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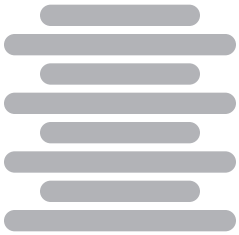

13 | MODELLING THE ECOLOGICAL CONTEXT OF EVOLUTIONARY CHANGE: DÉJÀ VU OR SOMETHING NEW?

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13.1 | INTRODUCTION

A principle interest in evolutionary ecology is to understand how ecological interactions within and between species generate natural selection and, in turn, how such natural selection shapes these ecological interactions through evolutionary change (MacArthur 1972; Pianka 1974; Roughgarden 1979; Cockburn 1991; Bulmer 1994; Real 1994; Fox, Roff, & Fairbairn 2001). This feedback between ecological and evolutionary processes lies at the heart of this area of research. Creating a theory that adequately represents this mutual dependence, and that makes testable predictions about ecological and evolutionary processes, has presented a considerable challenge. It is difficult enough to obtain a faithful theoretical description of either ecological or evolutionary processes, let alone a coherent melding of the two. The development of such a theory is desirable because presumably the resultant bridge between ecology and evolutionary biology will inject new ideas into both fields and lead to an important consolidation and extension of our understanding of the earth's biota.

Given the complexity of both ecological and evolutionary processes, it is necessary to make several simplifying assumptions in the development of theory. In this chapter I will describe various theoretical devel-

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opments in this area, organizing and presenting them in a fashion that is chronological and is meant to illustrate the connections, similarities, and differences among them. My treatment is by no means an exhaustive review, and the perspective taken is necessarily biased towards areas in which I have some knowledge (and thus is biased towards my interests). Although I was alive throughout most of these developments, the earliest of them happened when I was too young to be fully cognizant (or even interested) in such arcane topics; therefore, much of my historical perspective has been gleaned from the literature and talking with more senior scientists.

I begin by briefly presenting some background on theoretical ecology and theoretical evolutionary biology as independent fields of study, and I introduce some examples that will illustrate various approaches later in this chapter. In the bulk of this chapter, I consider the various ways in which researchers have sought to merge these two areas. I conclude by asking where we stand, by asking where we go next, and by considering whether there have been quantum leaps or paradigm shifts along the way.

13.2 | THEORETICAL ECOLOGY

There are many processes and types of interactions between organisms that have been the focus of ecological theory. A few of the most important include competition for resources, predation, parasitism, mutualism, and facilitation (Begon, Harper, & Townsend 1986). Although each process is distinct from the others, the ecological theories developed for each of them share at least one common feature: They have been directed towards describing and explaining the distribution and abundance of different kinds of organisms (typically species) as a result of these processes. Individuals within a given species (or sometimes within an age, condition, or size class of that species) are treated as being effectively identical. No allowance is made for genetic variation among individuals in traits that affect these ecological interactions, thereby precluding any evolutionary change.

At first this neglect of evolutionary potential might appear surprising. When theoretical ecology was born, it was well appreciated that populations can and have evolved. The reason for this omission was a feeling (by some, at least) that evolutionary change proceeds on a timescale much longer than that of ecological change. Therefore, the inclusion of evolutionary change is not critical for understanding the implications of various ecological interactions. After all, the point of theory is to simplify reality in a way that captures only those features important for the ques-

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tion at hand. It is now well recognized that this separation of timescales is artificial and that rapid evolution can (and often does) occur (see Hendry & Kinnison 1999 for a review). This recognition formed an important part of the motivation for the development of theoretical evolutionary ecology. (Ironically, the most recent theoretical developments in evolutionary ecology have returned to the assumption of a separation of timescales, but I will explain this later.)

To better illustrate these ideas, I will consider one of the simplest examples in theoretical ecology: a discrete-time version of logistic growth (Case 2000). This model is meant to capture the population dynamics of a single species under density dependence. There are many formulations of this model, and one possibility that has been used extensively in the literature is to suppose that the number of individuals in the next generation is given by the number in the current generation plus the number of new individuals produced. Suppose that each individual in generation t gives rise to $r(1 - N(t)/K)$ new individuals, where r and K are constant parameters representing the number of individuals produced in the absence of competition and the population carrying capacity, respectively, and $N(t)$ is the population size in generation t. Then you have the following:

$$N(t+1) = N(t) + N(t)r\left(1 - \frac{N(t)}{K}\right). \tag{13.1}$$

The effects of within-species competition are represented by the fact that an individual's reproductive output, $r(1 - N(t)/K)$, declines (linearly) as population size increases, and it reaches zero when the population attains carrying capacity (i.e., $N = K$). Equation 13.1 can be rewritten as follows:

$$N(t+1) = \left\{1 + r - r\frac{N(t)}{K}\right\}N(t). \tag{13.2}$$

The quantity in the braces of Equation 13.2 is the *total* contribution of an individual (i.e., per capita) to the next generation (i.e., it is the total per capita number of individuals produced in generation t, including an individual's own survival—which happens with probability 1 in this model). The absence of the possibility for evolutionary change is reflected by the fact that this per capita production is identical for all N individuals of the population.

The analogous model for both intra- and interspecific competition has also received a large amount of attention in the ecological literature.

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I present this here because it features prominently in the merger of ecological and evolutionary theory:

$$N_1(t+1) = N_1(t) + r_1\left(1 - \frac{N_1(t) + \alpha_{12}N_2(t)}{K_1}\right) \tag{13.3A}$$

$$N_2(t+1) = N_2(t) + r_2\left(1 - \frac{N_2(t) + \alpha_{21}N_1(t)}{K_2}\right). \tag{13.3B}$$

Here, α_{ij} (the competition coefficients) represents the competitive effect of an individual of species *j* on an individual of species *i*, relative to a conspecific individual *i*. The subscript numbers refer to species 1 and 2. Equations 13.3A and B are often referred to as the Lotka–Volterra competition equations (Case 2000), and like the logistic-growth model in Equation 13.1, this model is phenomenological because it does not treat the dynamics of resource consumption and the competition that results in mechanistic manner. Rather, this is described qualitatively because higher densities of individuals (of either species) reduce the per capita production of individuals of either species. The strength of these effects is controlled by the parameters α_{ij}. More realistically, I could construct a mechanistic model of competition for resources by modelling the resource dynamics, leading to a so-called consumer–resource model (MacArthur 1972). Interestingly, it has been shown (MacArthur 1970, 1972) that if the dynamics of the resource turnover are fast relative to those of the consumer, then a system analogous to Equations 13.3A and B can be obtained from consumer–resource models.

As with the logistic model, the assumption of a separation of timescales between ecological and evolutionary processes in Equations 13.3A and B is reflected by the lack of within-species variation in the traits that affect competition for resources. Thus, the parameters governing the interactions between the two species (e.g., *r*, *K*, and α_{ij}) remain constant during the ecological dynamics. There is an enormous number of extensions and further developments of this sort of model, but all are dynamic systems (often in continuous time) in which there are several state variables describing the density of different organisms and in which all parameters governing the interactions are treated as constants. In other words, the parameters do not change during the dynamics. This theory is typically used to understand and predict population dynamics over time (or space or both). For example, do you expect stable equilibrium population sizes, cycling, or other more complex nonequilibrium behaviour? Moreover, how do the various parameters affect the outcome? Issues surrounding this last question are

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of interest to evolutionary ecologists because the community dynamics themselves will generate natural selection on these parameters, causing them to evolve and thereby altering these dynamics. Thus, you need to include evolutionary change to understand how ecological interactions shape their own evolutionary trajectories.

13.3 | THEORETICAL EVOLUTIONARY BIOLOGY

As with theoretical ecology, the field of theoretical evolutionary biology is now enormous. Here I restrict attention to two relatively self-contained and influential areas: classical population genetics (which has developed into its own subdiscipline) and optimization–game theory. To my knowledge there is not yet a comprehensive treatment of the history and development of game theory in evolutionary biology, but interested readers should consult the book by William Provine (2001) for a wonderful historical account of the development of population genetics.

13.3.1 | Classical Population Genetics

Most theory in population genetics (and virtually all such theory in the classical population-genetic literature) treats population densities as being either constant or irrelevant (Hartl & Clark 1989). In addition, although ecological interactions will often be important causes of natural selection through their effects on the fitness of different individuals, most population-genetic theory ignores the particular causes of natural selection and instead treats it in a phenomenological fashion. The most frequent approach is to suppose that different alleles (or genotypes) have different fitnesses, then to simply specify what these fitnesses are. Thus, the fitnesses of various alleles are specified as constant parameters in classical population genetics in much the same way that the parameters governing ecological interactions are treated as constants in ecological theory.

