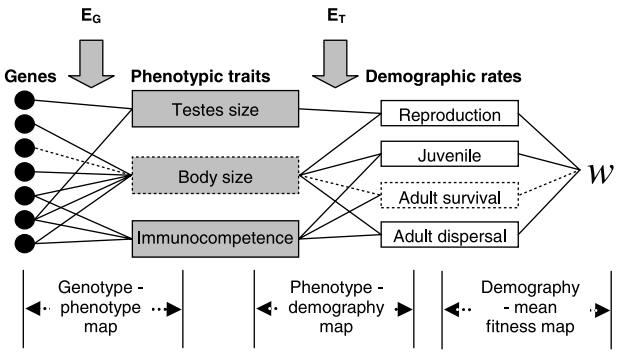

Fig. 1. A schematic of the demographic framework showing how alleles (or the proteins they code for) can interact to produce traits, which in turn influence demographic rates and w. $\mathbf{E_G}$ is the influence of the environment on gene expression and $\mathbf{E_T}$ is environmental effects on trait expression that influences demographic rates. The dotted lines represent a path between an allele, a trait (body size), a demographic rate (adult survival), and w.

distribution (**G**), influencing the phenotypic trait distributions (**T**), influencing demographic rates (**D**), i.e.

$$w = f(\mathbf{D}(\mathbf{T}(\mathbf{G}))) \tag{1}$$

The function f translates demography into population growth, so w is the population growth rate, sometimes referred to as mean fitness. From equation (1) it follows that natural selection on genes can be written as:

$$\frac{\partial w}{\partial \mathbf{G}} = \frac{\partial w}{\partial \mathbf{D}} \frac{\partial \mathbf{D}}{\partial \mathbf{T}} \frac{\partial \mathbf{T}}{\partial \mathbf{G}} \tag{2}$$

The three components of equation (1) operate at distinct levels of biological organization: responses to selection occur at the genetic level, selection occurs at the phenotypic level, and opportunities for selection arise from variation among individuals in their demographic performance. The components of equation (2) are maps (Fig. 1) that describe the associations between these levels. Each of these maps can be thought of as sensitivities (Lande, 1982; van Tienderen, 2000) and describe the change in one level resulting from a perturbation to another level, so there is a map that links genes to traits (or phenotypes), one that links traits to demography, and one that links demography to population growth rate. For the links between genes and traits and between traits and demographic rates, these sensitivities will often be estimated statistically, although these can be replaced by mechanistic links if they are known.

The environment affects the maps by affecting the relationship between genotype and phenotype and between phenotype and demography. Incorporating the environmental effects in equation (1) gives

$$w = f(\mathbf{D}(\mathbf{T}(\mathbf{G},\mathbf{E_G}),\mathbf{E_T})) \tag{3}$$

where $\mathbf{E_G}$ is the influence of the environment on gene expression and $\mathbf{E_T}$ is environmental influences on trait expression that affect demographic rates. The phenotype–demography

map can be defined as $\mathbf{D} \sim \mathbf{T} + \mathbf{E_T} + \mathbf{T} \cdot \mathbf{E_T}$ and the genotype–phenotype map as $\mathbf{T} \sim \mathbf{G} + \mathbf{E_G} + \mathbf{G} \cdot \mathbf{E_G}$. These functions can be analysed with techniques such as standard generalized linear or non-linear regression (McCullagh and Nelder, 1989). In contrast, $\frac{\partial w}{\partial \mathbf{D}}$ is the sensitivity matrix calculated from the demographic transition matrix and the population growth rate between two points in time (Caswell, 2001).

There are various points of clarification required concerning equations (2) and (3). First, their purpose is not to provide a fully developed formal model, but rather to show how equations of the derivatives describing each map could be linked. Some of these derivatives may be best formulated as continuous functions, others – such as $\partial \mathbf{T}/\partial \mathbf{G}$ – as discrete functions. The construction of the derivatives can be complex. For example, even in the absence of complicating factors like epistasis and linkage disequilibrium, models of the genotype–phenotype map are substantially more complicated than the simple derivatives often invoked by theoretical biologists (Turelli and Barton, 1994). Second, we have only specified the framework in terms of changes $\partial w/\partial \mathbf{G}$ (the rate of change of 'fitness' in response to genes) and not in terms of $\partial \mathbf{G}/\partial w$ (the rate of change of allele distributions resulting from a change in mean fitness perhaps driven by external processes influencing demography), although changes in each level of biological organization have the potential to impact on all other levels. Third, the equations above describe the map between genes, phenotypes, demography, and fitness at an instant in time. A further map – the phenotype–genotype map – that describes reproduction is required to make the equations dynamic. We do not further develop general theory associated with the derivatives here, but rather focus on parameterization of each of the maps before considering potential uses of such a framework.

