doi:10.1111/j.1420-9101.2005.00948.x

TARGET REVIEW

20 Questions on Adaptive Dynamics

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Keywords:

Adaptive Dynamics;
assortative mating;
evolution;
population genetics;
speciation;
theory.

Abstract

Adaptive Dynamics is an approach to studying evolutionary change when fitness is density or frequency dependent. Modern papers identifying themselves as using this approach first appeared in the 1990s, and have greatly increased up to the present. However, because of the rather technical nature of many of the papers, the approach is not widely known or understood by evolutionary biologists. In this review we aim to remedy this situation by outlining the methodology and then examining its strengths and weaknesses. We carry this out by posing and answering 20 key questions on Adaptive Dynamics. We conclude that Adaptive Dynamics provides a set of useful approximations for studying various evolutionary questions. However, as with any approximate method, conclusions based on Adaptive Dynamics are valid only under some restrictions that we discuss.

1. Introduction

Over the past 10 years or so, a large number of papers employ a set of techniques and methods that the authors call 'Adaptive Dynamics'. These techniques are used to approach a number of interesting and important issues in evolution and related subjects. These include investigations into the maintenance of genetic variation, coevolution and sympatric speciation, i.e. subjects that both puzzle and intrigue practising biologists. However, the technical or mathematical nature of most papers on Adaptive Dynamics makes it very difficult for a typical biologist to understand the essence, usefulness or limitations of this approach. At the outset, we considered writing a review of the subject in a traditional format. We are, however, talking about a subject that is still in a rapid stage of development and which has not yet achieved a mature form. Thus although a review might be appropriate for a mature subject, it seemed more appropriate to us to frame a set of questions that someone newly encountering the subject would like to ask – and have answered – in plain language.

It should be noted that the authors of this review do not come from the core of individuals that are recognized as practitioners of Adaptive Dynamics, but rather, from the much larger group of outsiders, including population geneticists, who are interested in this recent development in theoretical population biology. Indeed, when, below, we describe Adaptive Dynamics, we do so from the perspective of population geneticists, rather than following the interpretations and emphases that have been adopted by practitioners of Adaptive Dynamics.

This paper is arranged as a main body of 20 key questions. Despite the often highly theoretical/technical content of papers in this area, we have endeavoured to answer the questions with an absolute minimum of mathematical formalism.

Much of the exposition presented here closely follows that of Geritz *et al.* (1998) and this paper is a good starting point for readers interested in a more technical introduction to the subject.

We begin with a description of the methods and concepts of Adaptive Dynamics. After establishing the basics of Adaptive Dynamics, we discuss connections with previous work.

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2. General description

2.1 What is Adaptive Dynamics all about?

Adaptive Dynamics is a theoretical approach for studying some of the phenotypic changes that take place, over time, in evolving populations.

It grew out of early work of game theorists (e.g. Eshel & Motro, 1981; Eshel, 1983; Taylor, 1989; Hofbauer & Sigmund, 1990; Nowak & Sigmund, 1990), population geneticists (e.g. Christiansen, 1991; Abrams *et al.*, 1993a,b) and theoretical ecologists (e.g. Schaffer, 1977; Reed & Stenseth, 1984; Metz *et al.*, 1992) seeking simple tools to study evolutionary change when fitnesses are density or frequency dependent. It is based on the assumption that mutations occur rarely, and cause very small changes in existing phenotypic values.

In most applications, organisms are assumed to be asexual and the initial population – termed the resident population – consists of phenotypically identical individuals, i.e. is monomorphic. Most calculations are performed in terms of a specific fitness function, which is termed the invasion fitness.

To the best of our knowledge, the term 'Adaptive Dynamics' was first introduced in the papers of Hofbauer & Sigmund (1990) and Nowak & Sigmund (1990).

2.2 How are fitness functions derived in Adaptive Dynamics?

The most basic models in evolutionary population genetics involve fitness functions that depend only on the phenotypic trait values under selection. In reality, fitness depends on far more than just trait values including (i) the frequencies of individuals with different trait values; i.e. fitness is frequency dependent, (ii) the density of the population, as measured, e.g. by the number of individuals/unit area; i.e. fitness is density dependent, and (iii) the absolute number of individuals in the population. Investigations involving such quantities are typically the domain of ecology, where absolute numbers or densities are the focus of attention. One of the really interesting features of Adaptive Dynamics is that its practitioners have attempted to provide a framework where interactions and fitnesses originating from explicitly ecological considerations are incorporated into population genetic models of evolutionary dynamics. Thus, for example, some of the Adaptive Dynamics literature, explicitly refers to quantities such as fitness – a primarily population genetics concept – while also directly employing a model of ecological dynamics, for example that of Lotka Volterra.

2.3 What is invasion fitness?

Much of the work on Adaptive Dynamics has centred around a concept known as 'invasion fitness'. To understand invasion fitness, it is easiest to think of a population of asexual organisms that have discrete generations and are characterized by a single, continuously varying, phenotypic trait. We assume this population – the resident population – initially consists of individuals that all possess the same phenotypic value x (i.e. is monomorphic). Mutations that differ from the monomorphic resident population are randomly and recurrently generated, and these can be thought of as attempting to 'invade' the initial population. The fitness function of very rare mutations of phenotype y, in an (almost monomorphic) resident population, with phenotype x, is just a function of x and y and it is and is usually written as $s(y,x)$ (or $s_x(y)$). This function is referred to as the *invasion fitness* and the precise form of $s(y,x)$ depends on the specific biological situation under consideration. The invasion fitness governs the dynamics of the frequency of the mutants, while they are at low frequency. As the resident population is stable over the long term in the absence of mutation, the residents have a fitness of unity, i.e. $s(x,x) = 1$. Neglecting stochasticity associated with genetic drift (which is discussed in the answer to Question 4.3), mutant types will usually initially exhibit near exponential growth or decay in their frequency, depending on whether $s(y,x) > 1$ or $s(y,x) < 1$. Invasion fitness can be (and sometimes is) defined for a polymorphic population of residents (e.g. see Question 2.9).

2.4 What is an invasion fitness landscape?

The problems that density and frequency-dependent fitnesses create for predicting evolutionary change can be envisaged by thinking about fitness landscapes. The notion of fitness (or adaptive) landscapes was introduced by Wright (1932, 1988) and has proved to be extremely useful in evolutionary biology (e.g. Provine, 1986; Gavrilets, 1997, 2004; Fear & Price, 1998; Arnold *et al.*, 2001). In the case of continuously varying traits, a fitness landscape is often visualized as a three-dimensional plot containing a surface of individual fitness that lies above a horizontal plane. The coordinate axes of the plane correspond to the values of *two* phenotypic characters. Although we believe that there are usually more than two characters affecting fitness, fitness is, in such plots, represented as a function of only two phenotypic characters because we cannot plot a graph with more than three axes.

As long as fitness depends only on the trait values of an individual, the fitness landscape remains rigid and unchanging. However, if fitness is density or frequency dependent, then instead of remaining fixed, the fitness landscape heaves and bulges as the population moves over it.

There is, however, something intuitively appealing and useful about visualizing fitnesses in terms of a *fixed* landscape. We therefore introduce the notion of an 'invasion fitness landscape' which, despite sounding similar to a traditional fitness landscape, is fundamentally different. An 'invasion fitness landscape' is a three-dimensional plot where invasion fitness, $s(y,x)$, is plotted as a surface above a horizontal plane. In this case, one coordinate axis of the plane is determined by possible values of the phenotypic character of the resident population, whereas the other axis is determined by the

values *of the same character*, in mutation bearing individuals. Notice that our definition implies that the invasion fitness landscape does not change shape as evolutionary change occurs – even when fitnesses are density or frequency dependent. Invasion fitness landscapes also differ from traditional fitness landscapes in the way the population moves over the landscape during evolutionary change. Instead of moving uphill, the population tracks a path among the hills of the landscape, along a line where fitness has the value of unity, as the answer to the next question shows.

2.5 How does the population move on the invasion fitness landscape?

To answer this question we make the assumption that the vast majority of mutant phenotypes are very close to the phenotype of the monomorphic resident population; this is the standard assumption that most mutations are of small effect. In a population of large size, where deleterious mutations have a negligible chance of fixation, only mutants that have higher fitness than the residents will invade.

Figure 1 illustrates some of the processes associated with adaptive evolution on the invasion fitness landscape. An adaptive mutation occurs to a member of the resident population, whose phenotypic value is represented by the x coordinate of the point p. The mutation is represented by a dashed line, from p to q, that is parallel to the y axis and corresponds to the instantaneous phenotypic change associated with the mutation. In order to be successful, a mutant must have greater fitness than that of a mutant, so successful mutational change is always in an 'uphill' direction. Fixation results in the mutant phenotype (y) becoming the new resident phenotype (x), and is represented by the solid line that is parallel to the x axis and runs from q to r. This line shows the resident phenotype, x, being reset to equal the mutant phenotype, y (the thin line, on which p, r and t lie, is the line $y = x$). The move is effectively in a 'downhill' direction, so that the point r has the same value of $s(y,x)$, namely unity, as the starting point, p. The movement downhill does not imply that the population experiences any fitness loss but rather reflects the resetting of fitnesses so that new resident population has a fitness of unity. The dashed line from r to s represents the change caused by another adaptive mutation (again uphill) and the line from s to t represents fixation of the mutant (again downhill). Over the course of time the population can be thought of as performing many such jagged steps (of the form $p \rightarrow q \rightarrow r$) and if the mutational effects are very small, as assumed, then the population closely traces out a trajectory along the diagonal line $y = x$. The overall effect is that the population does not ascend the invasion fitness landscape over time.

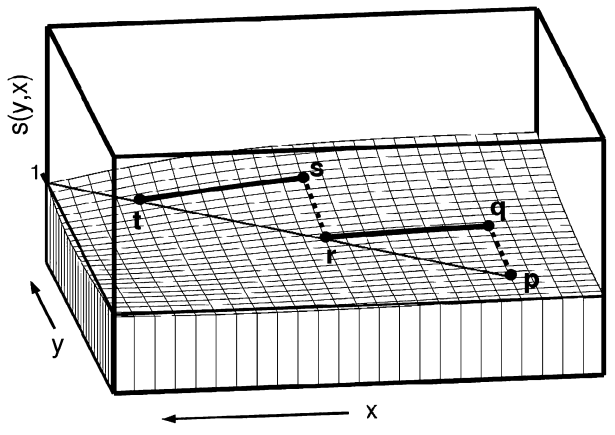

Fig. 1 Evolution on an invasion fitness landscape. For a description of the dynamics illustrated in this figure, see the answer to Question 2.5.