To illustrate this approach, consider a single-locus, diallelic model for a diploid species with nonoverlapping generations. For simplicity I focus on an autosomal locus with alleles "A" and "a." As a result there are three genotypes: "AA," "Aa," and "aa." In such models I specify the fitness of these three genotypes: W_{AA}, W_{Aa}, and W_{aa}. If you are measuring the frequency of the "A" allele in each generation (denoted by $p(t)$) in the gamete pool, then you can view W_{ij} as the number of gametes produced by an individual with genotype ij. Letting $N(t)$ denote the population size in generation t, there will be $N(t)p(t)^2$ "AA" homozygotes in that genera-

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tion, $N(t)2p(t)(1 - p(t))$ "Aa" heterozygotes, and $N(t)(1 - p(t))^2$ "aa" homozygotes. Each "AA" homozygote will produce W_{AA} gametes (all of which carry the "A" allele), each heterozygote will produce W_{Aa} gametes (only half of which carry the "A" allele), and each "aa" homozygote will produce W_{aa} gametes (none of which carry the "A" allele). Thus, the total number of "A"-carrying gametes in generation $t + 1$ will be $N(t)p(t)^2W_{AA} + N(t)p(t)(1 - p(t))W_{Aa}$, whereas the total number of gametes in generation $t + 1$ will be $N(t)p(t)^2W_{AA} + N(t)2p(t)(1 - p(t))W_{Aa} + N(t)(1 - p(t))^2W_{aa}$. Thus, the frequency of the "A" allele in generation $t + 1$ is as follows:

$$p(t+1) = \frac{N(t)\left(p(t)^2 W_{AA} + p(t)(1-p(t))W_{Aa}\right)}{N(t)\overline{W}(t)} = \frac{p(t)^2 W_{AA} + p(t)(1-p(t))W_{Aa}}{\overline{W}(t)}. \quad (13.4A)$$

Here, $\bar{W}(t) = p(t)^2W_{AA} + 2p(t)(1 - p(t))W_{Aa} + (1 - p(t))^2W_{aa}$ is the average fitness of the population at time t (see Hartl & Clark 1989, p. 151). Equation 13.4A reveals that if fitnesses W_{ij} do not depend on population density, the evolutionary dynamics of the population are unaffected by population density.

Further insight can be gained by dividing the numerator and the denominator of Equation 13.4A by W_{aa} to obtain the following:

$$p(t+1) = \frac{p(t)^2 w_{AA} + p(t)(1-p(t))w_{Aa}}{\overline{w}(t)}. \quad (13.4B)$$

In this case, $w_{ij} = W_{ij}/W_{aa}$. W_{ij} is referred to as the absolute fitness of genotype ij, where w_{ij} is the relative fitness of genotype ij (i.e., relative to genotype "aa," although you can use any genotype as the "standard" in this normalization). Equation 13.4B reveals that relative fitness, not absolute fitness, determines the evolutionary dynamics. Thus, even if the *absolute* fitnesses depend on the population density (i.e., W_{ij} is a function of N), the evolutionary dynamics will still be independent of this "ecological" variable provided that the *relative* fitnesses do not. For example, if the genotypic absolute fitnesses depend on population density and have the form $W_{ij} = F(N)c_{ij}$, where c_{ij} is a genotype-specific constant and $F(N)$ is some function of population density, then the evolutionary dynamics will still be independent of population density because the relative fitnesses are $w_{ij} = c_{aa}/c_{ij}$. This observation, that population density often cancels out the equation for allele frequency change, has lead to the widespread use of evolutionary models that ignore explicit ecological interactions involving population densities.

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Another useful formulation for the evolutionary dynamics is obtained by deriving an equation for the *change* in allele frequency in one generation—that is, $\Delta p(t) = p(t + 1) - p(t)$. Equation 13.4A, after some rearrangement, yields Wright's equation (Wright 1935, 1969):

$$\Delta p = \frac{p(1-p)}{2} \frac{1}{\overline{W}} \frac{d\overline{W}}{dp}. \tag{13.5A}$$

From Equation 13.4B, you find the equivalent equation in terms of relative fitness:

$$\Delta p = \frac{p(1-p)}{2} \frac{1}{\overline{w}} \frac{d\overline{w}}{dp}. \tag{13.5B}$$

Equations 13.5A and B reveal that natural selection results in a change in allele frequency such that mean absolute fitness, $\overline{W}$, and mean relative fitness, $\overline{w}$, increase (Crow & Kimura 1970, Hartl & Clark 1989, Hofbauer & Sigmund 1988). Moreover, because Equation 13.4B or 13.5B reveals that relative fitness (rather than absolute fitness) is the determinate of evolutionary change, many researchers standardize the fitnesses such that $w_{Aa} = 1 + s$, $w_{Aa} = 1 + s/2$, and $w_{aa} = 1$, where s is the selective advantage (or cost, if it is negative) of the "A" allele and the $s/2$ for the heterozygote assumes that alleles act additively (Crow & Kimura 1970). In this case, Equation 13.5 reduces to the following, particularly simple form:

$$\Delta p = \frac{p(1-p)}{2} \frac{s}{\overline{w}}. \tag{13.6}$$

From Equation 13.6 you can clearly see that the ecological dynamics (in terms of population density) can be safely ignored when trying to understand evolutionary change (in this simple model, at least) provided that population density has no effect on the relative selective advantage of the "A" allele, s.

Equation 13.6 represents a simple evolutionary model, but it has been widely used to address a variety of issues and partly forms the basis for the initial neglect of ecological details when studying evolutionary dynamics. As with ecological theory, evolutionary theory has gone far beyond this simple incarnation to explore how a range of other factors affects evolutionary change. It was the recognition that the selective advantage of any given allele, s, likely will depend on ecological context in many circumstances that lead to the first attempts to integrate the

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two. Treating the effects of natural selection arising from ecological interactions as a constant parameter, *s*, is simply not good enough for many situations.

13.3.2 | Optimization and Game Theory

An alternative approach for modelling evolution is the use of optimization and game-theoretic models. I treat them together because optimality models can be viewed as a special case of game-theoretic models. Typically, optimality models ignore the details of how the genotype of an organism gives rise to its phenotype and simply seek to characterize the phenotype that yields the highest fitness. Thus, optimality models require the specification of a fitness function, and the underlying assumption is that natural selection proceeds so as to maximize this function (Maynard Smith 1978, Parker & Maynard Smith 1990).

Optimality thinking and modelling has a long history in evolutionary biology, but the introduction of game-theoretic thinking and modelling to evolutionary biology took this approach to an entirely new level. Optimization models assume that the fitness of an individual depends only on that individual's phenotype, but it has long been appreciated that an individual's fitness is determined by the phenotypes of other individuals in the population as well. The introduction of game-theoretic ideas addressed this complexity, and it was motivated largely to model the evolution of social interactions for which optimality models were simply not tenable (Maynard Smith & Price 1973, Maynard Smith 1982). An individual's fitness as a result of some social interaction depends on the behaviours of all individuals involved; therefore, it no longer even makes sense to ask the question of what is optimal. The optimal behaviour is context specific, depending upon the behaviour of other individuals. As a result, focus moved from optimal phenotypes to evolutionarily stable phenotypes (Maynard Smith 1982). An evolutionarily stable strategy (ESS) is one such that if all individuals are using this phenotype, then no single individual can do better by unilaterally altering its phenotype (Maynard Smith 1982, Bulmer 1994). Optimality models are then a special case of such game-theoretic models in which the fitness of an individual depends only on its own phenotype.

As with optimality models, these original game-theoretic ideas were focused on the end point of evolution. The underlying idea was that new mutations periodically arise, and these either sweep to fixation or die out. Thus, the population is imagined as being monomorphic with the periodic introduction of new mutations. Eventually, after a series of new mutations and periodic allelic replacements, you might expect the population to arrive at a phenotype that is evolutionarily stable.

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The game-theoretic approach has been extended to many other situations involving different roles played by individuals (e.g., male versus female) and the possibility that a single phenotype is not an ESS but, rather, that a polymorphism is maintained. In addition, although this approach was often used to model the evolution of social interactions, it was soon appreciated that its utility extended well beyond this (e.g., see Lawlor & Maynard Smith 1976 and Reed & Stenseth 1984). For this chapter's purposes, it is important to note that this approach also proved useful for modelling ecological interactions because, for example, the resources available to an individual depend not only on its phenotype but also on the phenotypes of other individuals in the population (e.g., a particular resource will be abundant if few other individuals use it; see Lawlor & Maynard Smith 1976). Analogous considerations hold for other ecological interactions, making this a powerful approach for developing theory in evolutionary ecology (Abrams 2001).

13.4 | THEORETICAL EVOLUTIONARY ECOLOGY

A primary motivation for the development of theoretical evolutionary ecology was the realization that the separation of timescales assumed in much of the ecological literature, with the lack of explicit ecological detail in the evolutionary literature, was unrealistic. Are there new insights to be gained by creating a theory that bridges these two areas? Can evolutionary biology inform ecology by providing a new perspective on the study of the distribution and abundance of organisms? Can ecology inform evolutionary biology by providing a new perspective on the study of how natural selection guides evolutionary change? To answer these questions, a theory was built that explicitly examines the feedback between ecological and evolutionary processes.

As seen in the previous section, both ecological and evolutionary theory has centred on the development of dynamic systems models describing population dynamics and allele frequency dynamics, respectively. (Although game-theoretic models originally had no explicit dynamic, there was an implicit underlying dynamic.) As a result, from a mathematical standpoint, the mutual dependence and feedback between ecological and evolutionary processes has typically been modelled using some form of a coupled dynamic system between the two. As you will see here, this general structure underlies virtually all of the various approaches used in theoretical evolutionary ecology. In this section I highlight and explain four of these: (1) single-locus theory, (2) quantitative-genetic theory, (3) game theory, and (4) adaptive dynamics.