THE GENOTYPE–PHENOTYPE MAP $\left(\frac{\partial \mathbf{T}}{\partial \mathbf{G}}\right)$

This component of the framework specifies the link between alleles, the proteins they code for, and phenotypic traits – the domain of systems biology including evolutionary developmental biology. Systems biology is the quantitative study of biological processes as whole systems instead of isolated parts. A product of the molecular revolution, systems biology concerns understanding intra- and inter-cellular systems from genes, through their expression to their functioning, with the concomitant emergent properties that occur through their interactions (Wiley *et al.*, 2003). The link between genotype and phenotype is complex, but substantial theoretical and some empirical progress has recently been made (Ancel, 2000; Ancel and Fontana, 2000; Galicka *et al.*, 2002; Reidys and Stadler, 2002; Barton and Turelli, 2004; Turelli and Barton, 2004). Each coding gene produces a protein, and these interact in pathways and networks to develop and maintain the phenotype. Genes (and specifically the proteins they produce) influence traits in a variety of ways. In the simplest case, alleles at one locus determine the value of only one trait. Assuming no environmental influences on the expression of the gene, all such traits are categorical, taking one of a few possible discrete values. Other traits may be under the control of multiple, interacting gene products, with each gene or gene–gene interaction contributing to a different extent (quantitative trait loci, Fig. 2). Such traits are often considered as continuous and include most morphological traits.

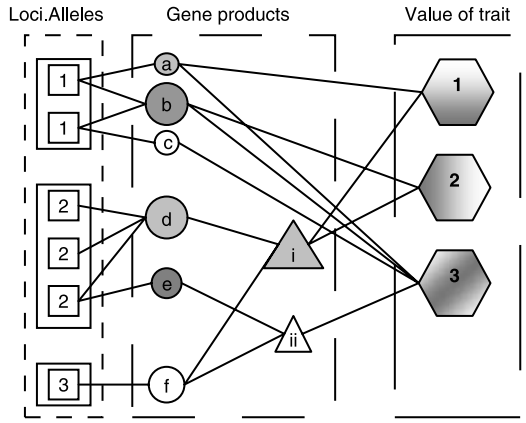

Fig. 2. A graphical representation of a genotype–phenotype map for a pleiotropic trait. Each line represents a link between an allele and a gene product (protein), between two proteins, or between a protein and a trait value. On the right-hand side of the figure are three representations of trait values. Value 1 occurs if gene products *a* and *i* interact, which in turn depends on gene products *d* and *f*. Locus 2 produces gene product *d* as long as the locus is not homozygous for allele 3. Trait value 2 occurs if proteins *b* (itself a result of locus 1 being heterozygous) and *i* are present, and trait value 3 occurs when protein *ii* combines with either protein *a*, *b*, or *c*. The function relating the proteins to the trait values can be obtained by measuring the trait value and the presence/absence or concentrations of gene products *a*, *b*, *c*, *d*, *e*, *f*, *i*, and *ii*. Because there may be a very large number of ways gene products (and the alleles that produce them) can be related in a modelling framework, automated methods for distinguishing between the goodness-of-fit of competing models could be used: trans-dimensional simulated annealing (Brooks *et al.*, 2003) or reversible jump Markov chain Monte Carlo (Green, 1995) are two methodologies with considerable potential. An even better approach would be to identify the mechanisms determining gene products and trait values.

The genetics of many traits is substantially more complex and may involve pleiotropy, epistasis, multi-gene interactions, and multiple gene products and steps (Arthur, 2002). To incorporate them into the demographic framework requires the developmental pathways leading to the trait to be broken down into separate stages and each stage statistically or mechanistically modelled (Thomas and Klaper, 2004) (Fig. 2).

If the genetic architecture of a trait is known and the genotype and phenotype of individuals within a population recorded, it is straightforward to statistically infer the link between genotype and phenotype (Goodnight, 1995) using generalized linear or non-linear regression modelling with an error structure chosen to best suit the data (McCullagh and Nelder, 1989). The response variable is each individual's trait value and the genotype can be modelled by nesting alleles within loci, with both alleles and loci treated as categorical variables. Over-dominance at a locus can be identified if there is a significant interaction between alleles at a locus, and gene–gene interactions can be identified if there is a significant interaction between loci (Fig. 2).