In Figs 2a and 3a the population's trajectory, as a result of many tiny mutational steps, followed by resetting of the resident phenotype to that of the mutant, is well approximated by the smooth line from point p to close to point q. This line remains horizontal. Figures 2 and 3 will be repeatedly used, below, to illustrate various issues. The picture, just given, of the dynamics of the population applies, as long as there is a well defined 'uphill' direction for the mutants to take. We discuss next, a measure of the steepness or slope of the invasion fitness landscape so that we can recognize and deal with the important case where this is not so and the slope vanishes.

2.6 What is the local fitness gradient?

The fitness gradient, $D(x)$, is a measure of the steepness of the invasion fitness landscape as experienced by a small-effect mutation, when the resident population has phenotype x. Mathematically, $D(x)$ is the slope along the y direction: $D(x) = [\partial s(y,x)/\partial y]_{y=x}$. If $D(x)$ is positive (as it is in Figs 2a and 3a, in the vicinity of the point p) then mutants with larger trait values than that of the residents will invade. Conversely, if $D(x)$ is negative, mutants with smaller trait values will invade. Such gradient-type dynamics are analogous to those studied in standard population genetics (e.g. Wright, 1935; Lande, 1976; Barton & Turelli, 1987).

If initially successful mutants completely supplant the residents then the local fitness gradient determines not just which mutants are adaptive, but also the *direction of change* of phenotypes, via substitution. However, although initial invasion occurs when the frequency of mutants is small and the resident population monomorphic, during the time when substitution occurs the mutant frequencies do not remain small and the population is far from a monomorphic state. In recent work, Mylius & Diekmann (2001) questioned the assumption that initially successful mutants completely supplant the residents, whereas Geritz *et al.* (2002) explored its validity. However, the

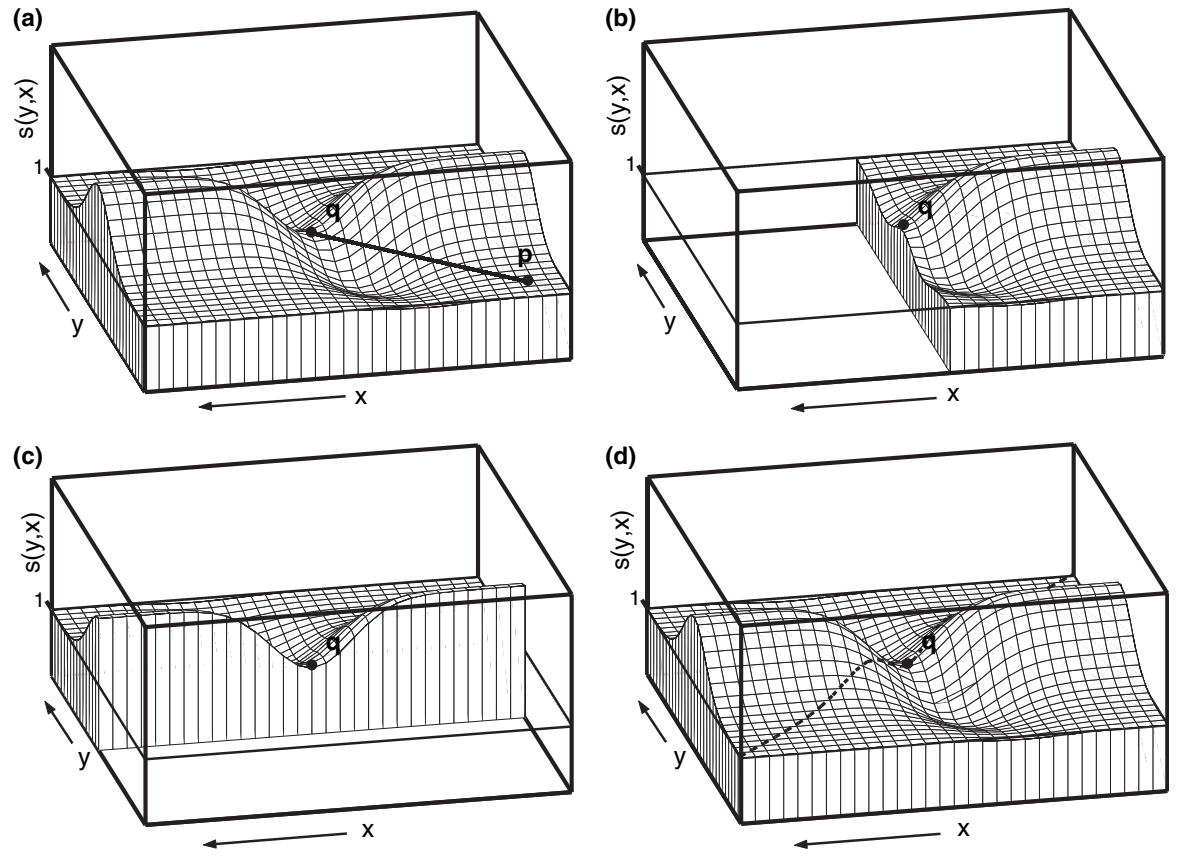

Fig. 2 (a) An example of an invasion fitness landscape and illustration of the trajectory of a population. The many jagged steps that make up the recurrent processes of mutation, followed by resetting the residents to the mutant phenotypic value, are approximated by the trajectory along the solid line from p to q. (b, c) Only sections of the invasion fitness surface have been plotted to illustrate the behaviour of this surface. (d) The dashed line corresponds to the invasion fitness along the line $y - x^* = -(x - x^*)$. It is apparent that the point q corresponds to a local maximum along the y direction (b), to a local minimum along the x direction (c), and a local minimum along the line $y - x^* = -(x - x^*)$ (d).

set of conditions required for fixation of initially successful mutants remains an open question.

Typically, the local fitness gradient changes with phenotype, x. Under some circumstances, a sequence of successful substitutions results in the magnitude of the local fitness gradient, $|D(x)|$, becoming progressively smaller. An example of this is illustrated in Figs 2a and 3a, where the population traces a trajectory, from p to q. At point q in both figures, the local fitness gradient vanishes: $D(0) = 0$. Figures 2 and 3 show some subtly different features in the vicinity of the point q. These features turn out to be highly significant for the subsequent dynamics of the population, once it has approached close to point q. To deal further with this requires some additional analysis: see the answers to Questions 2.7 and 2.8.

2.7 What are pairwise invasibility plots?

Not all the information in the invasion fitness landscape is needed to predict the outcome of invasion in a monomorphic population. The necessary information can be summarized in a 'pairwise invasibility plot' (Christiansen & Loeschcke, 1980; Matsuda, 1985; van Tienderen & de Jong, 1986). This is a horizontal slice of the invasion fitness landscape, that is taken at the level where $s(y,x)$ equals 1. Parts of the slice that pass under the solid hillside are indicated by '+' signs along the region where the slice first enters the hillside. Parts of the slice that pass into the 'thin air' above the hillside are indicated by '−' signs where the 'thin air' above the hillside first begins. (In some publications this convention is replaced by dark shading in the parts of the slice passing through the hillside.)

Figure 4 shows the pairwise invisibility plot taken from the invasion fitness landscape in Fig. 1. Along the line $y = x$, where mutants are phenotypically identical to residents, there is no fitness difference of mutants and residents. It may be the case that the fitness difference $s(y,x)-s(x,x)$ is equal to zero for other combinations of y and x other than just $y=x$.

In Fig. 5, it is assumed that in addition to the diagonal line $y = x$, where $s(y,x) - s(x,x) = 0$, there is another line

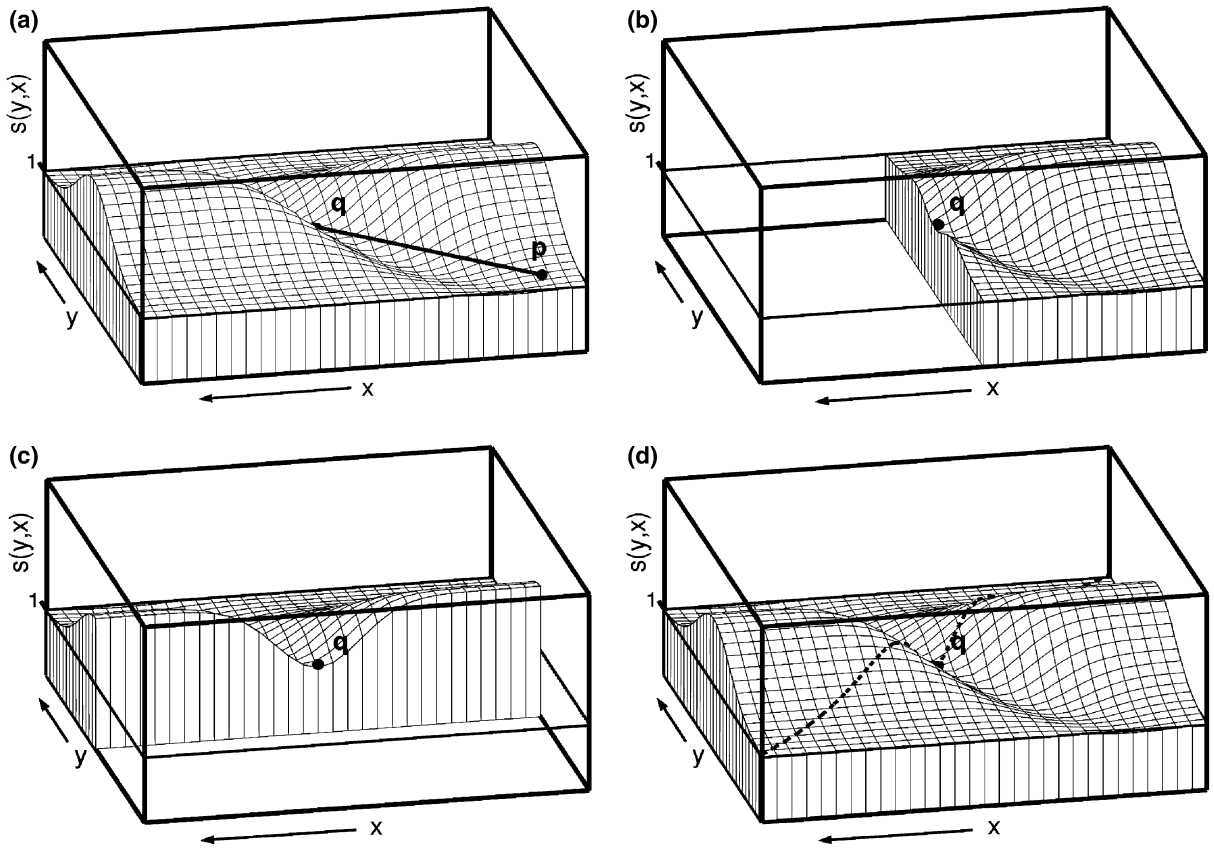

Fig. 3 (a) The trajectory of a population in a different invasion fitness landscape to that of Fig. 2a. The trajectory of the population is along the solid line p to q. (b, c) Only sections of the invasion fitness surface have been plotted to illustrate the behaviour of this surface. (d) The dashed line corresponds to the invasion fitness along the line $y - x^* = -(x - x^*)$. It is apparent that the point q corresponds to a local minimum along the y direction (b), to a local minimum along the x direction (3c), and a local minimum along the line $y - x^* = -(x - x^*)$ (d).