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13.4.1 | Single-Locus Theory

Some of the earliest attempts to create a synthetic theory in evolutionary ecology simply merged models of classical population genetics with those from ecology (Roughgarden 1996). Underlying this idea was the recognition that the per capita production of an individual in models such as that of Equation 13.2 (i.e., $\{1 + r - r(N/K)\}$) is the absolute fitness, W, in classical population-genetic models. Therefore, you can construct an ecological–evolutionary model by specifying different per capita productions (i.e., different fitnesses) for different potential genotypes. For example, in the logistic model of Equation 13.2 you might use the following:

$$W_{AA}(N) = \{1 + r_{AA} - r_{AA}(N/K_{AA})\} \tag{13.7A}$$

$$W_{Aa}(N) = \{1 + r_{Aa} - r_{Aa}(N/K_{Aa})\} \tag{13.7B}$$

$$W_{aa}(N) = \{1 + r_{aa} - r_{aa}(N/K_{aa})\}. \tag{13.7C}$$

In these equations, the parameters of the per capita production are now genotype specific. You can still define the population average fitness as follows:

$$\overline{W}(N,p) = p^2 W_{AA}(N) + 2p(1-p)W_{Aa}(N) + (1-p)^2 W_{aa}(N). \tag{13.8}$$

Equation 13.5A is still valid for the evolutionary dynamics. Now, however, you must also have an equation that governs the ecological dynamics because the population density, N, does not cancel out the equation for allele frequency change. Adding up the production of the three different genotypes in the population yields the following equation:

$$N(t+1) = \overline{W}(N(t), p(t))N(t). \tag{13.9}$$

If you instead derive an equation for the *change* in population size, $\Delta N(t) = N(t + 1) = N(t)$, you get the coupled evolutionary–ecological model:

$$\Delta N = (\overline{W}(N,p) - 1)N \tag{13.10A}$$

$$\Delta p = \frac{p(1-p)}{2} \frac{1}{\overline{W}(N,p)} \frac{\partial \overline{W}}{\partial p}. \tag{13.10B}$$

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Equations 13.10A and B represent one of the first attempts to construct a theory of evolutionary ecology (Roughgarden 1971, 1996; Charlesworth 1971). Notice, however, that the form of the fitness functions in Equation 13.7 is somewhat restrictive in that a genotype's reproductive success depends only on the total *density* of the population but not on its genetic composition. More generally, you might expect different genotypes to have different competitive effects on one another (e.g., perhaps similar genotypes compete more strongly with one another). In this case, the fitness of an "AA" homozygote would generalize to the following:

$$W_{AA}(p,N) = \left\{1 + r_{AA} - r_{AA}\frac{(\alpha_{AA,AA}Np^2 + \alpha_{AA,Aa}N2p(1-p) + \alpha_{AA,aa}Np(1-p))}{K_{AA}}\right\}$$
$$= \left\{1 + r_{AA} - r_{AA}\frac{N\overline{\alpha}_{AA}(p)}{K_{AA}}\right\}. \quad (13.11)$$

Here, $\alpha_{ij,kl}$ is the competitive effect of genotype *ij* on genotype *kl*, and $\overline{\alpha}_{AA}(p) = (\alpha_{AA,AA}p^2 + \alpha_{AA,Aa}2p(1-p) + \alpha_{AA,aa}p(1-p))$ is the population average competitive effect on genotype "AA." Thus, in general, you have the following:

$$W_{ij}(p,N)\left\{1 + r_{ij} - r_{ij}\frac{N\overline{\alpha}_{ij}(p)}{K_{ij}}\right\}. \quad (13.12)$$

Here, $\overline{\alpha}_{ij}(p) = (\alpha_{ij,AA}p^2 + \alpha_{ij,Aa}2p(1-p) + \alpha_{ij,aa}p(1-p))$.

Notice that the fitness of each genotype is now both density and *frequency* dependent (i.e., it depends on allele frequency, *p*); therefore, Equation 13.5A is no longer valid because it was derived under the assumption that the genotypic fitnesses were not functions of allele frequency. You can generalize this equation for the present purposes, however, to obtain the following (e.g., see Taper & Case 1992):

$$\Delta N = (\overline{W}(N,p) - 1)N \quad (13.13A)$$

$$\Delta p = \frac{p(1-p)}{2}\frac{1}{\overline{W}(N,p)}\left[\frac{\partial \overline{W}}{\partial p} + \overline{\frac{\partial W}{\partial p}}\right]. \quad (13.13B)$$

This is the coupled ecological–evolutionary model, where $\overline{W}(N, p) = p^2W_{AA}(p,N) + 2p(1-p)W_{Aa}(p,N) + (1-p)^2W_{aa}(p,N)$ and $\overline{\partial W/\partial p} = p^2(\partial W_{AA}/\partial p) + 2p(1-p)(\partial W_{Aa}/\partial p) + (1-p)^2(\partial W_{aa}/\partial p)$.

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One limitation of the preceding approach is that the evolutionary dynamics are restricted to those alleles that start in the system. No allowance is made for the introduction of new alleles through mutation. As a result, the model makes explicit predictions about short-term evolutionary change, but it has nothing to say about the more long-term process of evolutionary change that occurs as a result of continued mutation and repeated allelic replacements. This difficulty has been alleviated to some degree by considering multiple alleles; still, mutation and longer-term evolution are neglected using this approach.

Interestingly, the single-locus approach has been used to make predictions about the ultimate end point of long-term evolution by using what amounts to a game-theoretic argument. You would ask, is there an allele that, if present, can exclude all other possible mutations? For example, in some models similar to Equation 13.10B, it can been shown that the allele that can exclude all others is that which sustains the highest population density (Roughgarden 1971, Charlesworth 1971). Once this allele is determined, you can then use the preceding theoretical framework to predict its short-term dynamics in terms of its frequency. Of course, this theoretical approach still cannot make predictions about the long-term evolutionary dynamics of a population towards this end point as a result of recurrent mutation and selection. For that, an alternative approach is required.

13.4.2 | Quantitative-Genetic Theory

The underpinnings of quantitative-genetic theory date back to the development of classical population genetics (Provine 2001), but the specific incarnation most frequently used today was developed at roughly the same time as the preceding single-locus theory in evolutionary ecology (Kimura 1965; Lande 1976a, 1976b, 1979; Lande & Arnold 1983). The initial development of this theory was not motivated by (or even clearly suited to) modelling in evolutionary ecology. Rather, the first versions of this theory took the population geneticist's perspective of not treating ecological interactions explicitly (for reasons outlined earlier) and simply assuming a largely fixed selective regime under which a population evolves. A major advantage of this theory was that it allowed for standing genetic variation in a trait (as is commonly observed), and this variation was maintained through a balance between mutation and recombination, with selection. Also, as opposed to the single-locus theory, the quantitative-genetic approach typically supposed that there were numerous loci affecting the trait of interest, with each locus having a small effect. As a result, the distribution of genotypes in the popula-

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tion could often be well approximated by a Gaussian (normal) distribution with a particular mean and variance.

The central question of interest in this framework is, then, How does the distribution of genotypes (and the resulting distribution of phenotypes) evolve over time? (Lande 1976a). As selective conditions change, evolutionary change occurs, with abundant genetic variation being maintained through a balance between loss of alleles and mutational input. If a researcher is willing to assume that this distribution remains Gaussian, then its evolutionary dynamics can be tracked simply by following the evolution of the mean and the variance of this distribution (because these two parameters completely specify a Gaussian distribution). Even more simply, many researchers have further assumed that the variance of the distribution remains largely constant over the time span of interest and, therefore, that evolutionary change can be tracked simply by following the population mean.

This quantitative-genetic framework was soon generalized to allow ecological interactions. One of the primary interests in doing so was to model competition for resources and character displacement (Brown & Wilson 1956, Slatkin 1980). When two species compete for a common resource pool, do you expect evolutionary divergence in their resource use? If so, then you would expect the phenotypic characteristics related to resource extraction in such species to be divergent where their geographical ranges overlap (Brown & Wilson 1956, Grant 1972).

To gain an appreciation for how this theoretical approach is used, consider a model analogous to that of the logistic growth model used earlier. Now, rather than having a few discrete genotypes (and thus phenotypes), you have a continuous distribution of genotypes (and thus phenotypes; Roughgarden 1983; Taper & Case 1992). An individual's fitness might be density dependent, in which case you have the following fitness of an individual with quantitative trait z:

$$W(z,N) = 1 + r(z) - r(z)\frac{N}{K(z)}. \tag{13.14}$$

Now, r and K are functions of the quantitative trait z.