The framework can also incorporate mutations. The introduction of a mutation could require the re-calculation of the genotype–phenotype maps (and possibly other maps within the framework), especially if the mutation has dramatic effects on the phenotype, perhaps by introducing an entirely new trait (Sole *et al.*, 2002). If a mutation has a large effect on the function of a gene, then the genotype–phenotype map will require re-parameterizing –

in this case, that the partial derivatives we have based our maps on may make little biological sense.

The influence of the environment on the genotype–phenotype map within the demographic framework can be estimated if multiple time points are considered. Environmental effects include phenotypic plasticity – when trait values vary with environment within a genotype (Stearns, 1992) – and genotype-by-environment interaction – when the effect of environment differs between genotypes (Pooni and Jinks, 1980). The lack of any statistical association between genotype and trait may represent no link, or may reflect genetic canalization. Similarly, the lack of any significant genotype-by-environment effect may suggest no link or environmental (developmental) canalization. Simple regression approaches cannot distinguish between the lack of a link and canalization – this knowledge can only come from a detailed mechanistic understanding of the genetic pathways or networks. Although systems biology including evolutionary developmental biology has made enormous advances recently in understanding the development of traits (Leroi, 2004) through disabling gene function, for most complex traits a mechanistic understanding is currently unavailable and therefore the correlative approach is most appropriate.

THE PHENOTYPE–DEMOGRAPHY MAP $\left(\frac{\partial \mathbf{D}}{\partial \mathbf{T}}\right)$

The aim of the phenotype–demography map is to identify the association between the value of a phenotypic trait and the probability of an individual expressing that trait value surviving, reproducing, or dispersing (Kingsolver *et al.*, 2001). The map describes the changes in the values of demographic rates resulting from changes in the values of the traits. Each link of the map can be thought of as a selection gradient (Arnold and Wade, 1984) because fitness is a function of the demographic rates and the traits that influence them (see equations 1–3).

Phenotype-by-environment interactions ($\mathbf{T} \cdot \mathbf{E_T}$) that influence demographic rates are commonplace (Tuljapurkar and Caswell, 1997). Environmental variables can be biotic or abiotic and may include climate variation, resource availability, fluctuations in the density of prey, predator or competitor species, or prevalence of a disease (see section below entitled 'The demographic framework and ecological dynamics'). The ecological or environmental mechanisms generating changes in the strength of a selection gradient are usually unknown but are often characterized by changes in density-dependent or -independent processes (e.g. climate) (Fowler, 1981; Fowler and Genoways, 1987). The environment that influences a trait need not be the current environment (Beckerman *et al.*, 2002). For example, the neonatal environment affects many adult traits (Lindstrom, 1999).

Selection operates through multiple demographic rates simultaneously (equation 3) and the strength of selection through each demographic rate may vary with time as a function of the environment (Coulson *et al.*, 2003). If selection is to be estimated accurately, the associations between a trait and *all* demographic rates constituting population growth have to be considered simultaneously. Such an approach, however, raises a new issue. How much weight should be given to each demographic rate?

THE DEMOGRAPHY–MEAN FITNESS MAP $\left(\frac{\partial w}{\partial \mathbf{D}}\right)$

The maps described above allow genotype to be linked to phenotype and phenotype to be linked to demographic rates. For selection to occur there needs to be variation between individuals, which generates opportunity for selection defined as variation in demographic rates between individuals expressing different phenotypic trait values (Caswell, 2001). The objective of the demography–mean fitness map is to describe the way that trait variation contributes to variation in population growth rate via demography and hence provides opportunity for selection.

In evolutionary ecology, population growth rate is often equated with mean fitness. Many evolutionary biologists estimate mean fitness from the asymptotic properties of the demographic transition matrix, **D** (Leslie, 1945; Caswell, 2001). This equivalence formally only occurs when the population is at demographic equilibrium (Caswell, 2001). Because populations are rarely at demographic equilibrium, we estimate population growth rate as the proportional change in population size between two points in time. To avoid possible confusion we no longer use the term 'mean fitness' but instead use w_t – the population growth rate between time t and some appropriately defined time in the future (N_{t+i}/N_t).

The weights of the demographic rates are the sensitivities (Caswell, 2001) [or elasticities (Horvitz *et al.*, 1997)] of w_t. To estimate these sensitivities of w_t, a demographic model of w_t is required. We construct the demographic framework around a demographic projection model, for example a Leslie matrix (Caswell, 2001). The model is parameterized using the state-specific survival and recruitment rates of individuals within the population between the two time points under consideration. Sensitivity of w_t to a demographic rate is estimated numerically by perturbing the model element incorporating that demographic rate and multiplying the resulting matrix by the observed population structure.