where $s(y,x) - s(x,x) = 0$. This additional line is determined by the detailed properties of the invasion fitness and, as there is no general mathematical reason why it should be straight, it will typically be curved. This line intersects the diagonal line, $y = x$, at a resident phenotypic value denoted x^*. As $D(x)$ changes sign as x passes through x^*, $D(x)$ is zero at $x = x^*$.

Phenotypic trait values where the fitness gradient vanishes are given very special significance in Adaptive Dynamics and such a value is called an 'Evolutionarily Singular Strategy' or a 'Singular Point'. Many people would call such a point an 'equilibrium point', irrespective of whether it is stable or not. For the case depicted in the pairwise invasibility plot of Fig. 5, the population evolves (by successively fixations of beneficial mutations), until it reaches a neighbourhood where $D(x)$ is zero – the neighbourhood of an Evolutionarily Singular Strategy. The approach to the same Evolutionarily Singular Strategy is indicated by the line from p to q in Fig. 2a, which shows the invasion fitness landscape corresponding to the pairwise invasibility plot given in Fig. 5. The Evolutionarily Singular Strategy in Fig. 2a is the point q.

In general, an Evolutionarily Singular Strategy is only a single point on an invasion fitness landscape. *A priori*, it might be considered an improbable phenotype that is of little relevance to the dynamics of a population. However, considerations, such as those given above, make it clear that the population may be driven to such a point by its intrinsic substitutional dynamics.

2.8 What Evolutionarily Singular Strategies are possible?

In general, a population will not start off at a singular strategy (a point with $D(x) = 0$) and the dynamical significance of any such singular strategy (phenotype) has to be determined by its stability properties.

An analysis of invasion fitness, close to a singular strategy, indicates that ordinarily, there are only a limited number of different types of Evolutionarily Singular Strategies. Furthermore, each singular point belongs to only one of these types and each of these has a unique form of pairwise invasibility plot. Mathematically, as the fitness gradient and, it can be shown, the derivative

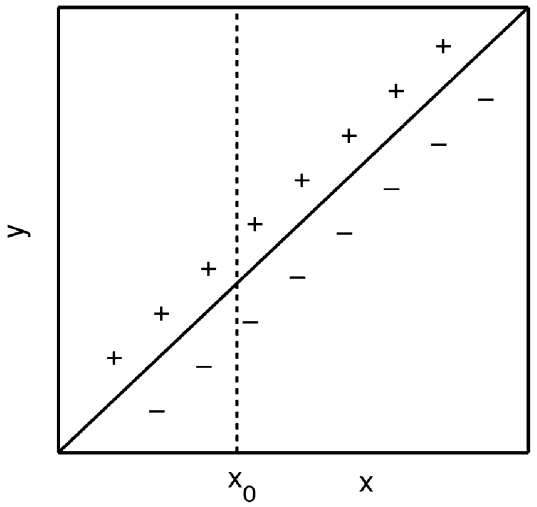

Fig. 4 A very simple pairwise invasibility plot, where mutant phenotypes are denoted by y and resident phenotypes by x. Only in regions marked with '+' signs do mutants have a fitness that is larger than that of the residents and therefore can invade. In the regions with '−' signs, the residents have higher fitness than any mutants and invasion is not possible. The differences between mutant and resident phenotypes, $y - x$, are assumed small, so all attention can be restricted to a narrow band along the diagonal line $y = x$. To use a pairwise invasibility plot to determine which mutants will invade (i.e. increase in frequency), when initially rare, given a resident population with specific phenotype, say x_0, one simply looks along a vertical line passing through the specific resident phenotypic value, at $x = x_0$, on the x axis. The mutant phenotypes along this vertical line that are above the diagonal line, $y = x$, have $y > x$, whereas those below the diagonal line have $y < x$. Only those mutant phenotypes lying in a '+' region can invade. This figure shows a case where only mutants with $y > x_0$ can invade.

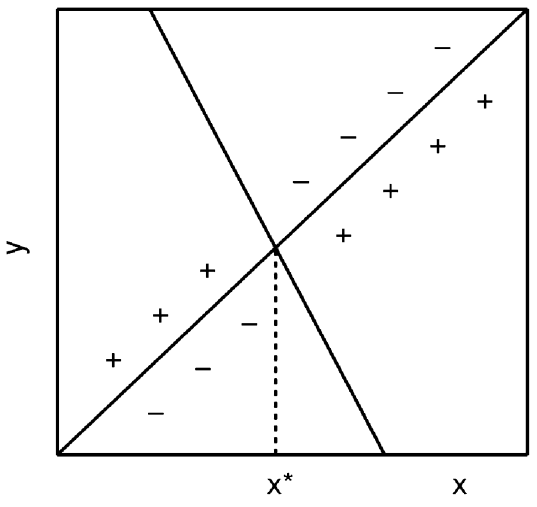

Fig. 5 A pairwise invasibility plot, where mutant phenotypes are denoted by y and resident phenotypes by x. The diagonal corresponds to the line $y = x$. The point of intersection of the two solid lines, corresponds to $x = x^*$ and at this point there is a vanishing fitness gradient: $D(x^*) = 0$. The point, x^* is termed an 'Evolutionarily Singular Strategy'.

$\partial s(y,x)/\partial x$, both vanish at a singular strategy, i.e. when $x = x^*$ and $y = x^*$, an Evolutionarily Singular Strategy is a *stationary point* of $s(y,x)$. It is thus intuitively reasonable that the first nonvanishing derivatives – generically second-order derivatives – are the means of telling different types of singular strategy apart. It is not immediately obvious, and it requires some detailed reasoning to infer that, to quadratic deviations from a particular Evolutionarily Singular Strategy, x^*, we have, with A and B constants (Metz *et al.*, 1996).

$$s(y,x) \simeq 1 + \frac{A}{2}(x-x^*)^2 - \frac{A+B}{2}(x-x^*)(y-x^*) + \frac{B}{2}(y-x^*)^2. \quad (1)$$

The classification of the Evolutionarily Singular Strategy can thus be *completely achieved* from knowledge of just two second-order derivatives, namely

$$A = \left[\frac{\partial^2 s(y,x)}{\partial x^2}\right]_{x=x^*,y=x^*}, \qquad B = \left[\frac{\partial^2 s(y,x)}{\partial y^2}\right]_{x=x^*,y=x^*} \quad (2)$$

which are evaluated at the singular strategy and which, generally, do not vanish.

Let us introduce the convenient mathematical notation sign(x) to represent a step function that has the value −1 (+1) when x is negative (positive):

$$\text{sign}(x) = \begin{cases} +1, & \text{when } x > 0, \\ -1, & \text{when } x < 0. \end{cases}$$

We then define

$$a = \text{sign}(A), \quad b = \text{sign}(B), \quad c = \text{sign}(|A| - |B|). \quad (3)$$

The coefficient a tells us whether the Evolutionarily Singular Point sits at the bottom of a valley ($a = 1$) or top of a hill ($a = -1$) when viewed by cutting the invasion fitness landscape parallel to the x axis. The coefficient b tells us the same thing when the Evolutionarily Singular Point is viewed by cutting the invasion fitness landscape parallel with the y axis. The coefficient c tells us whether the curvature of the invasion fitness landscape at the Evolutionarily Singular Point is larger along the x ($c = 1$) or y ($c = -1$) direction. It turns out that the different types of Evolutionarily Singular Strategy can be characterized by the triplet of numbers (a,b,c). One virtue of this labelling scheme is that we can easily count the number of different singular strategies. As a, b and c can each independently take on two different values (namely ±1), it follows that there are $2 \times 2 \times 2 = 8$ different types of Evolutionarily Singular Strategy.

The important properties of an Evolutionarily Singular Strategy, that hold in a very small range of phenotypes around the strategy, are as follows (Eshel, 1983; Taylor, 1989; Christiansen, 1991; Geritz *et al.*, 1998).

(i) An Evolutionarily Singular Strategy, x^*, is *not invasible*, if a resident population consisting solely of x^*

phenotype individuals cannot be invaded by any nearby mutant. This is the case if $s(x^*,x^*) > s(y,x^*)$ for all y close to x^*. Geometrically, this implies that at the singular point the invasion fitness landscape $s(y,x)$ has a local maximum with respect to y. Mathematically, this corresponds to $B < 0$. Conversely, if $B > 0$, then x^* is not locally stable, and it can be invaded by any nearby mutant. Note that at the singular point q of Fig. 2b, the invasion fitness landscape has a local maximum with respect to y ($B < 0$), whereas in Fig. 3b, $s(y,x)$ has a local minimum ($B > 0$) at this point. Thus, in Fig. 2, the singular point q is not invasible, whereas in Fig. 3, the singular point q is invasible.