Denote the average phenotypic value in the population in generation t by $\bar{z}(t)$. This will also be the average genotypic value in the population in that generation if you assume that an individual's phenotype, z, is equal to its genotype, x, plus some random environmental deviation, e; that is, $z = x + e$. Also denote the average phenotypic value in the population after selection has occurred by $\bar{z}(t)_s$. Importantly, this is no longer the average genotypic value of the population because natural selection has acted on the phenotypes, and if, for example, it favoured larger phenotypes, then

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some of the "selected" population will have genotypes coding for small traits because they happened to have a large positive environmental deviation. Thus, the average phenotype in the next generation, $\bar{z}(t+1)$, (which is equivalent to the average genotype after selection assuming random mating) is given by $\bar{z}(t+1) = \bar{z}(t) + h^2(\bar{z}(t)_s - \bar{z}(t))$, where h^2 is the heritability of the trait given by $h^2 = \sigma_g/\sigma_p$. Here, σ_g is the additive genetic variance of the trait, and $\sigma_p = \sigma_p + \sigma_e$ is the total phenotypic variance of the trait (assumed to equal the additive genetic variance plus the variance in the environmental deviation, σ_e) (Lande 1976a, 1976b). Therefore, the evolutionary change in one generation is as follows:

$$\Delta\bar{z}(t) = h^2(\bar{z}(t)_s - \bar{z}(t)). \tag{13.15}$$

In the present model, population average fitness is $\overline{W} = \int_{-\infty}^{\infty} p(z)W(z,N)dz$; therefore, you can verify that, under theassumption that the phenotypic trait distribution, $p(z)$, is Gaussian with mean $\bar{z}$ and variance σ_p, Equation 13.15 can be rewritten in terms of the population average fitness, giving the following coupled evolutionary–ecological system (Slatkin 1980, Taper & Case 1992):

$$\Delta N = (\overline{W} - 1)N \tag{13.16A}$$

$$\Delta\bar{z}(t) = \sigma_g \frac{1}{\overline{W}} \frac{\partial \overline{W}}{\partial \bar{z}}. \tag{13.16B}$$

Note the similarity between Equations 13.16A and B and the single-locus system in Equations 13.10A and B. The ecological dynamics and evolutionary dynamics take an identical form in each. This is readily apparent for the ecological dynamics, and the evolutionary dynamics can both be viewed as the genetic variance in trait ($p(1-p)/2$ versus σ_g) multiplied by the proportional increase in mean fitness that occurs with an increase in population mean trait value.

Interestingly, you can also account for frequency-dependent selection using this approach, just as you did in the single-locus approach. In this case, the fitness of an individual with trait z is as follows (Slatkin 1980, Taper & Case 1992):

$$\begin{aligned} W(z,N) &= 1 + r(z) - r(z)\frac{N\int_{-\infty}^{\infty} p(y)\alpha(z,y)dy}{K(z)} \\ &= 1 + r(z) - r(z)\frac{N\bar{\alpha}(z)}{K(z)}. \end{aligned} \tag{13.17}$$

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Now, $\alpha(z, y)$ is a function of two variables, giving the competitive effect of an individual with phenotype y on an individual with phenotype z, and I have defined $\overline{\alpha}(z) = \int_{-\infty}^{\infty} p(y)\alpha(z, y)dy$ to be the average competitive effect on an individual with trait z. You can verify that because Equations 13.16A and B now become the following (Taper & Case 1992):

$$\Delta N = (\overline{W} - 1)N \tag{13.18A}$$

$$\Delta\bar{z} = \sigma_g \frac{1}{\overline{W}}\left[\frac{\partial \overline{W}}{\partial \bar{z}} + \frac{\overline{\partial W}}{\partial \bar{z}}\right]. \tag{13.18B}$$

Here, $\overline{\partial W/\partial \bar{z}} = \int_{-\infty}^{\infty} p(z)(\partial W/\partial \bar{z})dz$. Again, when there is both frequency and density dependence, note the correspondence between the quantitative-genetic system in Equations 13.18A and B and the single-locus system in Equations 13.13A and B.

The preceding case of intraspecific competition (i.e., Equation 13.18A or B) has received considerable attention as an example of quantitative-genetic theory in evolutionary ecology; therefore, it is worth examining in more detail. To do so, you need to specify explicit functions for $r(z)$, $\alpha(z,y)$ and $K(z)$ in the fitness function of Equation 13.17. Common assumptions are that r is independent of z, that K (the carrying capacity) is maximal for some intermediate value of z, and that $\alpha(z, y)$ is a unimodal function with a value of unity when $y = z$ (the competitive effect of any individual on an individual with phenotype z is one when the two have identical phenotypes) and it decreases to zero as the difference between the two phenotypes increases (Roughgarden 1972, Taper & Case 1992). With these assumptions, Equations 13.18A and B predict that the species will evolve towards a phenotype that maximizes the carrying capacity. Note that here I have focused solely on the evolution of the mean trait value, and I have assumed that the distribution remains Gaussian (in accord with most quantitative-genetic models).

Interestingly, some of the earliest work in this area examined the simultaneous evolution of the genetic variance in the trait as well, and these results demonstrated that some parameter values result in a stable equilibrium variance whereas others result in the variance decreasing to zero (Slatkin 1980). The latter case occurs whenever the carrying capacity function, $K(z)$, is very narrow relative to the competition coefficient function $\alpha(z,y)$. Conversely, variation is maintained in the former case when the resource base is broad enough to support the evolution of a variety of resource extraction strategies (relative to the competition coefficient function).

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As mentioned earlier, a two-species version of the preceding model has received the most attention in the literature, particularly in the context of studying interspecific competition and evolutionary character displacement (Slatkin 1980; Roughgarden 1983; Brown & Vincent 1987; Taper & Case 1992; Vincent, Cohen, & Brown 1993). The details of such models are a natural extension of Equations 13.18A and B but for the Lotka–Volterra competition Equations 13.3A and B. If you assume that the genetic variance does not evolve (or does so slowly enough that you can ignore it and simply follow the mean phenotype), such models have demonstrated that evolutionary character displacement occurs for some parameter values and not for others (Taper & Case 1992). Moreover, character displacement occurs precisely for those conditions under which the natural selection favours a stable level of genetic variance for the single-species model (Slatkin 1980). This is intuitively reasonable. If the resource base is broad enough relative to the spectrum of resource use by any given species (i.e., the carrying capacity function, K, is wide relative to the width of the competition coefficient functions, α), then the system can support two different (i.e., divergent) resource extraction phenotypes. Indeed, as you will see shortly in the section on adaptive dynamics, natural selection favours this evolutionary divergence into two phenotypes in the single-species model, but the assumptions of sexual reproduction and recombination that underlie the use of a Gaussian distribution in quantitative genetics prevent a single species from evolving such a dimorphism. Assortative mating within phenotype would also have to evolve to allow such divergence when starting with a single species (e.g., see Dieckmann & Doebeli 1999).

The quantitative-genetic framework has been extremely influential in evolutionary ecology, and it has been extended and used for a variety of questions and ecological interactions. In more complex models, however, Equations 13.18A and B (or analogous equations for the ecological situation of interest) become difficult to use because it is often not possible to explicitly calculate the expression on the right-hand side of Equation 13.18B. As a result, some researchers have begun to use an approximation of this equation. This approximation is most easily derived by first noting that Equation 13.18B can be rewritten as follows:

$$\Delta\bar{z} = \frac{\sigma_g}{\bar{W}}\frac{\overline{\partial W}}{\partial z}. \tag{13.19}$$

Here, $\overline{\partial W/\partial z} = \int_{-\infty}^{\infty} p(z)(\partial W/\partial z)dz$ (Lande & Arnold 1983; Iwasa, Pomiankowski, & Nee 1991; Taylor 1996). Notice the subtle distinction between

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this and the second term on the right-hand side of Equation 13.18B. In Equation 13.18B, the expectation (over the distribution p) is of the derivative of W with respect to $\bar{z}$ (the population mean phenotype), whereas in Equation 13.19, both terms on the right-hand side of Equation 13.18B have reduced to the expectation (again over the distribution p) of the derivative of W with respect to z (an individual's phenotype). Importantly, Equations 13.18B and 13.19 are equivalent, but Equation 13.19 is written in a much simpler form. It reveals that evolutionary change in the population mean phenotype occurs in a direction given by the sign of the change in fitness that occurs with an increase in an individual's phenotype, averaged over all individuals in the population (Lande & Arnold 1983).

An approximation to Equation 13.19 is then easily obtained under the condition that the variance among phenotypes in the population is relatively small. In such cases, the expectation of any function is approximately equal to the function evaluated at the mean; therefore, you obtain the following approximation (Iwasa, Pomiankowski, & Nee 1991; Taylor 1996):

$$\Delta\bar{z} = \frac{\sigma_g}{\bar{W}} \left. \frac{\partial W}{\partial z} \right|_{z=\bar{z}}. \tag{13.20}$$

Because the right-hand side of Equation 13.20 is readily calculated under most circumstances, this approximation has been used extensively in recent years as a simpler means for constructing quantitative-genetic models (reviewed in Abrams 2001).

13.4.3 | Game Theory

The introduction and development of game theory in evolutionary biology essentially paralleled the development of the single-locus and the quantitative-genetic theories. As with quantitative genetics, the game-theoretic approach was not originally devised explicitly for modelling in evolutionary ecology. Rather, its initial focus was largely on the evolution of social traits (Maynard Smith & Price 1973, Maynard Smith 1982). Nevertheless, it was occasionally used to model the evolutionary consequences of ecological interactions (e.g., see Lawlor & Maynard Smith 1976; Reed & Stenseth 1984; Brown & Vincent 1987a, 1987b; Vincent, Cohen, & Brown 1993; and Abrams *et al.* 1993), and the most recent incarnation of the game-theoretic approach (which will be described shortly under the heading "adaptive dynamics") focuses largely on such ecological interactions.

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Although one of the main motivations for the original development of theoretical evolutionary ecology was to dispense with the artificial separation of timescales between ecological and evolutionary processes, it is interesting to note that such a separation is invariably used in game-theoretic models of ecological interactions (e.g., see Lawlor & Maynard Smith 1976 and Reed & Stenseth 1984). The typical approach is to suppose that the population in question is monomorphic (i.e., all individuals have identical phenotypes, and this phenotype is termed the "resident") and then assumes that this population reaches a population dynamic equilibrium in terms of the underlying ecological model of interest. Then imagine introducing a rare mutant allele coding for different phenotypes and ask if this mutant can increase in numbers (i.e., if it can invade).