INTEGRATING THE THREE MAPS

In the sections above, we have discussed how the genotype maps to the phenotype, how the phenotype maps to demography, and how demography links to population dynamics. We have also argued that these three maps can be integrated together and briefly suggested statistical approaches to examining associations. In this section, we demonstrate one way in which the maps can be linked, and show how the maps are best considered describing the association between genes, phenotypes, demography, and w_t at a point in time. We then explain how environmental variation can generate changes in the maps over time. We also show how reproduction is incorporated into the framework. In the example provided here we only consider discrete traits, although we accept that often the more interesting traits are continuous. Future work will demonstrate how to incorporate continuous traits.

The approach we use is matrix-based. We start by describing the population using a vector, $\mathbf{P_t}$, which describes the genotypic and phenotypic structure of the population in year t. As a simple case, we consider a population consisting of two alleles, A and B, two phenotypes, red and green, and three age classes, 1, 2, and >2. In theory the population vector could be written as a list with each class containing one individual – although the resulting transition matrix would be difficult to parameterize. The vector $\mathbf{P_t}$ consists of integers describing the number of individuals within each class – for example, we could nest genotypes within phenotypes within age classes and write the vector $\mathbf{P_t}$ = (1.red.AA,

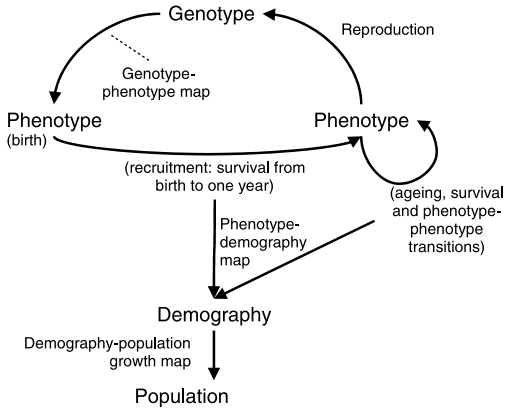

Fig. 3. Graphical representation of how to link maps within the demographic framework.

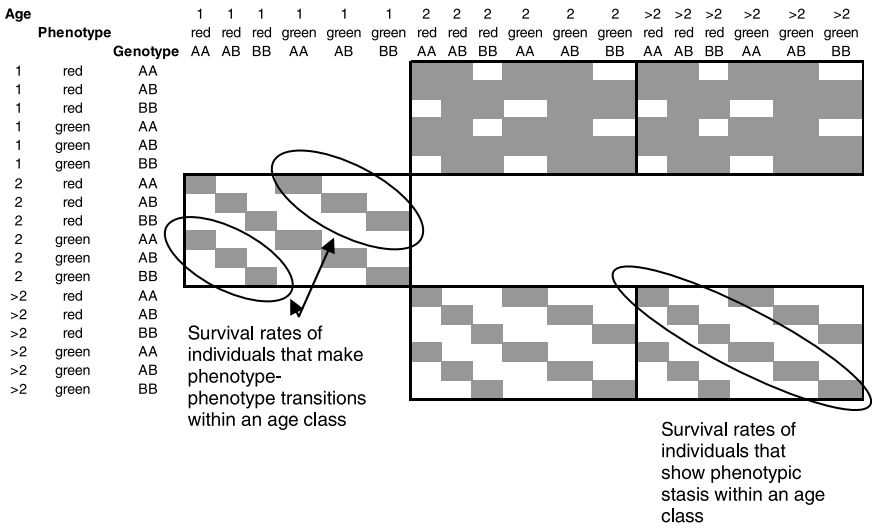

Fig. 4. A caricature of a transition matrix that could be used to link maps together. The matrix elements towards the top of the matrix describe age-specific recruitment. These cells are a combination of the reproduction, the genotype–phenotype map, and aspects of the phenotype–demography map. Note that unless there is perfect assortative mating, the maps cannot be written as a linear transition matrix – even if all other processes are linear. The recruitment functions become frequency dependent for cases other than perfect assortative mating (see text).

1.green.AA, 1.red.AB, 1.green.AB, 1.red.BB, 1.green.BB, 2.red.AA, . . . , >2.green.BB)^{T}, where T represents the matrix transpose. Note that multiple elements in this vector could be zero – it might not be possible for an AA genotype to develop into a green phenotype, for example. This vector is then mapped to a vector $\mathbf{P}_{t+1}$ by a partitioned matrix, $\mathbf{A}_t$. Different parts of this matrix represent different transitions (Fig. 3). We now discuss each in turn.