(ii) Another important property of an Evolutionarily Singular Strategy is the *ability of rare mutants*, with *phenotype* x^*, *to invade* a population whose phenotype differs from x^*. This is the case if $s(x^*,x) > s(x,x)$ for all x close to x^*. Geometrically, this implies that at the singular point the invasion fitness landscape $s(y,x)$ has a local minimum with respect to x. Mathematically, this corresponds to $A > 0$. Reversal of the inequality indicates the *inability* of rare mutants with phenotype x^*, to invade a population of different phenotype. Note, in both Figs 2c and 3c, that at the singular point q, the invasion fitness landscape has a local minimum with respect to x ($A > 0$) and thus the singular strategies, depicted in these figures, are able to invade another nearby strategy, when rare.

(iii) An Evolutionarily Singular Strategy, x^*, possesses the property of being the stable end-point of a sequence of successive successful substitutions – i.e. possesses *convergence stability* – if a resident population's phenotype, x, can only be invaded by mutants with a phenotype closer to x^* than x is itself. This is the case if the fitness gradient, $D(x)$, is positive for $x < x^*$ and negative for $x > x^*$. This implies that at the singular point the fitness gradient $D(x)$ is a *decreasing* function of x. Expressed mathematically, this corresponds to $A - B > 0$ and if the inequality is reversed, then successive substitutions will move the population's phenotype progressively further away from x^*. Figures 2a and 3a exhibit the property that along the trajectory of the population (the line from p to q), the slope of the invasion fitness surface in the y direction, i.e. the fitness gradient, decreases with x. Thus, the point q in both of these figures is convergence stable.

(iv) Lastly there is the possibility of the existence of a *protected polymorphism*. A protected polymorphism arises if two strategies, say y_1 and y_2, making up the polymorphism can mutually invade, i.e. if $s(y_1,y_2) > 1$ and $s(y_2,y_1) > 1$. The set of all pairs of mutually invasible traits is given by the overlapping parts of the '+' regions in the pairwise invasibility plot and its mirror image taken along the main diagonal. Geometrically, these conditions imply that along the secondary diagonal, that is, on the line $y - x^* = -(x - x^*)$, the invasion fitness landscape $s(y,x)$ has a local minimum at x^*. Expressed mathematically, this corresponds to $A + B > 0$. Reversal of the inequality signals that a protected polymorphism does not exist.

Table 1 Different possible Evolutionarily Singular Strategies.

No.	Labels (a,b,c)	x^* locally stable (noninvasible)	x^* can invade	x^* convergence stable	Protected polymorphism
1	(−1,−1,−1)	✔	×	✔	×
2	(−1,−1,+1)	✔	×	×	×
3	(−1,+1,−1)	×	×	×	✔
4	(−1,+1,+1)	×	×	×	×
5	(+1,−1,−1)	✔	✔	✔	×
6	(+1,−1,+1)	✔	✔	✔	✔
7	(+1,+1,−1)	×	✔	×	✔
8	(+1,+1,+1)	×	✔	✔	✔

A listing of the eight different generic Evolutionarily Singular Strategies that are possible, along with their key invasion properties, as outlined in the main text. The listing is made in terms of the triplet of parameters a, b and c given in eqn 3. Note that Fig. 2 is an example of strategy no. 6 in this table, whereas Fig. 3 corresponds to strategy no. 8.

Note that both in Figs 2d and 3d, at the singular point q, the invasion fitness landscape has a local minimum on the line $y - x^* = -(x - x^*)$. Thus, protected dimorphism can exist near the singular point q in both cases.

We have not provided the detailed considerations of how the local properties of any of the Evolutionarily Singular Strategies are related to A and B , or equivalently (a,b,c) because these have been clearly presented elsewhere (Geritz *et al.*, 1998). We have, however, summarized the eight possible Evolutionarily Singular Strategies in Table 1.

2.9 What are Evolutionary Branching Points?

In Table 1, Evolutionarily Singular Strategy no. 8 has the features of being (i) invasible, (ii) able, when rare, to invade another nearby strategy, (iii) convergence stable and (iv) has protected polymorphisms. Such an Evolutionarily Singular Strategy is termed an Evolutionary Branching Point (see Fig. 3 for an example of the invasion fitness landscape in a neighbourhood of a branching point).

As the population evolves towards a branching point x^* by a sequence of small effect mutational substitutions, at some moment a mutant will be produced that has its trait value y on the opposite side of x^* to the resident trait x. From property (iv) above, the mutant y and the resident x will not oust each other but will coexist. For consistency we rename their trait values as x_1 and x_2 (the rationale for this will become apparent below).

What happens as new mutants are introduced? To answer this question one has to consider the (invasion) fitness function of a rare mutant y introduced into a *dimorphic* resident population with phenotypes x_1 and x_2. We will write this function as $s(y; x_1, x_2)$. It turns out (e.g. Geritz *et al.*, 1998) that $s(y; x_1, x_2)$ can be approximated as $1 + B(y - x_1)(y - x_2)/2$. Because $B > 0$,

it follows that only mutants outside the two resident types can invade (i.e. if $y > x_1$ and x_2 or $y < x_1$ and x_2). After invasion, the (former) resident in the middle is ousted. Therefore, with each successful invasion, the two remaining trait values will be more and more different. This process of phenotypic divergence is called 'Evolutionary Branching'.

From eqn 1, the invasion fitness of a mutation of a population that has been driven very close to the Evolutionarily Singular Strategy is $s(y,x^*) \simeq 1 + B(y - x^*)^2/2$. The fact that at the branching point $B > 0$ means that this function has a *minimum* in the vicinity of $y = x^*$. For an example of this, see Fig. 3b, where $s(y,x^*)$ is plotted as a function of y. In a sense, one can say that the population in driven towards a fitness minimum where it subsequently undergoes the process of branching.

Branching points do seem especially interesting, as they are the points near which genetic variation becomes protected. Moreover, some practitioners of Adaptive Dynamics have identified branching points with the process of sympatric speciation because in asexual models, or sexual models with, e.g. assortative mating, the clusters of individuals associated with the two branches remain distinct and may dynamically diverge from each other, thereby inducing strong differentiation (see also Question 5.3).

In Fig. 6 we present an example of branching observed in numerical simulations.

As a concrete example of an Evolutionary Branching Point, let us consider a classical model of intraspecific competition (Christiansen & Loeschcke, 1980). In this model there are two important functions that influence fitness: a function $\gamma(x,y) = \exp\left(-(x-y)^2/(2\sigma_C^2)\right)$, characterizing competition between individuals with phenotypes x and y, and a phenotype-dependent carrying capacity $K(x) = K_0 \exp\left(-x^2/(2\sigma_K^2)\right)$. Here $K_0 > 0$ is the maximum possible carrying capacity, σ_C^2 and σ_K^2 are positive parameters characterizing the strength of competition and stabilizing selection, respectively. The invasion fitness of rare, y phenotype, mutants in a resident population with phenotype x is $s(y,x) = 1 + V[K(y) - \gamma(y,x)K(x)]$ where V is a constant that lies in the range $1 > V > 0$. From this invasion fitness it can be shown that $x^* = 0$ is a branching point when $B = \left(\sigma_K^2 - \sigma_C^2\right) VK_0/(\sigma_C^2\sigma_K^2)$ is positive. Other conditions required in order for x^* to be a branching point – which correspond to x^* being able to invade when rare, x^* being convergence stable and x^* being a protected polymorphism – are satisfied for all parameter values. Thus $x^* = 0$ is a branching point when $\sigma_K^2 > \sigma_C^2$. This inequality corresponds to competition between individuals being stronger than the stabilizing selection that individuals are subject to. Figure 3 illustrates the invasion fitness landscape corresponding to this case. The Adaptive Dynamics prediction is that an asexual population first evolves towards the point $x = 0$ where it then splits into two clusters of distinct phenotype (see Fig. 6). If, on the other hand, stabilizing selection is stronger than competition, i.e. if $\sigma_K^2 < \sigma_C^2$, then the population evolves to the monomorphic state $x^* = 0$ and stays there. Figure 2 illustrates the invasion fitness landscape corresponding to this case.

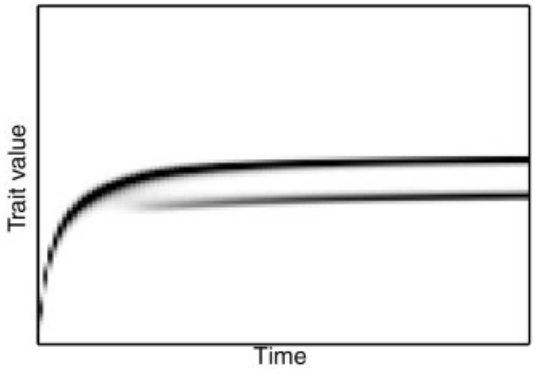

Fig. 6 An illustration of Evolutionary Branching, where an initially monomorphic population splits into two distinct branches. Darker areas correspond to higher frequencies of the corresponding trait values in the population.

The phenomenon of Evolutionary Branching is a general process for the origin and amplification of genetic heterogeneity within populations experiencing frequency-dependent selection. The word 'branching' implies that only very well defined and differentiated strategies (or trait values) are observed in the population. However, Evolutionary Branching as defined by Adaptive Dynamics (i.e. in terms of the conditions on A and B) is quite compatible with continuous and/or unimodal distributions of the trait value in the population, *if* mutations are not extremely rare or have appreciable effects (see Question 4.2). Therefore, the term 'branching' may be somewhat misleading, when the Adaptive Dynamics assumptions are not met or their validity cannot be checked. The appropriate way to interpret the conditions for 'Evolutionary Branching' generally needs to be wider than the interpretation it receives in Adaptive Dynamics, namely as conditions for the maintenance of genetic variation.

3. Relation to previous work

3.1 What is the relation to theoretical population genetics?

Theoretical population genetics is a general quantitative theory of evolutionary change whose emergence and formation in the first half of the last century allowed what is now known as the modern synthesis of the 1930s and 1940s (e.g. Provine, 1971). Although models of constant selection are most advanced in population genetics, analyses of frequency-dependent selection have a long history. There have been numerous approaches to

building models incorporating changes in both allele (or genotype) frequencies and population sizes (e.g. Kostitzin, 1937; Ludwig, 1950; Roughgarden, 1979; Ginzburg, 1983). In studies of frequency-dependent selection fitnesses have been defined both phenomenologically and from explicit ecological considerations (e.g. Bulmer, 1974, 1980; Roughgarden, 1979; Slatkin, 1979a,b, 1980; Christiansen & Loeschcke, 1980; Asmussen, 1983; Wilson & Turelli, 1986; Bürger, 2002a,b). Adaptive Dynamics continues and extends these traditions (although, unfortunately, often without acknowledging their existence).