As a simple example, consider the logistic model of one species presented earlier (i.e., Equation 13.1), but where the carrying capacity, K, depends on some quantitative trait, $\hat{z}$ ("hats" are often used to signify the phenotype of the resident in game theory):

$$\begin{aligned} N(t+1) &= N(t) + N(t)r\left(1 - \frac{N}{K(\hat{z})}\right) \\ &= N(t)\left\{1 + r - r\frac{N}{K(\hat{z})}\right\}. \end{aligned} \tag{13.21}$$

At ecological equilibrium, the population size will be $N = K(\hat{z})$, which reveals that the equilibrium density depends on the resident phenotype. Then you can ask what the growth rate of a rare mutant will be if it has phenotype z. When rare, it will have a negligible effect on the population size; therefore, its initial (i.e., invasion) dynamics will be governed by the following equation:

$$\begin{aligned} N_{mut}(t+1) &= N_{mut}(t)\left\{1 + r - r\frac{N_{res}}{K(z)}\right\} \\ &= N_{mut}(t)\left\{1 + r - r\frac{K(\hat{z})}{K(z)}\right\}. \end{aligned} \tag{13.22}$$

Here, you use the equilibrium density of the resident type as the mutant's per capita growth factor because the mutant is rare and therefore the density of the resident type will be the main determinate of the mutant's growth factor. Thus, you can see that the mutant will invade if (and only if) the following is true:

$$\lambda(z, \hat{z}) \equiv 1 + r - r\frac{K(\hat{z})}{K(z)} > 1. \tag{13.23}$$

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I have defined $\lambda(z, \hat{z})$ to be the growth factor of a rare mutant using strategy z in a population dominated by strategy $\hat{z}$. $\lambda(z, \hat{z})$ is sometimes called the "mutant's invasion fitness" (Metz, Nisbet, & Geritz 1992) or the "mutant's fitness," but it (and more general extensions of it for other ecological interactions) have also been referred to as the fitness "generating-function" or "G-function" in the literature (Brown & Vincent 1987a, 1987b; Vincent, Cohen, & Brown 1993). Notice that the mutant dies out if $\lambda(z, \hat{z})$ is less than one, and it is neutral if $\lambda(z, \hat{z})$ equals one (which, of course, occurs when $z = \hat{z}$).

The primary goal of the game-theoretic approach is to characterize phenotypes that are evolutionarily stable (i.e., ESSs). An ESS has the property that, if all individuals in the population adopt this strategy, no alternative can invade (Maynard Smith 1982). Using the definition in Equation 13.23, you can see that an ESS, z^*, must satisfy the following:

$$\lambda\left(z, z^*\right) \leq \lambda\left(z^*, z^*\right). \tag{13.24}$$

This must be satisfied for all mutant strategies z (and equality occurs when $z = z^*$). The inequality in Equation 13.24 is referred to as the Nash equilibrium condition (Bulmer 1994), and using Equation 13.23 in this produces the following:

$$1 + r - r\frac{K\left(z^*\right)}{K(z)} \leq 1 + r - r\frac{K\left(z^*\right)}{K\left(z^*\right)}. \tag{13.25A}$$

Alternatively, it can produce the following:

$$K(z) \leq K\left(z^*\right). \tag{13.25B}$$

This reveals that the ESS trait value in this model maximizes the carrying capacity.

Often it is difficult to use the condition in Equation 13.24 to characterize the ESS; therefore, researchers use "local" conditions instead. In particular, because the condition in Equation 13.24 states that $\lambda(z, \hat{z})$ must be maximized in its first argument (i.e., in z) at $z = \hat{z} = z^*$, we know from calculus that the first derivative at this point must equal zero. In addition, if this point is to represent a maximum rather than a minimum,

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we know that the second derivative at this point must be negative. This gives the following two conditions:

$$\left.\frac{\partial \lambda}{\partial z}\right|_{z=\hat{z}=z^*} = 0 \tag{13.26A}$$

$$\left.\frac{\partial^2 \lambda}{\partial z^2}\right|_{z=\hat{z}=z^*} \leq 0. \tag{13.26B}$$

In the preceding example, you can verify that the conditions in Equations 13.26A and B evaluate to $dK/dz = 0$ and $d^2K/dz^2 \leq 0$.

The preceding game-theoretic approach was simple because the underlying ecological model of interest was simple. As the ecological scenario becomes more sophisticated, the underlying approach remains the same, but the expression for a mutant's fitness becomes more complex (Metz, Nisbet, & Geritz 1992). This approach has also been used to model the coevolutionary dynamics of more than one species, and such cases are simply treated as "two-player" games in which each species has an expression specifying mutant fitness and, in general, depending on the densities of both species, the resident phenotypes of both species, or the combination of these (Hofbauer & Sigmund 1988, Abrams 2001).

The game-theoretic approach initially placed most emphasis on characterizing ESSs with the underlying idea that such phenotypes would be the ultimate end points of evolutionary change. Implicit in this technique is the notion that, when a new mutation invades, the evolutionary–ecological system is perturbed and the mutant strategy then sweeps through to fixation (it is almost always assumed that a polymorphism does not result). The ecological dynamics will then have reached new equilibrium, and the invasion process occurs again. Thus evolution is viewed as a succession of mutants arising, but on a timescale much slower than the ecological dynamics, and the notion was that the system would eventually attain the uninvadable strategy (i.e., the ESS).

Although the previously mentioned evolutionary processes implicitly formed the foundation of game theory, initially little attention was paid to the evolutionary dynamics of the population as it approached this ESS. This shortcoming was recognized relatively early in the development of game theory (Eshel 1983), and attempts were made to address this issue more quantitatively. One of the most profound insights to come out of this research was the finding that populations need not evolve towards an ESS (Eshel 1983, Taylor 1989, Christiansen 1991, 4 Abrams *et al.* 1993, Geritz *et al.* 1998). Evolutionarily stable strategies can be evolutionarily unattainable (Eshel 1983). This counterintuitive

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finding arises because natural selection in most game-theoretic models is frequency dependent. As a result, a phenotype can be an ESS in that, if most members of the population adopt this phenotype then no alternative can do better. Nevertheless, it can still be evolutionarily unattainable in the sense that, if most members of the population adopt a phenotype slightly different from this ESS, only those phenotypes that are even more different from the ESS can invade. Thus, natural selection can drive the evolution of a population from an ESS even though, if the population was started at the ESS, it would remain there (Taylor 1989, Christiansen 1991).

Even more interesting, it was found that the conditions for evolutionary attainability and the ESS conditions (i.e., the Nash equilibrium condition) are essentially independent. There can be phenotypes that are evolutionarily attainable but not ESSs, phenotypes that are ESSs but not evolutionarily attainable, and phenotypes that are both ESSs and evolutionarily attainable (Geritz *et al.* 1998). As you will see later, the first of these situations has come to be the primary focus of adaptive dynamics.

As an example, again consider Equation 13.21, but now include the assumption that selection is both density-dependent and frequency-dependent as in Equations 13.12 and 13.13B and in Equations 13.17 and 13.18A and B. In this case, you have the following:

$$N(t+1) = N(t)\left\{1 + r - r\frac{\alpha(\hat{z},\hat{z})N}{K(\hat{z})}\right\}. \tag{13.27}$$

Again, $\alpha(x, y)$ is the competitive effect of an individual with phenotype y on an individual with phenotype x (and $\alpha(x, x) = 1$). The mutant's fitness function, Equation 13.23, then becomes as follows:

$$\lambda(z,\hat{z}) \equiv 1 + r - r\frac{\alpha(z,\hat{z})K(\hat{z})}{K(z)}. \tag{13.28}$$

To be more concrete, I will use the particular functions $K(z) = \kappa e^{-z^2/2\sigma_k}$ and $\alpha(z, \hat{z}) = e^{-(z-\hat{z})^2/2\sigma_\alpha}$. These functions have been used numerous times in the literature (reviewed in Day 2000) and are chosen largely for mathematical convenience. With these, the conditions in Equations 13.26A and B become $z^* = 0$ and $\sigma_k \le \sigma_\alpha$, respectively. Thus, the phenotype "0" is an ESS if (and only if) $\sigma_k \le \sigma_\alpha$.