The cells in the top rows of the matrix (Fig. 4) describe recruitment to the population. They include reproduction, the genotype–phenotype map, and part of the phenotype–demography map (Fig. 3). The calculation of these matrix elements requires various steps.

First, reproduction between individuals within the population generates genotypes. Individuals generally mate randomly or choose mates as a function of their phenotypes. Consequently, the probability of a specific genotype being generated may be a frequency-dependent function of genotypes with phenotypes. We consider reproduction as the update from one time step to the next within our framework, but it could be considered as a further map – the phenotype–genotype map. The probability of an individual reproducing in a specific genotype–phenotype–age class can vary with time as a function of the ecological environment as well as with the frequencies of other classes within the population.

Unless there is perfect assortative mating, the transition matrix in Fig. 4 cannot be written as a linear matrix transition – even if all the other processes are linear – and at least one of the sensitivities is non-constant. A general formulation of genotype production assuming no age dependence in reproduction is:

$$0.AA = f_{rr}^{AA}(red.AA, red.AB, N) + f_{rg}^{AA}(red.AA, green.AA, red.AB, green.AB, N) + f_{gg}^{AA}(green.AA, green.AB, N) \tag{4}$$

where $red.AA$ is the total number of adults with that genotype–phenotype combination, N is the total number of adults (included to allow for frequency dependence), and the f's incorporate both the frequency of a particular phenotypic pairing as well as the fertility of that pairing (the superscript is the genetic outcome; the subscript is the phenotypes of the two parents). With random mating, for example,

$$f_{rg}^{AA}(\bullet) = \frac{\phi_{rg}}{2N}\begin{bmatrix} (red.AA \times green.AA) \\ +\frac{1}{2}(red.AA \times green.AB + red.AB \times green.AA) \\ +\frac{1}{4}(red.AB \times green.AB) \end{bmatrix} \tag{5}$$

where ϕ_{rg} is the fertility of a red–green pair. In contrast, with perfect assortative mating by phenotype, $f_{rg}^{AA} = 0$.

Once genotypes have been generated they develop into phenotypes. In the discrete case, this can be achieved by using transition matrices. For example, assume reproduction has generated a vector of the number of genotypes within the population $(AA, AB, BB)^T$ which maps to red and green phenotypes. The following matrix can achieve this map and can generate a list of phenotypes nested with genotypes.

$$\begin{bmatrix} red.AA \\ red.AB \\ red.BB \\ green.AA \\ green.AB \\ green.BB \end{bmatrix} = \begin{bmatrix} q_{AA}(red) & 0 & 0 \\ 0 & q_{AB}(red) & 0 \\ 0 & 0 & q_{BB}(red) \\ q_{AA}(green) & 0 & 0 \\ 0 & q_{AB}(green) & 0 \\ 0 & 0 & q_{BB}(green) \end{bmatrix} \bullet \begin{bmatrix} AA \\ AB \\ BB \end{bmatrix} \tag{6}$$

where q represents the transition probabilities between a genotype and a phenotype. In the simplest case, where one genotype always determines a specific phenotype, three of the probability values in the matrix in equation (4) will be unity. The vector generated by equation (6) is then subjected to the phenotype–demography map. In this map, individuals

can either survive or die and may change phenotype (individuals can shrink or grow, although we accept it is unlikely that they really change colour). They cannot, however, change genotype. The following matrix describes this aspect of the phenotype–demography map:

$$\begin{bmatrix} 1.red.AA \\ 1.red.AB \\ 1.red.BB \\ 1.green.AA \\ 1.green.AB \\ 1.green.BB \end{bmatrix} = \begin{bmatrix} p_r g_{rr} & 0 & 0 & p_g g_{rg} & 0 & 0 \\ 0 & p_r g_{rr} & 0 & 0 & p_g g_{rg} & 0 \\ 0 & 0 & p_r g_{rr} & 0 & 0 & p_g g_{rg} \\ p_r g_{gr} & 0 & 0 & p_g g_{gg} & 0 & 0 \\ 0 & p_r g_{gr} & 0 & 0 & p_g g_{gg} & 0 \\ 0 & 0 & p_r g_{gr} & 0 & 0 & p_g g_{gg} \end{bmatrix} \bullet \begin{bmatrix} 0.red.AA \\ 0.red.AB \\ 0.red.BB \\ 0.green.AA \\ 0.green.AB \\ 0.green.BB \end{bmatrix} \quad (7)$$

The indices on the g's follow the same convention as for matrix elements, so that g_{rg} represents the probability that a green newborn becomes a red adult, conditioned on survival, p (so $g_{rg} + g_{gg} = 1$). We also assume that survival depends only on the starting phenotype, which is consistent with standard size-structured models.