To illustrate the differences between standard population genetics approaches (see e.g. Crow & Kimura, 1970) and those employed by Adaptive Dynamics, let us consider a simple model of a haploid population experiencing frequency-dependent selection. Assume that the population has no more than two distinct phenotypes simultaneously present. Let variables x and y specify the two phenotypes present. To develop a model describing this system the standard population genetics approach is to (i) specify the frequencies of the phenotypes in the population, say p_x and p_y, (ii) specify a frequency-dependent fitness function, say $W(y,x; p_y)$, that gives the fitness of phenotype y, with frequency p_y, in a population that also has phenotype x present, at frequency p_x, (iii) derive dynamic equations governing how p_x and p_y change in time, and (iv) analyse the transient and steady-state dynamics predicted by these equations. In contrast, the Adaptive Dynamics approach attempts to predict evolutionary change on the basis of two invasion functions: $s(y,x) = W(y,x; 0)$ – the fitness of individuals of phenotype y that are at very low frequency ($p_y \approx 0$) in a resident population where phenotype x is almost fixed ($p_x \approx 1$), and $s(x,y) = W(x,y; 0)$ which is the fitness of individuals of phenotype x that are at very low frequency ($p_x \approx 0$) in a resident population where phenotype y is almost fixed ($p_y \approx 1$). Transient dynamics are disregarded, as the focus is only on equilibrium states and on 'long-term evolution'. So, basically, steps (i) and (iii) above, are skipped altogether, whereas step (ii) uses a 'truncated' version of fitness (i.e. the invasion fitness). Using the invasion fitnesses instead of full, frequency-dependent, fitnesses is often sufficient to gain significant insight. However, the range of possible applications is narrower because a number of assumptions (discussed throughout this paper) have to be satisfied. In this regard, Adaptive Dynamics is less general.

Specifying the number and the types of possible genotypes is often a standard step in building a population genetics model. By contrast, models in Adaptive Dynamics always specify a range of possible phenotypic values that organisms can have. In this regard, Adaptive Dynamics may be considered more general. We note, however, that in the context of the modelling of the genetics of quantitative traits, the continuum-of-alleles model (Crow & Kimura, 1964) is an example where a range of effects is also specified.

3.2 What is the relation to alternative approaches to Adaptive Dynamics?

The methods of Adaptive Dynamics are heavily based on (and extend) those developed within a sub-area of population genetics that studies the dynamics of invasions as well as different attempts to generalize the Evolutionarily Stable Strategy approach (e.g. Eshel & Motro, 1981; Eshel, 1983; Vincent & Brown, 1988; Taylor, 1989; Christiansen, 1991; Abrams *et al.*, 1993a,b; Matessi & DiPasquale, 1996; Abrams, 2001). Some of these previous approaches derive the dynamic equations from population genetics considerations, explicitly accounting for sex, diploidy, etc. Some of them use more general mutation schemes which, in particular, do not require smallness of mutations (e.g. Matessi *et al.*, 2001). We note that it is possible to identify two major classes of alternative methods: 'quantitative genetic methods' and 'Evolutionarily Stable Strategy methods' (Abrams, 2001). The 'quantitative genetic methods' use quantitative genetic equations for the evolution of mean trait values, commonly assuming that the corresponding genetic variances remain constant (but for a recent example where this assumption is not made, see e.g. Waxman & Peck, 1999). The 'Evolutionarily Stable Strategy methods' concentrate on finding a complete set of evolutionarily stable phenotypes, given a set of fitness functions.

3.3 What is the relation of Evolutionarily Singular Strategies to Evolutionarily Stable Strategies?

In game theory, which may be viewed as a formulation of phenotypic evolution with frequency-dependent fitnesses, a key concept is the notion of an Evolutionarily Stable Strategy (see e.g. Maynard Smith, 1989), where a strategy or behaviour – more generally a phenotype – cannot be invaded by any other nearby phenotype. It is natural to ask whether there is any connection between an Evolutionarily Stable Strategy and the notion, introduced in the answers to Question 2.7, of an Evolutionarily Singular Strategy.

We have been discussing matters in the context of a population that experiences the recurrent appearance of new beneficial mutations, thereby introducing new phenotypes into the population. Such populations are sometimes driven to Evolutionarily Singular Strategies by their intrinsic dynamics. Clearly, some (but not all) Evolutionarily Singular Strategies are prime candidates for identification with Evolutionarily Stable Strategies. Local stability of the game theoretic strategies allow us to firmly identify these with the singular strategies numbered 1, 2, 5 and 6 in Table 1, as they have the same noninvasibility property.

Although a purely Static Equilibrium Analysis might determine a particular Evolutionarily Stable Strategy, this is not the whole story. A population will generally start some 'distance' from such a point, and local stability does

not guarantee that the population will actually be driven to the singular strategy by its intrinsic dynamics. For this to occur, the requirement of convergence stability is also required and strategies numbered 1, 5 and 6 have both of these properties. These singular strategies are both locally and convergence-stable and strategies with this feature are called 'Continuously Evolutionarily Stable' (Eshel & Motro, 1981; Eshel, 1983). An initially monomorphic population, in the vicinity of a strategies 1, 5 or 6, will, ultimately, end up in a monomorphic state from which no further evolutionary change occurs. Note that although a protected polymorphism can exist near strategy 6, this polymorphism is unstable to the introduction of mutants with the trait value closer to the singular point than both residents, so that the population will eventually end up at a monomorphic equilibrium (Geritz *et al.*, 1998).

Overall, Adaptive Dynamics is more general than the theory of Evolutionarily Stable Strategies in that it studies the convergence to singular strategies and allows for more complex dynamics.

3.4 Is there a need for Adaptive Dynamics?

Using standard population genetics approaches for analysing frequency-dependent selection is notoriously difficult. Any approximate approaches that make analysis tractable are definitely welcome. Approximate methods and models of quantitative genetics and of the theory of Evolutionarily Stable Strategies have been successful in answering this requirement to a certain extent. Adaptive Dynamics provides a new and general way for analysing a variety of interesting problems that would be difficult to approach using standard approaches. In this regard, the advent of Adaptive Dynamics has been very useful from a theoretical point of view – which is one of the reasons it has generated so much interest. Adaptive Dynamics has already had a significant impact on theoretical evolutionary research, e.g. by stimulating a new round of heated discussions on the plausibility and generality of sympatric speciation. Ultimately, though, the biological community will only maintain their interest in Adaptive Dynamics if this approach turns out to be useful in some conceptual or predictive aspects. It is up to the practitioners of Adaptive Dynamics to make a convincing case for this.

We note that the simplicity of the approach has to have some price which one should always keep in mind. This is what we consider in the following few questions.

4. Major assumptions and the consequences of their violation

4.1 What features of fitness are assumed?

The fitness functions used in Adaptive Dynamics are of a wide sort, that cover populations that are subject to density and frequency-dependent processes as well as to selection on trait values. They are implicitly assumed to be smooth functions of mutant frequency. More details of fitness are contained in the answers to Questions 2.2, 2.3 and 2.6.

Although continuity of fitness functions may be an implicit assumption of Adaptive Dynamics, it may not actually be a feature of the fitness functions actually realized in nature. Thus in the event that fitness functions are not continuous functions of mutant frequency, the frequency of mutant phenotypes cannot be neglected – even initially – and may have a significant influence on the dynamics of the population. We know, however, of no concrete examples of this and it might be interesting to see this pursued further.

4.2 What features of mutation are assumed?

There are a number of assumptions made about mutation in Adaptive Dynamics that include the following:

(i) A continuum of possible phenotypic effects is assumed (cf. Crow & Kimura, 1964), where a mutation of phenotype x results in the phenotype $x + \xi$ where ξ is a random number that is drawn from a continuous distribution (the distribution of mutant effects). Continuity of the distribution has the immediate consequence that all mutations are unique, i.e. have never previously appeared in the population. Furthermore, the phenotype of a mutation can take on any possible value and thus can range from $\infty > x > -\infty$.

(ii) Staying with continuum-of-alleles models, we note that in most calculations it is either implicitly or explicitly assumed that the distribution of the deviation of the mutant from the parental phenotype is independent of the parental phenotype. This is the original continuum-of-alleles model of mutation (Crow & Kimura, 1964). See, however recent work (Matessi *et al.*, 2001), where an alternative model of mutation, namely the House of Cards model has been employed. In the House of Cards model the trait value of a mutant is independent of the trait value of the parent (Kingman, 1978). There is yet another model of mutation that interpolates between the original mutation model of Crow & Kimura (1964), and the House of Cards model (Kingman, 1978), namely the regression model of mutation (Zeng & Cockerham, 1993). We are not aware of this somewhat flexible mutation model having been employed in an Adaptive Dynamics context.

Last, we consider two assumptions that are crucial for the conclusions of Adaptive Dynamics to be justified. These assumptions are easily violated in natural populations.

(iii) A third assumption about mutations is that the typical range of a mutation (i.e. the difference between mutant and resident phenotypes) is small. If mutations are not of small effect, then some conclusions may break down. For example, Adaptive Dynamics methodology, based on using eqn 1, predicts that polymorphism *cannot* be maintained when the singular point is locally stable.

In terms of the parameter B, that characterizes the Evolutionarily Singular Strategy (see eqn 2), this requires $B < 0$. However, the exact results of Christiansen & Loeschcke (1980) show that in their model there exist cases where polymorphism, i.e. genetic variation, can be maintained despite $B < 0$. Also, if mutations are not of small effect, the population does not necessarily approaches a convergence stable equilibrium but can 'get stuck' at a polymorphic equilibrium away from the singular point.

The question 'How small must the effect of a mutation be, for the Adaptive Dynamics results to accurately apply?', cannot be answered *a priori*.