Now consider the question of the evolutionary attainability of $z^* = 0$. To begin, suppose that the majority of the population is using a phenotype slightly below z^*; that is, $\hat{z} < z^*$. For natural selection to drive the population towards z^*, mutants slightly above $\hat{z}$ must be able to invade

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(i.e., have higher fitness than the resident) and mutants slightly below $\hat{z}$ must not be able to invade (i.e., have lower fitness than the resident). Mathematically, you can express this by requiring the following:

$$\left.\frac{\partial \lambda}{\partial z}\right|_{z=\hat{z}} > 0 \quad \text{when} \quad \hat{z} < z^*. \tag{13.29A}$$

The inequality in Equation 13.29A states that the fitness gradient (i.e., the direction of increasing fitness) points towards z^* when the resident phenotype is below z^*. An analogous consideration also leads to the following condition:

$$\left.\frac{\partial \lambda}{\partial z}\right|_{z=\hat{z}} < 0 \quad \text{when} \quad \hat{z} > z^*. \tag{13.29B}$$

This states that the fitness gradient points towards z^* when the resident phenotype is above z^* as well. Together, the conditions in Equations 13.29A and B imply that the fitness gradient (which is a function of the population resident strategy $\hat{z}$ only; i.e., $\partial\lambda/\partial z|_{z=\hat{z}}$) decreases as the population resident strategy, $\hat{z}$, increases, passing from positive to negative at $\hat{z} = z^*$. Locally (i.e., near $\hat{z} = z^*$), you can express this by requiring that the derivative of $\partial\lambda/\partial z|_{z=\hat{z}}$ with respect to $\hat{z}$ be negative at $\hat{z} = z^*$:

$$\frac{d}{d\hat{z}}\left\{\left.\frac{\partial \lambda}{\partial z}\right|_{z=\hat{z}}\right\}_{\hat{z}=z^*} < 0. \tag{13.30A}$$

This can also be expressed as follows:

$$\left[\frac{\partial^2 \lambda}{\partial z^2} + \frac{\partial^2 \lambda}{\partial \hat{z} \partial z}\right]_{z=\hat{z}=z^*} < 0. \tag{13.30B}$$

The condition in Equation 13.30A, or equivalently in Equation 13.30B, is often referred to as the convergence stability condition (Bulmer 1994) because it implies that natural selection acts in such a way as to cause the population resident strategy to converge to z^*.

Returning to the example in Equation 13.28, you can use either Equation 13.30A or Equation 13.30B to show that $z^* = 0$ is convergence stable provided that $-r/\sigma_k < 0$, which is always satisfied. Therefore, you have the following two possibilities: (1) $\sigma_k < \sigma_\alpha$, in which case $z^* = 0$ is convergence stable and an ESS, or (2) $\sigma_k < \sigma_\alpha$, in which case $z^* = 0$ is convergence stable but *not* an ESS. In case 1, you can expect the population to evolve towards $z^* = 0$ and to remain there indefinitely. In case 2, you again can expect the population to evolve towards $z^* = 0$, but once there,

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natural selection becomes disruptive, favouring any phenotype other than $z^* = 0$. At this point some form of evolutionary diversification will occur (Taylor 1989, Christiansen 1991).

Biologically, case 2 can be understood as follows: Competition for resources always makes it beneficial to have a phenotype that is different from other individuals. At the same time, because the carrying capacity is maximized at $z = 0$, natural selection favours evolution towards this phenotype. When the population is not at this phenotype, mutants closer to $z = 0$ gain in both ways (i.e., they have the benefit of being different and the benefit of having a higher carrying capacity). This is why $z^* = 0$ is always convergence stable. If the width of the competition function is narrow relative to the carrying capacity function, however (i.e., if $\sigma_\alpha < \sigma_k$, meaning that any given phenotype is specialized in its resource use), then once the population reaches $z^* = 0$, the strength of selection for being different is strong enough to more than outweigh the loss in carrying capacity that comes from having a phenotype $z \neq 0$ and evolutionary diversification occurs (see Werner & Sherry 1986, Bolnick *et al.* 2002, Bolnick *et al.* 2003, and Bolnick 2004 for interesting empirical examples). At this stage, the preceding model no longer provides an adequate description of the evolutionary dynamics and therefore must be extended in some way to allow for a polymorphism (Christiansen 1991, Geritz *et al.* 1998). 5

This distinction between stability against invasion of rare mutants (i.e., ESS) and convergence stability is biologically interesting because it illustrates the potential for a trait to evolve to a point at which natural selection becomes disruptive. This finding, that biological interactions give rise to endogenously generated disruptive selection, was implicit in the early results of Ilan Eshel (1983), and it was noted more explicitly by Peter Taylor (1989) that this should result "in a polymorphic population which is [no longer] described by the [original fitness] function." Christiansen (1991) developed these ideas of the evolution of polymorphisms more explicitly and illustrated them with the preceding example, as did Joel Brown and Tom Vincent (1987a, 1987b; Vincent, Cohen, & Brown 1993; Abrams *et al.* 1993). 6 7

Researchers using the game-theoretic approach to modelling evolutionary ecology have also noted the fundamental similarities between the preceding results and the previous quantitative-genetic models (Charlesworth 1990; Iwasa, Pomiankowski, & Nee 1991; Taper & Case 1992; Abrams *et al.* 1993; Abrams 2001; Taylor 1996; Taylor & Day 1997). In particular, Eshel's (1983) idea that natural selection should drive the evolution of a population in a direction given by the sign of $\partial\lambda/\partial z|_{z=\hat{z}}$ closely parallels the quantitative-genetic equation for the evolutionary 8

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dynamics of the mean trait value. More specifically, Eshel's ideas were based on the idea that the evolutionary change in the population resident strategy is proportional to $\partial\lambda/\partial z|_{z=\hat{z}}$:

$$\Delta\hat{z} \propto \left.\frac{\partial\lambda}{\partial z}\right|_{z=\hat{z}}. \tag{13.31}$$

Note the correspondence between this equation and Equation 13.19 or its approximation, Equation 13.20. These results are identical if you make the identification $\lambda = W$ except that, unlike quantitative-genetic models, the game-theoretic approach assumes a separation of ecological and evolutionary timescales and therefore does not have a coupled equation for the ecological dynamics. This correspondence also reveals that the convergence stability condition of Eshel (1983) corresponds to the dynamics stability of equilibria in quantitative-genetic models (provided that the ecological dynamics are fast relative to evolution).

It has also been shown that the ESS condition in Equation 13.26B corresponds to stability of the genetic variance in quantitative-genetic models (Taylor & Day 1997). It should come as no surprise that the conditions under which evolutionary diversification occurs in game-theoretic models of competition for resources are essentially identical to those in quantitative genetics under which a single species reaches an equilibrium variance. The potential for evolutionary diversification into a polymorphism was not fully recognized in single-species quantitative-genetic models because sexual recombination maintained a unimodal distribution of genotypes.

Lastly, it is important to stress that the preceding game-theoretic ideas and techniques are often employed under the assumption of asexual reproduction but this need not be the case. Indeed, several studies have used this approach in the context of explicit classical genetic models involving sexual populations with various forms of inheritance, including diploidy and haplodiploidy (Taylor 1989, 1996; Christiansen 1991 and references therein; Eshel, Motro, & Sansone 1997). It is also interesting to note that this game-theoretic approach is closely aligned with more recent developments of Fisher's geometrical model of evolution in which mutations periodically arise and either sweep to fixation or die out (Orr 1998). The chief difference with these recent developments is the focus on making predictions about the distribution of sizes of allelic effects for those mutations that reach fixation (Orr 1998, 2003). These recent models do not involve frequency-dependent selection, however, and it would be interesting to extend them in this direction so

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that they might be more readily applicable to modelling in evolutionary ecology.

13.4.4 | Adaptive Dynamics

In recent years there has been a flood of interest in modelling ecological–evolutionary feedbacks using a technique that has come to be referred to as adaptive dynamics (Gavrilets & Waxman, in press). Different researchers have different, and often strongly held, opinions about what this approach represents and how it differs from previous theoretical developments. In line with the motivation for the symposium that spawned this volume, in this section I present these recent developments and consider the question of whether they represent a paradigm shift from previous approaches or whether they are simply a refinement and natural extension. 9

In short, my perspective is that adaptive dynamics as a field of study best thought of as a natural outgrowth of previous game-theoretic ideas (as they have been applied to evolutionary ecology). The seeds of, and even some of the most fundamental developments in, adaptive dynamics were clearly present in game-theoretic modelling and in some aspects of quantitative-genetic modelling. That is not to say that the developments embodied by adaptive dynamics have not been important; instead, these contributions are better thought of as developments within game theory rather than as a new approach.

One fundamental focal point of adaptive dynamics is on situations in which a trait value is convergence stable but not an ESS (Geritz *et al.* 1998). Such trait values have been given various names within the game-theoretic literature, but adaptive dynamics refers to them explicitly as branching points under the idea that evolutionary branching (e.g., speciation) is favoured by selection at these points (Geritz *et al.* 1998). As already mentioned, it was well known that such points occur within game-theoretic models (Eshel 1983, Taylor 1989), including those for the evolution of traits involved in ecological interactions such as exploitative competition for resources (e.g., see Christiansen 1991 and Abrams *et al.* 1993). It was also appreciated in this literature that such points will tend to lead to some sort of evolutionary diversification, such as a genetic polymorphism (Christiansen 1991). Thus, the existence of such interesting phenomena, as well as their evolutionary significance and implications, is not a finding that can be attributed to developments of adaptive dynamics. There are, however, at least three important developments (in my opinion) that have grown out of this research. I consider each of these in turn. 10

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First, research in adaptive dynamics has demonstrated that branching points might be a general feature of natural systems, because it is common for models of all sorts of ecological interactions, as well as all sorts of traits, to give rise to such phenomena (Doebeli & Dieckmann 2000). Of course, these findings might just as well have been developed within the game-theoretic approach of the previous section simply by examining various models using this approach (and indeed, from a conceptual standpoint, that is what was done). Nevertheless, these developments have been carried out largely by researchers who work under the rubric of adaptive dynamics.