The matrix elements associated with survival are easier to describe, and these constitute the remainder of the phenotype–demography map. Each of these cells describes the transition probability between two age–phenotype–genotype classes. As above, these transition probabilities incorporate phenotype–phenotype transitions as well as survival.

Environmental variation – in either the developmental or ecological environment – can be incorporated by making each matrix element in reproduction the genotype–phenotype map and the phenotype–demography map a function that is environment-dependent. These functions may be frequency-dependent, density-dependent, dependent on the number of inter-specific competitors, predators or prey, or density-independent.

Each transition matrix $\mathbf{A_t}$ can be subjected to sensitivity or elasticity analyses using perturbation analyses. Two sensitivities of interest include the consequences on the population growth rate of a change in a matrix element $\left(\frac{\partial w_t}{\partial a_{ij}}\right)$, and the change in the structure of the population as a function of a matrix element $\left(\frac{\partial \mathbf{u}}{\partial a_{ij}}\right)$. The sensitivities associated with the genotype–phenotype map, the phenotype–demography map, and the demography–w_t map can be calculated by perturbing matrix elements associated with each map, and examining how the distribution of genotypes, phenotypes, or population growth rate is altered.

One immediate observation from the approach we suggest is that the number of functions and matrix elements that need to be estimated increases very rapidly with added complexity. In our simple example of three genotypes, two phenotypes, and three age classes, the transition matrix can have as many as 92 non-zero elements, with many of these non-zero estimates being generated using multiple steps. The robust parameterization of such a model would consequently require many data. However, the approach could prove a useful theoretical starting point to identify those transitions that are likely to have important sensitivities given a specific question of interest. For example, under what conditions can phenotype–phenotype transitions maintain genetic diversity within populations?

A second observation is that within the framework we are proposing, maps explain the mean transition rates for groups of individuals. The vector $\mathbf{P_t}$ could be expanded so that

each individual is in a class of its own – this way the maps become individual maps. The parameterization of such a framework will then become statistically difficult.

THE DEMOGRAPHIC FRAMEWORK AND ECOLOGICAL DYNAMICS

The framework above specifies $\frac{\partial w}{\partial \mathbf{G}}$ over a single time interval allowing us to estimate how a change at one level of biological organization generates change at other levels. But what makes evolutionary biology particularly interesting is that the ecological theatre is not static. To incorporate ecological dynamics we need to implement the framework across multiple time steps. To use the framework to simulate evolution in a varying environment, a dynamic population model is constructed and used to generate a transition model for each point in time. The model consists of a set of equations that generate environmentally induced variation in state-specific survival and recruitment rates (Coulson *et al.*, 2001). Environmental variation can be dealt with by mechanistically modelling inter- and intra-specific competition for resources (Hubbell, 2001; Chave, 2004), or by using descriptors of the mechanisms like density-dependence and environmental stochasticity (Lande *et al.*, 2003). The simulation can generate distributions of allele frequencies at multiple loci, distributions of traits and a description of the population structure at each time step.

The individual traits and the demographic rates they affect ultimately determine w_t (Lande, 1982), but this does not mean they are independent of the environment in which the population is embedded (equation 3) (Merila *et al.*, 2001). Thus the environment is not just external 'noise', because as much as the organism is affected by the world around it, the reverse is equally true. Any evolutionary change to an organism potentially generates changes in its biotic and abiotic environment that have further evolutionary consequences – for example, arms races between predators and prey, and ecosystem engineering. Due to the Red Queen nature of evolutionary change, any integrative framework of evolutionary biology should incorporate intra- and inter-specific processes that impact on $\mathbf{E_T}$ (Wade, 2003).

Just as the genotype–phenotype map can be diffuse because the genome is a complex and interactive system (Giot *et al.*, 2003), so too is the relationship between demography and ecological dynamics (Hubbell, 2001). $\mathbf{E_T}$ includes intra-specific processes like density- and frequency-dependence and inter-specific processes like competition. Therefore, some of the $\mathbf{T} \cdot \mathbf{E_T}$ components of the sensitivities $\frac{\partial \mathbf{D}}{\partial \mathbf{T}}$ can be interpreted as selection on traits that influence the inter-specific interactions. An example is selection on the trait body size due to a change in predation risk, such as occurs when *Daphnia* are exposed to *Chaoborus*. The environmental component of the genotype–phenotype map, $\mathbf{E_G}$, can also include ecological effects in addition to the intra-individual developmental environment. An example is defences induced during development (Tollrian and Harvell, 1998). The decomposition of $\mathbf{E_G}$ into its components, however, is substantially more complex than the decomposition of $\mathbf{E_T}$.