(iv) A fourth assumption about mutations is that their rate of occurrence is very small, so that no more than two or three alleles are segregating in the population at any time. In this case the phenotype distribution has a few 'spikes' representing the discrete phenotypes present. If there are many mutations segregating, the distribution will be continuous, with no single phenotype at an appreciable frequency. It is possible to construct biologically meaningful mathematical models where such a continuous distribution of phenotypes may be established at equilibrium (e.g. Roughgarden, 1972). We are not familiar with general analytical results on this topic and continuous distributions were observed in our simulations under generic conditions. The existence of such solutions, even for vanishingly small mutation rates, indicates that mutation rate alone is not an indicator of the applicability of Adaptive Dynamics techniques.

4.3 What features of genetic drift are assumed?

In virtually all of the Adaptive Dynamics literature we have read, we have seen little or no treatment of genetic drift. See, however, Metz *et al.* (1996), where branching processes are briefly mentioned in Section 2.1. See also Dieckmann & Law (1996) where the average rate of change in a trait value is approximated under the common assumption that the rate of fixation of new alleles equals the product of the number of new mutants, per unit time, and the probability of fixation of a mutant.

The particular aspect of genetic drift we are talking about concerns the fate of new mutations in a large population. In the Adaptive Dynamics literature to date, it has been implicitly assumed that beneficial mutations, i.e. those with a positive selection coefficient, will always initially increase in frequency, irrespective of the size of the selection coefficient, and irrespective of the copy number at which they occur. Well established calculations (Haldane, 1927) have shown that in large populations, the probability of a rare mutant surviving the initial highly stochastic dynamics is approximately twice the selection coefficient when the selection coefficient is small and positive. The mutations originally considered significant for adaptation in single-step adaptive walks (Fisher, 1930) were, for geometric reasons, inferred to be only those of very small effects. More recent work, however (Kimura, 1983; Orr, 1998, 1999; Welch & Waxman, 2003) indicates, in the context of Fisher's geometric model (Fisher, 1930), that once genetic drift is taken into account, the most significant mutations, as far as adaptation is concerned, may be those with intermediately sized effects. Thus beyond simply adding a stochastic component to the system, drift is likely to *shift the emphasis* away from very small-effect mutations.

It thus requires further investigation to fully appreciate the effects of incorporating the stochastic aspects of mutation establishment in an Adaptive Dynamics context, particularly in the light of the answer to Question 4.2.

4.4 What features of sex are assumed?

In much of the literature on Adaptive Dynamics, it is assumed that individuals are asexual. Such populations have relatively simple dynamics, as there is no 'mixing' of different types within the population. In particularly, under circumstances where Evolutionary Branching occurs, the two branches diverge, due to the disruptive effects of the fitness function (see Fig. 2). By contrast, in a randomly mating sexual population, Evolutionary Branching (in the sense of formation of discrete branches) will not occur in the absence of dominance or other effects, as the offspring resulting from any mating will have an intermediate phenotype to that of the parents, nullifying any tendency to discrete branching. Thus in order for Evolutionary Branching to occur in a sexual population, mating must be nonrandom, e.g. because of assortative mating (Dieckmann & Doebeli, 1999). Implications of assortative mating are covered in the answer to Question 5.2. At the present time, there are few analytical results for the Adaptive Dynamics of sexual populations.

In sexual populations, organisms are characterized by a number of genes which are reshuffled by recombination and segregation and which can interact nonlinearly in controlling the phenotypic value(s). In general, these features make Adaptive Dynamics inapplicable to sexual populations. However, there are exceptions. For example, if mating is random and selection is in the form of the differences in viability, then the state of a one-locus, multi-allele diploid population is uniquely defined by the set of allele frequencies. For such a one-locus model, let us assume that the diploid phenotype, z_{ij}, is uniquely defined by the contributions z_i and z_j of the corresponding two alleles. In this case, the diploid model (which may incorporate frequency-dependent selection) is mathematically equivalent to an asexual haploid model. The role of the fitness of a haploid organism will be played by the induced fitness of an allele in a diploid organism. All Adaptive Dynamic results will then be applicable to the diploid case. Note that the assumption

that a diploid phenotype is uniquely defined by the corresponding two alleles is, implicitly, a statement that the degree of dominance is not subject to evolutionary change. For example, it will be satisfied if the trait is assumed to be additive so that $z_{ij} = z_i + z_j$. This assumption has been made in various studies of diploid populations (Christiansen & Loeschcke, 1980; Kisdi & Geritz, 1999; Geritz & Kisdi, 2000; Matessi *et al.*, 2001). However, if dominance is allowed to evolve, then the two variables z_i and z_j are not enough to uniquely specify the three diploid phenotypes z_{ii}, z_{ij} and z_{jj}, and the methods considered above will not apply. We note that similar conclusions have been arrived at from consideration of invasion fitnesses (van Dooren, 2005). As far as the maintenance of genetic variation is concerned, we also expect the Adaptive Dynamics approximations to work in the case of a polygenic trait controlled by equivalent loci and experiencing weak selection. In this case, linkage disequilibria can be neglected and alleles at different loci will experience similar forces and have similar dynamics.

5. Applications

The more or less standard approach in Adaptive Dynamics for answering questions about biological systems is to use the analytical techniques outlined above supporting their conclusions with numerical simulations. Here we consider three applications of Adaptive Dynamics that we personally find particularly interesting.

5.1 What does Adaptive Dynamics teach us about polymorphism?

The standard population genetics approach for studying polymorphism would be to fix parameters of an appropriate model and then to identify the region(s) in the parameter space where genetic variation is maintained. If these regions are relatively small, a natural conclusion is that conditions for polymorphism are rather strict. For example, this approach was applied to the case of constant fitnesses (Lewontin *et al.*, 1978; Turelli & Ginzburg, 1983) and it was concluded that it is very unlikely that substantial genetic variation can be maintained by selection. However, later it was shown that populations experiencing constant viability selection (Spencer & Marks, 1988, 1992; Marks & Spencer, 1991) can evolve towards the narrow area of parameter space where variation is maintained by fixing mutant alleles. In this case, even very narrow areas of parameter space can become important if populations are 'attracted' to these areas by the joint action of mutation and selection.

The ability of frequency-dependent selection to maintain genetic variation under certain conditions is well appreciated (e.g. Cockerham et al., 1972; Udovic, 1980; Asmussen & Basnayake, 1990; Altenberg, 1991). Adaptive Dynamics extends both these previous results on frequency-dependent selection and the findings about constant selection for the case of frequency-dependent selection (Spencer & Marks, 1988, 1992; Marks & Spencer, 1991). Adaptive Dynamics has provided convincing examples showing that under certain types of ecological interactions (e.g. competition, multiple niches), biological populations naturally evolve, through a sequence of fixations of mutations, towards the area of parameter space where genetic variation is maintained (e.g. Kisdi & Geritz, 1999; Geritz & Kisdi, 2000). Adaptive Dynamics has indicated that polymorphism can be maintained under more general conditions than those identified by standard population genetics methods, thus, uncovering important limitations of the standard methods.

5.2 What does Adaptive Dynamics teach us about assortative mating?

There are two major points we would like to make. The first is that Adaptive Dynamics supports and extends the notion that frequency-dependent selection often creates conditions in which 'intermediate' genotypes (or phenotypes) are present at appreciable frequencies, in spite of the fact that they have reduced fitness. This situation favours the evolution of positive assortative mating, which would reduce production of intermediate genotypes. One has to realize, however, that selection against intermediate types would favour any mechanism that would result in a reduced production of intermediate genotypes. Positive assortative mating is but one such mechanism. Production of the intermediates can also be avoided by other mechanisms including the evolution of dominance, epistasis, phenotypic plasticity or sexual dimorphism (e.g. van Dooren, 1999; Matessi *et al.*, 2001; Bolnick & Doebeli, 2003).

The second point is that in certain situations the evolution of assortative mating can be understood (and modelled) in terms of a chain of successful invasions leading to stronger assortative mating (e.g. Matessi *et al.*, 2001) or to the establishment of polymorphism (i.e. 'branching') in alleles controlling mating (van Doorn & Weissing, 2001; Gavrilets & Waxman, 2002). An illuminating observation (van Doorn & Weissing, 2001) is that competition among males for access to females (and, potentially, the competition among females for access to males) can be modelled and understood by analogies with ecological competitions for a resource. In particular, 'branching' in the male trait is expected if the competition among males is sufficiently strong relative to the breadth of the distribution of the female trait.

Unfortunately, much less is currently known about the dynamics of assortative mating than about ecological traits dynamics and some existing results are contradictory. For example, it has been shown (Matessi *et al.*, 2001) that invasion of alleles for stronger assortative mating does not necessarily lead to ever increasing assortativeness; if disruptive selection is not extremely strong, the

population reaches a polymorphic state with partial assortativeness that is stable to the invasion of further alleles. By contrast, it has been claimed (Dieckmann & Doebeli, 1999) that assortative mating evolves to become very strong under very broad conditions.

However, a more careful reading of the paper of Dieckmann and Doebeli reveals that the two alternative alleles at each locus were present at frequency 1/2 at the beginning of the simulations. Therefore, this paper does not actually consider the invasion of new alleles for assortativeness but merely their ability to survive competition in a highly heterogeneous population.

5.3 What does Adaptive Dynamics teach us about sympatric speciation?

The evolution of complete (or very strong) assortative mating is one of the possible scenarios of sympatric speciation. Therefore, the conclusions from the previous question, on assortative mating, are directly applicable to sympatric speciation. That is, Adaptive Dynamics shows that certain types of ecological interactions create conditions favouring the maintenance of genetic variation under disruptive selection which in turn favour sympatric speciation. Whether sympatric speciation actually occurs is a different issue. The overwhelming majority of papers on sympatric speciation written under the umbrella of Adaptive Dynamics are based on numerical simulations (for a rare exception, see van Doorn & Weissing, 2001) the most interesting of which are actually modifications of standard population genetics models. The numerical nature of these studies makes generalizations difficult.