Second, although it was recognized that diversification is favoured by selection at branching points by earlier game-theoretic approaches, and even though some treatments even modelled the initial stages of such diversification, this evolutionary divergence was not the focus of much modelling until the field of adaptive dynamics began to grow (Geritz *et al*. 1998; Gavrilets & Waxman, in press). Again, I would argue that there is nothing distinct in doing this that necessarily warrants giving it a name other than game theory, but these developments have also been carried out largely by researchers working in adaptive dynamics.

Third, and I would argue most significantly, researchers in this field provided a coherent and explicit mathematical underpinning for the somewhat heuristic evolutionary dynamic that Eshel implicitly used (i.e., Equation 13.31). Eshel (1983) and subsequent authors (Taylor 1989, 11 Christiansen 1991, Abrams *et al*. 1993) identified branching points and their evolutionary significance (using a different terminology), but it was research within adaptive dynamics that provided an explicit account of the implicit evolutionary dynamic used in game theory. I briefly review this development here because it turns out to have a simple connection to quantitative-genetic models.

The underlying notion in game-theoretic models is that evolution is a mutation-limited process. The population (or community) reaches demographic equilibrium while it contains only a single phenotype (per species), and then a new mutation arises and either replaces the former resident or dies out. If it replaces the resident, then a new population dynamic equilibrium is attained. At this stage, another mutation arises and the process repeats. As such, these models assume a separation of ecological and evolutionary timescales.

Research in adaptive dynamics provided an explicit model of this process (in continuous time), and it involves two important elements of stochasticity (Dieckmann & Law 1996, Proulx & Day 2001): (1) stochasticity in the mutations that arise and (2) stochasticity in whether or not these mutations reach fixation. The stochasticity in element 1 is proba-

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bly clear, and the stochasticity in element 2 is meant to reflect that, in real biological populations, even if the new mutant that arises is selectively advantageous, it might still be lost because of chance events when it is initially present in small numbers (Dieckmann & Law 1996).

To begin the derivation, imagine a very large number of independent populations in which this mutation-limited evolutionary process occurs. Each population can be viewed as following a series of successive "jumps" to new resident phenotypes. Each population has its own series of jumps, and populations differ in these patterns solely because of chance in which mutations arise and in whether or not they reach fixation. Let $p(z,t)$ be the frequency distribution of populations with a current resident value of z at time t. The average resident value at time t is therefore $\bar{z} = \int_{-\infty}^{\infty} zp(z,t)dz$, and the rate of change in $\bar{z}$ is given by the following:

$$\frac{d\bar{z}}{dt} = \int_{-\infty}^{\infty} z \frac{dp(z,t)}{dt} dz. \tag{13.32}$$

You now need to obtain a more explicit expression for the right-hand side of Equation 13.32.

The frequency of the collection of populations that has resident trait value z after a small amount of time, Δt, has passed will be given by what this frequency was initially, plus the frequency of all other types of populations that have moved into that state during this time interval, minus the frequency all populations in that state that have moved to other states in this time interval:

$$p(z,t+\Delta t) = p(z,t) + \int \Omega(z,\tilde{z})\Delta t p(\tilde{z},t)d\tilde{z} - \int \Omega(\tilde{z},z)\Delta t p(z,t)d\tilde{z}. \tag{13.33}$$

Here, $\Omega(z,\tilde{z})\Delta t$ is the probability that a population in state $\tilde{z}$ moves to state z in the time interval Δt. Rearranging, dividing by Δt, and taking the limit $\Delta t \to 0$ gives the following:

$$\lim_{\Delta t \to 0} \frac{p(z,t+\Delta t) - p(z,t)}{\Delta t} = \frac{\partial p}{\partial t} = \int \Omega(z,\tilde{z})p(\tilde{z},t)d\tilde{z} - \int \Omega(\tilde{z},z)p(z,t)d\tilde{z}. \tag{13.34}$$

Therefore, Equation 13.32 becomes as follows:

$$\begin{aligned} \frac{d\bar{z}}{dt} &= \int_z \int_{\tilde{z}} z\Omega(z,\tilde{z})p(\tilde{z},t)d\tilde{z}dz - \int_z \int_{\tilde{z}} z\Omega(\tilde{z},z)p(z,t)d\tilde{z}dz \\ &= \int_z \int_{\tilde{z}} (z-\tilde{z})\Omega(z,\tilde{z})p(\tilde{z},t)d\tilde{z}dz. \end{aligned} \tag{13.35}$$

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Now, if you assume that the frequency distribution, $p(z, t)$, is tightly centred around its mean, $\bar{z}$ (analogous to the assumption in going from Equation 13.19 to Equation 13.20 in quantitative-genetic models), then Equation 13.35 can be approximated as follows:

$$\frac{d\bar{z}}{dt} = \int_z (z - \bar{z})\Omega(z, \bar{z})dz. \tag{13.36}$$

To complete the derivation, you then need to be more explicit about the function $\Omega(z, \tilde{z})$. In particular, the probability that a population moves to state z from state $\tilde{z}$ in the time interval Δt is the product of the probability that a mutation occurs in that time interval (denoted by $\rho(\tilde{z})\Delta t$—this might depend on the current trait value, $\tilde{z}$), with the probability that this new mutation has trait value z (denoted by $M(z, \tilde{z})$—this might depend on the current trait value, $(\tilde{z})$ and the probability that this new mutation ultimately reaches fixation (denoted by $U(z, \tilde{z})$—this might depend on the resident trait value, $\tilde{z}$):

$$\Omega(z, \tilde{z})\Delta t = \rho(\tilde{z})\Delta t M(z, \tilde{z})U(z, \tilde{z}). \tag{13.37}$$

Now, because you are supposing that all population states are clustered tightly around the mean, $\bar{z}$, you must at least also assume that the allowable mutational jumps are not very large. In this case, the probability density $M(z, \tilde{z})$ must be narrowly clustered around its mean; therefore, you can approximate Equation 13.37 using the first two terms of a Taylor series in z near $\bar{z}$:

$$\Omega(z, \tilde{z})\Delta t \approx \rho(\bar{z})\Delta t M(z, \bar{z})U(\bar{z}, \bar{z}) + \rho(\bar{z})\Delta t M(z, \bar{z})\left.\frac{\partial U}{\partial z}\right|_{z=\tilde{z}=\bar{z}} (z - \bar{z}). \tag{13.38}$$

Substituting this into Equation 13.36 gives the final result (Proulx & Day 2001):

$$\frac{d\bar{z}}{dt} = \rho(\bar{z})\mu(\bar{z})U(\bar{z}, \bar{z}) + \rho(\bar{z})\sigma^2(\bar{z})\left.\frac{\partial U}{\partial z}\right|_{z=\tilde{z}=\bar{z}}. \tag{13.39}$$

Here, $\mu(\bar{z})$and $\sigma^2(\bar{z})$ are the mean and variance in the mutational distribution, and $M(z, \bar{z})$. Equation 13.39 reveals that the evolutionary change in $\bar{z}$ is the result of two forces: any mutational bias (the first term) and selection (the second term). In the absence of mutational bias you

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have $\mu(\bar{z}) = 0$, and Equation 13.39 simplifies to the following (Dieckmann & Law 1996, Proulx & Day 2001):

$$\frac{d\bar{z}}{dt} = \rho(\bar{z})\sigma^2(\bar{z})\left.\frac{\partial U}{\partial z}\right|_{z=\tilde{z}=\bar{z}}. \tag{13.40}$$

Notice that Equation 13.40 is analogous to Eshel's equation (Equation 13.31) and to the approximated quantitative-genetic equation (Equation 13.20), where the probability of fixation, $U(z, \bar{z})$, plays the role of the fitness function.

You can obtain even closer correspondence between these modelling approaches if you assume a particular model for the way in which stochasticity affects the probability of fixation. It can be shown that, under a stochastic model based on branching processes (and therefore one in which selectively disadvantageous mutants *never* reach fixation; see Proulx and Day 2001), you have the relationship $U(z, \bar{z}) = (b(z, \bar{z}) - d(z, \bar{z}))/b(z, \bar{z})$, where $b(z, \bar{z})$ and $d(z, \bar{z})$ are the expected birth and death rates of the mutant with trait z in a population with resident trait, $\bar{z}$ (Dieckmann & Law 1996). Using this relationship in Equation 13.40 gives the following:

$$\frac{d\bar{z}}{dt} = V(\bar{z})\left.\frac{\partial \lambda}{\partial z}\right|_{z=\tilde{z}=\bar{z}}. \tag{13.41}$$

Here, $V(\bar{z}) = \rho(\bar{z})\sigma^2(\bar{z})/b(\bar{z}, \bar{z})$ is a measure of the rate at which genetic variation is introduced into the population through mutation, and $\lambda(z, \bar{z}) = b(z, \bar{z}) - d(z, \bar{z})$ is the per capita growth rate of the mutant (i.e., its fitness) (Dieckmann & Law 1996). This is identical in form to the quantitative-genetic equation (Equation 13.20), as well as to Eshel's (1983) equation (Equation 13.31), and thus provides an explicit mathematical justification for the evolutionary dynamic used by Eshel in distinguishing evolutionary stability from convergence stability. Notice, however, that unlike the quantitative-genetic models, there is no corresponding equation for the population dynamics because these are assumed to occur on a timescale much faster than evolutionary change. Therefore, the population is assumed to always be in population-dynamic equilibrium.

There have been other developments and elaborations on the adaptive-dynamic approach that take into account multiple species, finite-population sizes, environmental stochasticity, and nonequilibrium attractors (e.g., limit cycles and chaos) for the ecological dynamics, to name just a few (Metz, Nisbet, & Geritz 1992; Ferriere & Fox 1995).