Apart from the more subtle co-evolutionary consideration, the loss or introduction of competitors, predators, or diseases may, or may not, have effects at several levels of the demographic framework. Interacting species can alter demographic rates and the distribution of w_t without corresponding changes in the distribution of phenotypic trait values if the phenotypic trait of interest is not strongly correlated with the demographic traits that change.

In summary, changes to the environment can lead to responses to selection, which in turn feed back to alter the ecological dynamics (Dieckmann *et al.*, 2000).

PARAMETERIZING THE FRAMEWORK

The maps described above provide a framework that may provide insight into evolutionary and ecological change. It may also provide a useful platform from which to direct the development of theory.

The demographic framework requires an understanding of multiple maps. Although data may not exist to parameterize the framework for multiple phenotypic traits in any system, it is possible they will within the near future (Thomas and Klaper, 2004). Nevertheless, different systems currently allow the parameterization of individual maps. For example, detailed long-term individual-based studies of large ungulates provide good examples of the phenotype–demography–w_t maps (Coulson *et al.*, 2001), recent gene chip technology has provided examples for the genotype–phenotype map in *Drosophila* (Giot *et al.*, 2003), and protein networks in yeast provide examples of the formation of more complex traits (Han *et al.*, 2004). Indeed, some systems are tantalizingly close to having characterized diversity at the genetic, phenotypic, individual, and population levels, including *Tribolium*, *C. elegans*, *Drosophila*, *Arabidopsis*, rotifers, and large ungulates. One issue that we do not consider here is how error propagates through the system. Given that the maps potentially contain so many different links, it could prove impossible to gain sufficient satisfactory estimates of parameters and variance–covariance matrices to confidently parameterize the framework for any system.

Once the framework is parameterized for an organism, it could be used to infer the relative processes that contribute to any observed evolution. Perhaps more importantly it may allow us to make evolutionary predictions that are especially valuable in the face of climate change, habitat alteration, predicting invasion success of exotic species, and so on.

SHORT-CUTS AND THE DEMOGRAPHIC FRAMEWORK

A potential powerful use of the framework is as a test of the performance of the approximations on which evolutionary biology is necessarily so dependent. Indeed, the complexity of the simple example we give shows that realistic parameterization of the maps is probably not possible However, the question still remains, which approximations should be used in which circumstances? An approach to test the performance of approximations will provide insight on when we can overlook certain processes – putting them into a black box – and when we cannot do this and require information on a specific mechanism to gain useful insight into a specific problem. An objective of any theoretical modelling approach is to avoid complexity when absolutely possible; however, knowing which complexity can be ignored and which cannot when addressing a specific problem is non-trivial. The approach of simulating a complex phenomenon to generate data to examine when simple models adequately capture dynamics has proven popular in some fields of research (Peck, 2004), although many theoreticians find the approach unpalatable due to its relative inelegance. Evolutionary biology is complex and we require simple models that perform well. Below we suggest a way that the demographic framework could be used to identify what these models may be.

Previous attempts to produce a quantitative theory that links genes to fitness have deliberately avoided the complexity of the demographic framework by making restrictive or arbitrary assumptions to allow short-cutting of the details that link the maps together. This is sometimes justified as a means of avoiding the trap of reductionism, yet it is difficult because it is often hard to know *a priori* which assumptions will substantially impact on results. Selection, for example, is often assumed to operate through only one demographic rate (Kingsolver *et al.*, 2001); the performance criteria used as proxies for fitness in evolutionary arguments such as rate maximization or reproductive success have unknown relationships to w (Benton and Grant, 2000; Brommer, 2000; Shertzer and Ellner, 2002); restrictive assumptions such as constant density-independent environments are often made to allow analytical tractability (Lande, 1982); and processes at levels other than the focal level are assumed to be unimportant (e.g. adaptive dynamics ignores genetic architecture that could constrain evolutionary change). Some of these short-cuts are undoubtedly useful approximations; however, currently we have no means of assessing the constraints on their utility. By explicitly incorporating the range of biological complexity into this framework, we can provide an experimental arena in which to assess the performance of short-cuts.