The overall conclusions following from our analysis of numerical studies of sympatric speciation using Adaptive Dynamics models parallel previous results obtained within the standard population genetics approaches (e.g. Crosby, 1970; Dickinson & Antonovics, 1973; Caisse & Antonovics, 1978; Moore, 1979; Udovic, 1980; Felsenstein, 1981; Rice, 1984; Diehl & Bush, 1989; Gavrilets & Waxman, 2002; Gavrilets, 2003, 2004). That is, sympatric speciation is most plausible if disruptive selection is strong, if both viability and mating preferences are controlled by the same set of loci so that recombination does not prevent splitting of the population, if initial population variation and/or rates of mutation are very high, and if there is no selection for mating success so that choosy organisms pay no costs. Although to a certain degree it is a matter of personal interpretation, nothing in the recent Adaptive Dynamics results seriously challenges the common wisdom that conditions for sympatric speciation are rather specific (as listed above).

Common claims about a 'wide' range of conditions favouring sympatric speciation are, in our interpretation, usually based on models incorporating unrealistic assumptions or using unreasonable initial conditions and numerical values of parameters. For example, Geritz & Kisdi (2000) claim that sympatric speciation in their model does not require very strong selection against hybrids. Careful examination of the parameter values used by these authors reveals that in their model for sympatric speciation to be possible assortative mating had to be extremely strong from the start (with individuals mating with their own type with a probability 80–90%). van Doorn *et al.* (2001) claim that speciation in their model 'occurs for a wide range of parameters'. Careful examination of the parameter values used by these authors reveals that they assumed that a single mutation occurring with probability 10^{-4} per locus per generation in any of 120 loci reduces the probability of fertilization by 75%, whereas two mutations reduce it by almost 94%. This is, obviously, extremely strong assortative mating.

In the widely cited paper of Dieckmann & Doebeli (1999), initial genetic variation was set at the maximum possible level (the two alternative alleles at each locus were present at frequency 1/2), all females had equal mating success no matter how rare their preferred mates were, and the rate of mutation was set at least at two orders of magnitude higher than common estimates. None of these conditions are biologically justified. Our intuition tells us that introducing costs of choosiness and starting the population at a realistically low level of genetic variation (say, at a mutation–selection balance) with realistic values of mutation rate will almost definitely prevent sympatric speciation in the Dieckmann–Doebeli or in similar models. We note that this hypothesis is easily falsifiable.

Initial conditions with allele frequencies at 1/2 emerge in a population of hybrids between two diverged populations. In the strict sense, the paper by Dieckmann & Doebeli (1999) is not a paper about sympatric speciation (by which one usually means the emergence of a new species from within the old species) but about preventing the fusion of two species that have somehow diverged prior to their contact. Prevention of fusion of species as a result of hybridization is an important theoretical question which however is very different from the question of the origin of species (i.e. speciation).

We comment that spatial subdivision of natural populations is ubiquitous (e.g. Endler, 1977; Avise, 2000). In general, isolation by distance and spatial heterogeneity in selection resulting from spatial subdivision appear to be much more powerful and general in causing allopatric and parapatric speciation than the mechanisms envisaged in the Adaptive Dynamics models of sympatric speciation.

6. Recommendations for Adaptive Dynamics

We speculate that in the future Adaptive Dynamics will continue to be a useful method of attacking a variety of interesting problems. As we have seen, in the preceding parts of this somewhat nonstandard review, there are

hidden limitations and unconscious or implicit assumptions that are frequently made in calculations associated with Adaptive Dynamics. This is not unexpected, as the intrinsic dynamics of evolving systems is, without a doubt, complex. However, we believe that practitioners of Adaptive Dynamics will need to take a backward look at results obtained to date, with a view of establishing their full regions of validity, as well as ensuring that claims of novelty are justified and are not, in fact, reproducing or closely paralleling earlier work. They should be more careful in inventing new terms for old concepts. Consider, for example, the term 'Evolutionarily Singular Strategy'. We believe it would be simplest and most communicative to use the conventional and well understood mathematical term 'saddle point' to describe this point. This usage might allow the integration of Adaptive Dynamics into more standard theories of dynamical systems.

We also believe practitioners should be more open to referencing relevant recent work on evolutionary dynamics; more effort needs to be devoted to illuminate connections between Adaptive Dynamics approaches and alternative or well established methods. The same can be said of work that predates Adaptive Dynamics. For example, gradient-type dynamics has been well established in population genetics (e.g. Wright, 1935; Lande, 1976; Barton & Turelli, 1987) yet it is treated as a new development in Adaptive Dynamics (Dieckmann & Law, 1996; Metz *et al.*, 1996; Geritz *et al.*, 1998). Another example, already mentioned above, is the condition for 'Evolutionary Branching', $\sigma_K^2 > \sigma_c^2$ identified by Dieckmann & Doebeli (1999). This was previously found as a condition for the maintenance of genetic variation (Roughgarden, 1972; Christiansen & Loeschcke, 1980) 20 years before the advent of Adaptive Dynamics. Similarly, modelling the effects of selection gradients on the possibility of parapatric speciation has a long history (e.g. Endler, 1977; Caisse & Antonovics, 1978; Moore, 1981) which apparently was missed by recent Adaptive Dynamics papers on this subject (e.g. Doebeli & Dieckmann, 2003; Mizera & Meszéna, 2003).

On other matters, there will, no doubt, be special cases that are found where the simplest application of the theory breaks down, and for these, it will be necessary to understand and explain precisely where and what the failure is, so that a good intuition may be built up, for the benefit of later workers.

We believe that workers in Adaptive Dynamics need to come up with testable predictions of their approach to evolution, that are novel, in the sense that they cannot be easily achieved by any other approaches. It would be especially interesting to see a comprehensive comparison of empirical data and the corresponding predictions of Adaptive Dynamics.

Lastly, we note that quite a lot of the work in the literature on Adaptive Dynamics relies on numerical simulation of the behaviour of populations. We observe that there is often a somewhat sketchy description of the computational procedures adopted. To enable later workers to reproduce computational work, we would like to see clearer descriptions of this important aspect of the research, which should be viewed as being as important as an experimental protocol.

Acknowledgments

It is a pleasure to thank Tom van Dooren, Frans Jacobs, Joel Peck, John Welch and an anonymous reviewer for many helpful suggestions. We thank Roger Butlin for suggestions that initiated this project and for subsequent advice. We are also grateful to Kate Lessells, who generously went well beyond the call of duties, as handling editor of this paper.

One of us (DW) was supported by the Leverhulme Trust whereas the other (SG) was supported by National Institutes of Health grant GM56693 and by National Science Foundation grant DEB-0111613.

References

Abrams, P.A. 2001. Modelling the Adaptive Dynamics of traits involved in inter- and intraspecific interactions: an assessment of three methods. *Ecol. Lett.* **4**: 166–175.

Abrams, P.A., Harada, Y. & Matsuda, H. 1993a. On the relationship between quantitative genetic and ESS models. *Evolution* **47**: 982–985.

Abrams, P.A., Matsuda, H. & Harada, Y. 1993b. Evolutionarily unstable fitness maxima and stable fitness minima of continuous traits. *Evol. Ecol.* **7**: 465–487.

Altenberg, L. 1991. Chaos from linear frequency-dependent selection. *Am. Nat.* **138**: 51–68.

Arnold, S.J., Pfrender, M.E. & Jones, A.G. 2001. The adaptive landscape as a conceptual bridge between micro- and macroevolution. *Genetica* **112**: 9–32.

Asmussen, M.A. 1983. Density-dependent selection incorporating intraspecific competition. *Genetics* **103**: 335–350.

Asmussen, M.A. & Basnayake, E. 1990. Frequency-dependent selection: the high potential for permanent genetic variation in the diallelic, pairwise interaction model. *Genetics* **125**: 215–230.

Avise, J.C. 2000. *Phylogeography*. Harvard University Press, Cambridge, MA.

Barton, N.H. & Turelli, M., 1987. Adaptive landscapes, genetic distance and the evolution of quantitative characters. *Genet. Res.* **49**: 157–173.

Bolnick, D.I. & Doebeli, M. 2003. Sexual dimorphism and adaptive speciation: two sides of the same ecological coin. *Evolution* **57**: 2433–2449.

Bulmer, M.G. 1974. Density-dependent selection and character displacement. *Am. Nat.* **108**: 45–58.

Bulmer, M.G. 1980. *Mathematical Theory of Quantitative Genetics*. Oxford University Press, New York.

Bürger, R. 2002a. On a genetic model of intraspecific competition and stabilizing selection. *Am. Nat.* **160**: 661–682.

Bürger, R. 2002b. Additive genetic variation under intraspecific competition and stabilizing selection: a two-locus study. *Theor. Popul. Biol.* **61**: 197–213.

Caisse, M. & Antonovics, J. 1978. Evolution of reproductive isolation in clinal populations. *Heredity* **40**: 371–384.

Christiansen, F.B. 1991. On conditions for evolutionary stability for a continuously varying character. *Am. Nat.* **138**: 37–50.

Christiansen, F.B. & Loeschcke, V. 1980. Evolution and intraspecific exploitative competition I. One-locus theory for small additive gene effects. *Theor. Popul. Biol.* **18**: 297–313.

Cockerham, C.C., Prout, T., Young, S.S. & Burrows, P.M., 1972: Frequency-dependent selection in randomly mating populations. *Am. Nat.* **106**: 493–515.

Crosby, J.L. 1970. The evolution of genetic discontinuity: computer models of the selection of barriers to interbreeding between subspecies. *Heredity* **25**: 253–297.

Crow, J.F. & Kimura, M. 1964. The theory of genetic loads. *Proc. XIth Int. Congr. Genet.* **2**: 495–505.

Crow, J.F. & Kimura, M. 1970. *An Introduction to Population Genetics Theory*. Harper and Row, New York.

Dickinson, H. & Antonovics, J. 1973. Theoretical considerations of sympatric divergence. *Am. Nat.* **107**: 256–274.

Dieckmann, U. & Doebeli, M. 1999. On the origin of species by sympatric speciation. *Nature* **400**: 354–357.

Dieckmann, U. & Law, R., 1996. The dynamical theory of coevolution: a derivation from stochastic ecological processes. *J. Math. Biol.* **34**: 579–612.

Diehl, S.R. & Bush, G.L. 1989. The role of habitat preference in adaptation and speciation. In: *Speciation and Its Consequences* (D. Otte & J. A. Endler, eds), pp. 345–365. Sinauer, Sunderland, MA.