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These results have broadened the scope of applicability of this approach (but see Proulx & Day 2001 for a description of some limitations), but I believe it is fair to say that all of these developments are well within the normal scientific development of game theory and do not constitute a fundamentally different approach to theoretical evolutionary ecology. I would even question the need for using a separate (and potentially confusing) new label for these developments. Nevertheless, the adaptive-dynamic approach represents the latest development in theoretical evolutionary ecology, and its results have contributed important and interesting insights to this field.

13.5 | WHERE DO WE STAND? WHERE DO WE GO? IS ANYTHING NEW?

13.5.1 | Future Empirical Directions

The existence of phenotypic values that are evolutionary attractors yet give rise to disruptive selection is one of the most interesting findings to come out of theoretical evolutionary ecology. There are still few explicit tests of such predictions, but numerous opportunities exist for exploring these issues empirically. Some steps have been taken in this direction, with perhaps the most direct attempt being a study involving artificial selection in *Drosophila* (Bolnick 2001; reviewed in Day & Young 2004). Daniel Bolnick (2001) did not, however, address the critical prediction of the occurrence of evolutionary diversification (Day & Young 2004).

Interestingly, there have been several experiments carried out for reasons unrelated to this theory that nevertheless provide some of the most relevant data for testing the predictions about such branching points (Travisano & Rainey 2000, Rainey *et al*. 2000, Kassen 2002). Most of these have been conducted using microbial cultures such as *Pseudomonas* or *Escherichia coli*. Such model organisms are ideal for testing this theory because their rapid generation times and well-defined genetic stocks make appropriate evolutionary experiments feasible. Also, because such organisms are asexual, they represent the most conducive systems for finding evolutionary branching; unlike as occurs in quantitative-genetic models, there is no sexual recombination to hinder evolutionary diversification.

Several experiments have been conducted in which a single microbial clone is propagated for several generations in some well-defined resource medium. Although these experiments were not designed to look for branching points, the theory based on exploitative competition

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outlined earlier predicts that researchers should initially observe an evolutionary adaptation to the highest carrying capacity. At this stage, under some conditions, evolutionary diversification should occur.

Several experiments display this sort of evolutionary diversification. For example, in single strains of *E. coli* propagated in a glucose medium, evolutionary diversification eventually took place, resulting in the stable maintenance of two distinct physiological types (Turner, Souza, & Lenski 1996; Travisano & Rainey 2000). Similarly, in colonies of a single strain of *Pseudomonas* propagated in a complex liquid medium, evolutionary diversification eventually took place, resulting in three well-defined types that appear to coexist indefinitely (the "fuzzy spreader," "wrinkly spreader," and "smooth" types; see Rainey & Travisano 1998). These morphs appear to exploit different spatial niches in the liquid medium. Perhaps even more remarkably, these patterns of evolutionary diversification appear to be highly repeatable between experiments.

These results are extremely exciting, and it has been noted that these experiments have inadvertently provided empirical data consistent with these recent theoretical predictions (Travisano & Rainey 2000). Diversification occurred as expected in accord with theory. It still remains, however, to determine if this sort of phenomenon is relevant in organisms other than microbes. There is clearly increasing interest in this issue (Bolnick *et al*. 2003), and there are several documented examples of disruptive natural selection in the wild, but whether these examples are best explained by the sort of endogenously generated selective pressures predicted by the theory remains an open question deserving further study. A preliminary survey of these examples indicates that most do not fit well within this theoretical explanation (Day and Abrams, unpublished results), but more rigorous examinations (and experiments) are required to reach any definitive conclusion.

13.5.2 | Future Theoretical Directions

The preceding microbial examples clearly show that evolutionary diversification in experimental systems occurs; however, it is important to ask whether the ecological interactions embodied in the theory are likely to be the cause of this diversification. Most theory has focused on evolutionary diversification as a result of competition for resources, but is this the primary reason for the diversification seen in microbial systems?

Importantly, facilitation has been well documented in many of the aforementioned microbial experiments. Facilitation is an ecological interaction in which the presence of one species enhances the fitness of another (Whittaker 1977; Bruno, Stachowicz, & Bertness 2003). For

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example, in the experiments in which *E. coli* diversified during propagation in glucose, it has been demonstrated that the new variants that arise are specialized on acetate, a metabolite produced by the consumption of glucose by the original strain. This is often referred to as "cross-feeding" in the literature, and it demonstrates that evolutionary diversification in this case occurred primarily as a result of the first species having a facilitative effect on the second through its introduction of additional resources into the environment (Turner, Souza, & Lenski 1996). Similar facilitative interactions are likely important in the *Pseudomonas* system. For example, it has been demonstrated that the "fuzzy spreader" type cannot invade a population of the "wrinkly spreader" type without the third, "smooth" type also being present (Figure 4 in Travisano & Rainey 2000).

These findings contrast with competitive diversification in which the different consumer types do not introduce new resources; rather, they affect the relative value of specializing on the various resources already present. Under facilitation, the addition of new species creates new niches (Levins & Lewontin 1985) and thereby represents a fundamentally different type of ecological interaction that likely plays an important role in evolutionary diversification. To better understand the relative roles of competition and facilitation in diversification, researchers require a clear approach for distinguishing between the two. Currently, there is little theory addressing facilitative diversification in evolutionary ecology (e.g., see Doebeli 2002), but it is likely that the two interactions can be distinguished using relatively simple ecological experiments (Day & Young 2004). Nevertheless, further theoretical results in this area would be invaluable for better dissecting the causes of evolutionary diversification.

One other area requiring further theoretical development is a better characterization of the potential evolutionary outcomes at so-called branching points. Evolutionary splitting is one possibility (even for sexual populations; Dieckmann & Doebeli 1999), but it is not the only outcome or even necessarily a likely one. Other possibilities include a simple increase in genetic variance, the evolution of a within-species polymorphism among age or size classes (Taylor & Day 1997; Day, unpublished results), and the evolution of within-species sexual dimorphism (Bolnick & Doebeli 2003). All of these outcomes effectively fill the available niche space, but the most likely end point of evolution will depend on the specifics of within-species interactions, relative to between-species interactions, coupled with the particular genetic constraints imposed by the system of inheritance for the organism in question.

As seen in the description of the various approaches presented earlier, one chief difference between quantitative-genetic and single-locus

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models versus game-theoretic and adaptive-dynamic models is that the latter assume a separation of ecological and evolutionary timescales. Ironically, these recent approaches in theoretical evolutionary ecology have returned to using an assumption whose dubious validity was part of the motivation for the development of theoretical evolutionary ecology. An important question remains: What does this separation of timescales do in terms of predictions? For example, are the evolutionary consequences of branching points different if a researcher allows evolutionary change to proceed on a timescale comparable with ecological change? A powerful way to explore this question is through the use of the quantitative-genetic approximation in Equation 13.20 because no restriction on relative timescales is made in its derivation, and it is directly comparable to the evolutionary dynamics for game-theoretic and adaptive-dynamic models (i.e., Equations 13.31 and 13.41, respectively). This is an interesting area deserving further attention.

Finally, recent studies based in adaptive dynamics have demonstrated that sympatric speciation can occur seemingly easily in models of competitive diversification (like that presented earlier) by allowing assortative mating to evolve simultaneously (Dieckmann & Doebeli 1999). To some extent these results contradict earlier suggestions that sympatric speciation is unlikely as a result of tension caused by the buildup of linkage disequilibrium between alleles coding for ecological traits and alleles at other loci coding for mate preferences (Felsenstein 1981). It seems as though these recent models of sympatric speciation should suffer from the same tension because the chief difference from earlier theory on speciation is the inclusion of a mechanism by which the population is intrinsically maintained under disruptive selection (i.e., at a branching point) rather than having disruptive selection imposed on it. Presumably this should have no effect on the extent to which assortative mating (and the requisite linkage disequilibrium) can evolve, so it remains unclear why sympatric speciation appears to occur more readily in this recent theory. Further research examining the relationship between these results and those of earlier theory would help us understand where this difference comes from.

13.5.3 | Conclusions: Déjà Vu or Something New?

As will be clear by now, it is my opinion that the most recent techniques and approaches in theoretical evolutionary ecology do not represent a fundamental change in the way scientists are thinking about and modelling ecological–evolutionary feedbacks. Rather than being a paradigm shift, I believe it represents "normal" science. What it truly fascinating, however, is that the work on single-locus models, quantitative-genetic

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models, game-theoretic models, and adaptive-dynamic models has proceeded largely independent of one another, but these have led to what is fundamentally the same mathematical description of evolutionary change (compare the progression of results from the various modelling approaches: Equations 13.13B, 13.18B, 13.19, 13.20, 13.31, and 13.41). This suggests that there is something fundamental and robust being described by the different approaches because they have all converged on similar answers from different starting points. Indeed, the main differences in these approaches stem from their difference in the assumption of a separation of timescales. Game-theoretic or adaptive-dynamic models make such an assumption, whereas single-locus and quantitative-genetic models do not (and therefore have additional dynamic equations governing the ecological dynamics in conjunction with the evolutionary dynamics). The ongoing union of ecology and evolutionary biology is proving to be a fertile enterprise, and the most important advances in the near future will likely continue to be refinements, developments, and extensions of the important groundwork laid over the past half-century.

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