Many models in biology are not based on maps *per se*: they take short-cuts and often the validity of the short-cuts is unknown. Many of these short-cuts try to estimate the properties of one of the maps using details from another map. For example, empirical quantitative genetics makes inference about genetic variance by analysing the distribution of trait values (Falconer, 1960), and some population ecology attempts to infer demography by analysing time-series of w_t (Turchin *et al.*, 2000). It is possible that the quantitative or population genetic frameworks cannot empirically and theoretically be bettered; however, until we attempt to incorporate the phenotype into the population genetic framework, or explicitly consider genes in the quantitative genetic framework, it is hard to know the consequences of assumptions inherent in each on our general understanding of ecological and evolutionary change.

Another often used short-cut in adaptive dynamics is to use the expectation of the geometric mean of w_t as time tends to infinity (the dominant Lyapunov exponent) to infer evolutionarily stable strategies of alleles and traits (Dieckmann *et al.*, 2000). These adaptive dynamics methods typically ignore density-dependence in the mutant allele/trait. The likelihood of a mutant invading is measured by estimating the mutant's growth rate into a population of residents when the mutant is rare. Frequency dependence is assumed when allele A has a positive growth rate (when rare) into a population of allele B, and allele B has a positive growth rate (when rare) into a population of allele A. As the demographic framework would generate data on the actual distributions of these alleles under a stochastic equilibrium, this ability of the adaptive dynamic framework to identify evolutionarily stable strategies (ESSs) can be tested.

Behavioural ecologists typically arbitrarily identify 'currencies' – which relate short-term consequences of behaviour to presumed fitness – when analysing hypotheses about adaptive behaviour formally (Stephens and Krebs, 1986). The validity of these currencies is ultimately assessed by the success of specific models when challenged with data. However, since the demographic framework enables individual performance in different life-history or behavioural contexts to be mapped to the relative performance of alleles within populations, it offers the opportunity to deduce such currencies from long-term data sets for use in conventional optimization and game theory.

Another example of a frequently used, yet often untested model comes from theoretical population genetics, which assumes that a direct link can be made between an allele and environmental variation determining birth and death and that it is not necessary to consider the trait(s) that the allele may influence, or the individuals that the traits are nested in. Because traits are often overlooked in population genetics, there has been a tendency to overlook the importance of pleiotropy in the standard population genetic model until recently (Otto, 2004), even though its importance has long been assumed (Wright, 1968). The demographic framework could provide a test of the conditions under which the assumptions of the standard population genetic model provide realistic approximations.

Finally, population ecologists often take the pattern of population fluctuations and estimate the importance of endogenous and exogenous factors ignoring the demography (Royama, 1992; Berryman and Chen, 1999; Turchin, 2003). Although these models can capture the essential features of the dynamics (Grenfell *et al.*, 1998; Turchin, 2003), the range of ecological conditions under which they perform adequately is not known and without a proper understanding of the demography and the properties of the environment an irreducible uncertainty about mechanisms remains. Nonetheless, claims have been made that this modelling approach can be used to identify general laws for ecology (Berryman, 2003). A more illuminating question is when can the demography be ignored? Once again, the demographic framework developed here would allow a theoretical examination of the conditions under which demography can be ignored with little loss of biological interpretation.

Although the processes we discuss are responsible for all evolutionary change, this will not always be the most useful framework for large-scale temporal and spatial processes such as macro-evolution and the global distribution of biomes. These large-scale patterns will need to continue to be addressed using approximations and short-cuts such as those discussed above.

CONCLUSIONS

The framework we outline here will be familiar to most biologists. The proposition of deconstructing evolutionary and ecological change into simple maps is not new: figures similar to Fig. 1 have been proposed by Lewontin (1974) and Ricklefs and Wikelski (2002) to cite two examples. What is perhaps surprising is how little we know empirically about the distribution of sensitivities within and between maps. There are descriptions of mean $\partial w/\partial \mathbf{D}$ maps for asymptotic dynamics for some systems (Caswell, 2001), but there are few examples of temporal variation in such maps (Coulson *et al.*, 2004), very few descriptions of phenotype–demography maps (Coulson *et al.*, 2003), and it is only recently that examples of genotype–phenotypes maps have been produced (Han *et al.*, 2004). However, characterization of the maps we identify provides one way to identify ecological and evolutionary generality.

In recent years, there have been remarkable advances in: systems biology, including genomics, proteomics, metabolomics; evolutionary development biology and genetics; and population biology. These advances have had the effect of isolating biologists within their own domain of expertise such that none can incorporate the full spectrum of evolutionary change from genes to ecology. The framework we develop takes these substantial advances and attempts to integrate them to transform Darwin's conceptual vision into a quantitative theory. Specifically, we identify levels at which evolutionary processes operate from genes to ecology and demonstrate how to link them quantitatively.

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