Doebeli, M. & Dieckmann, U. 2003. Speciation along environmental gradient. *Nature* **421**: 259–264.

van Dooren, T.J.M. 1999. The evolutionary ecology of dominance-recessivity. *J. Theor. Biol.* **198**: 519–532.

van Dooren, T.J.M. 2005. Adaptive dynamics for Mendelian genetics. In: *Elements of Adaptive Dynamics* (U. Dieckmann & J. A. J. Metz, eds). Cambridge University Press, Cambridge. (in press).

van Doorn, G.S. & Weissing, F.J. 2001. Ecological versus sexual selection models of sympatric speciation: a synthesis. *Selection* **2**: 17–40.

van Doorn, G.S., Luttikhuizen, P.C. & Weissing, F.J. 2001. Sexual selection at the protein level drives the extraordinary divergence of sex-related genes during sympatric speciation. *Proc. R. Soc. Lond. B* **268**: 2155–2161.

Endler, J.A. 1977. *Geographic Variation, Speciation and Clines*. Princeton University Press, Princeton, NJ.

Eshel, I. 1983. Evolutionary and continuous stability. *J. Theor. Biol.* **103**: 99–111.

Eshel, I. & Motro, U. 1981. Kin selection and strong evolutionary stability of mutual help. *Theor. Popul. Biol.* **21**: 430–439.

Fear, K.K. & Price, T. 1998. The adaptive surface in ecology. *Oikos* **82**: 440–448.

Felsenstein, J. 1981. Skepticism towards Santa Rosalia, or why are there so few kinds of animals? *Evolution* **35**: 124–138.

Fisher, R.A.. 1930. *The Genetical Theory of Natural Selection*. Oxford University Press, Oxford.

Gavrilets, S. 1997. Evolution and speciation on holey adaptive landscapes. *Trends Ecol. Evol.* **12**: 307–312.

Gavrilets, S. 2003. Models of speciation: what have we learned in 40 years? *Evolution* **57**: 2197–2215.

Gavrilets, S. 2004. *Fitness Landscapes and the Origin of Species*. Princeton University Press, Princeton, NJ.

Gavrilets, S. & Waxman, D. 2002. Sympatric speciation by sexual conflict. *Proc. Natl Acad. Sci. USA* **99**: 10533–10538.

Geritz, S.A.H. & Kisdi, E. 2000. Adaptive Dynamics in diploid, sexual populations and the evolution of reproductive isolation. *Proc. R. Soc. Lond. B* **267**: 1671–1678.

Geritz, S.A.H., Kisdi, É., Meszéna, G. & Metz, J.A.J. 1998: Evolutionarily singular strategies and the adaptive growth and branching of the evolutionary tree. *Evol. Ecol.* **12**: 35–57.

Geritz, S.A.H., Gyllenberg, M., Jacobs, F.J.A. & Parvinen, K. 2002. Invasion dynamics and attractor inheritance. *J. Math. Biol.* **44**: 548–560.

Ginzburg, L.R. 1983. *Theory of Natural Selection and Population Growth*. Benjamin Cummings Pub. Co., Menlo Park, CA.

Haldane, J.B.S. 1927. A mathematical theory of natural and artificial selection. V. Selection and mutation. *Proc. Camb. Philos. Soc.* **23**: 838–844.

Hofbauer, J. & Sigmund, K. 1990. Adaptive Dynamics and evolutionary stability. *Appl. Math. Lett.* **3**: 75–79.

Kimura, M. 1983. *The Neutral Theory of Molecular Evolution*. Cambridge University Press, Cambridge.

Kingman, J.F.C. 1978. A simple model for the balance between selection and mutation. *J. Appl. Probab.* **15**: 1–12.

Kisdi, E & Geritz, S.A.H. 1999. Adaptive Dynamics in allele space: evolution of genetic polymorphism by small mutations in a heterogeneous environment. *Evolution* **53**: 993–1008.

Kostitzin, V.A. 1937. *Mathematical Biology*. G.G. Harrap & Company Ltd, London, Toronto.

Lande, R. 1976. Natural selection and random genetic drift in phenotypic evolution. *Evolution* **30**: 314–334.

Lewontin, R.C., Ginzburg, L.R. & Tuljapurkar, S.D. 1978. Heterosis as an explanation for large amounts of genetic polymorphism. *Genetics* **88**: 149–169.

Ludwig, W. 1950. Zur theorie der Konkurrenz. Die Annidation (Einnischung) als funfter Evolutionsfaktor. *Zool. Anz. (Ergänzungsband zu Band)* **145**: 516–537.

Marks, R.W. & Spencer, H.G. 1991. The maintenance of single-locus polymorphism. II. The evolution of fitnesses and allele frequencies. *Am. Nat.* **138**: 1354–1371.

Matessi, C. & DiPasquale, C. 1996. Long-term evolution of multilocus traits. *J. Math. Biol.* **34**: 613–653.

Matessi, C., Gimelfarb, A. & Gavrilets, S. 2001. Long term buildup of reproductive isolation promoted by disruptive selection: how far does it go? *Selection* **2**: 41–64.

Matsuda, H. 1985. Evolutionarily stable strategies for predator switching. *J. Theor. Biol.* **115**: 351–366.

Maynard Smith, J. 1989. *Evolutionary Genetics*. Oxford University Press, Oxford.

Metz, J.A.J., Nisbet, R.M. & Geritz, S.A.H. 1992. How should we define fitness for general ecological scenarios. *Trends Ecol. Evol.* **7**: 198–202.

Metz, J.A.J., Geritz, S.A.H., Meszena, G., Jacobs, F.J.A. & van Heerwaarden, J.S. 1996. Adaptive Dynamics, a geometrical study of the consequences of nearly faithful reproduction. In: *Stochastic and Spatial Structures of Dynamical Systems* (S. J. van Strien & S. M. Verdyan Lunel, eds), pp. 183–231. North Holland, Amsterdam, The Netherlands.

Mizera, F. & Meszéna, G. 2003. Spatial niche packing, character displacement and adaptive speciation along an environmental gradient. *Evol. Ecol. Res.* **2**: 363–382.

Moore, W.S. 1979. A single locus mass-action model of assortative mating, with comments on the process of speciation. *Heredity* **42**: 173–186.

Moore, W.S. 1981. Assortative mating genes selected along a gradient. *Heredity* **46**: 191–195.

Mylius, S.D. & Diekmann, O. 2001. The resident strikes back: invader-induced switching of resident attractor. *J. Theor. Biol.* **211**: 297–311.

Nowak, M & Sigmund, K. 1990. the Evolution of stochastic strategies in the prisoner's dilemma. *Acta Appl. Math.* **20**: 247–265.

Orr, H.A. 1998. The population genetics of adaptation: the distribution of factors fixed during adaptive evolution. *Evolution* **52**: 935–949.

Orr, H.A. 1999. The evolutionary genetics of adaptation: a simulation study. *Genet. Res.* **74**: 207–214.

Provine, W.B. 1971. *The Origins of Theoretical Population Genetics*. Chicago University Press, Chicago.

Provine, W.B. 1986. *Sewall Wright and Evolutionary Biology*. University of Chicago Press, Chicago.

Reed, J & Stenseth, N. 1984. On evolutionarily stable strategies. *J. Theor. Biol.* **108**: 491–508.

Rice, W.R. 1984. Disruptive selection on habitat preferences and the evolution of reproductive isolation. *Evolution* **38**: 1251–1260.

Roughgarden, J. 1972. Evolution of niche width. *Am. Nat.* **106**: 683–718.

Roughgarden, J. 1979. *Theory of Population Genetics and Evolutionary Ecology: An Introduction*. Macmillan, New York.

Schaffer, W. 1977. Evolution, population dynamics, and stability: a comment. *Theor. Popul. Biol.* **11**: 326–329.

Slatkin, M. 1979a. Frequency- and density-dependent selection on a quantitative character. *Genetics* **93**: 755–771.

Slatkin, M. 1979b. The evolutionary response to frequency- and density-dependent interactions. *Am. Nat.* **114**: 384–398.

Slatkin, M. 1980. Ecological character displacement. *Ecology* **61**: 163–177.

Spencer, H.G. & Marks, R.W. 1988. The maintenance of single-locus polymorphism. 1. Numerical studies of a viability selection model. *Genetics* **120**: 605–613.

Spencer, H.G. & Marks, R.W. 1992. The maintenance of single-locus polymorphism. 4. Models with mutation from existing alleles. *Genetics* **130**: 211–221.

Taylor, P.D. 1989. Evolutionary stability in one-parameter models under weak selection. *Theor. Popul. Biol.* **36**: 125–143.

van Tienderen, P.H. & de Jong, G. 1986. Sex-ration under the haystack model – polymorphism may occur. *J. Theor. Biol.* **122**: 69–81.

Turelli, M. & Ginzburg, L.R. 1983. Should individual fitness increase with heterozygosity? *Genetics* **104**: 191–209.

Udovic, D. 1980. Frequency-dependent selection, disruptive selection, and the evolution of reproductive isolation. *Am. Nat.* **116**: 621–641.

Vincent, T.L. & Brown, J.S. 1988. The evolution of ESS theory. *Ann. Rev. Ecol. Syst.* **19**: 423–443.

Waxman, D. & Peck, J. 1999. Sex and adaptation in a changing environment. *Genetics* **153**: 1041–1053.

Welch, J. & Waxman, D. 2003. Modularity and the cost of complexity. *Evolution* **57**: 1723–1734.

Wilson, D.S. & Turelli, M. 1986. Stable underdominance and the evolutionary invasion of empty niches. *Am. Nat.* **127**: 835–850.

Wright, S. 1932. The roles of mutation, inbreeding, crossbreeding and selection in evolution. In: *Proceedings of the Sixth International Congress on Genetics* (Jones, ed.), Austin, TX, Vol. 1, pp. 356–366.

Wright, S. 1935. Evolution in populations in approximate equilibrium. *J. Genet.* **30**: 257–266.

Wright, S. 1988. Surfaces of selective value revisited. *Am. Nat.* **131**: 115–123.

Zeng, Z.-B. & Cockerham, C.C. 1993. Mutation models and quantitative genetic variation. *Genetics* **133**: 729–736.

Received 1 August 2003; revised 6 December 2004; accepted 11 March 2005