J. Math. Biol. (1996) 34: 654–674

Journal of Mathematical Biology

Inclusive fitness arguments in genetic models of behaviour

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Received 28 June 1993; received in revised form 15 December 1995

Abstract. My purpose here is to provide a coherent account of inclusive fitness techniques, accessible to a mathematically literate graduate student in evolutionary biology, and to relate these to standard one-locus genetic models. I begin in Sect. 2 with a general formulation of evolutionary stability; in Sect. 3 and Sect. 4 I interpret the basic stability conditions within genetic and inclusive fitness models. In Sect. 5 I extend these concepts to the case of a class-structured population, and in Sect. 6 I illustrate these notions with a sex ratio example. In Sect. 7 I give a proof of the result that under additive gene action and weak selection, an inclusive fitness argument is able to verify an important stability condition (2.5) for one-locus genetic models. Most of these results have been published.

1 Introduction

The idea of inclusive fitness was originally proposed by Hamilton (1964) as a simple, heuristic explanation for altruistic behaviour among relatives. Since that time, it has shown itself to be a powerful method of calculating evolutionarily stable levels of a variety of types of social behaviour, for example, sex allocation, dispersal, parental care, sib-mating, etc. Its importance as an analytical tool derives from the simplicity of its calculations and the heuristic value of its arguments. It has also been demonstrated by a number of authors that these arguments are faithful to genetic processes; in particular, under simple general conditions, inclusive fitness arguments are known to give the same results as one-locus genetic models.

I fasten attention on a behavioural parameter α, called a *strategy*, which I assume lies in the unit interval: $0 \leqq \alpha \leqq 1$. For example, α might represent the proportion of sons among an individual's offspring, the probability that an offspring will disperse, or the proportion of time or energy devoted to a certain activity such as grooming, guarding or feeding. I will restrict attention here to

effectively *monomorphic* populations, that is populations in which only a single strategy α is employed. Of course, there will always be population-wide variation in the strategies used, but I assume that the range of strategies has a distribution with a small variance, and I will work with the mean of this distribution. Indeed, for a population with such a distribution, I will assume that the fitness of any strategy β used depends only on β and on the average population strategy α. My general objective is to find a value of α which is evolutionarily stable in the sense that selection pressures caused by differing fitness for different strategies will cause the average population strategy to converge to α.

There are a number of modelling approaches that might do this, and we are concerned here with two of the simplest and most common: a one-locus genetic model and an inclusive fitness model. A main result of this paper will be that, with certain assumptions (weak selection and additive gene action) the genetic and inclusive fitness models provide mathematically identical conditions for "population" stability of a strategy α.

I now summarize the general notation. I assume we have an infinite population of individuals.

α the average strategy in the population.
β the mutant strategy.
α_x the strategy used by individual x.
h_x the phenotypic value of x, defined by the formula

$$\alpha_x = h_x\beta + (1 - h_x)\alpha \tag{1.1}$$

where α is the normal strategy. We often interpret h_x as the probability that x will exhibit mutant behaviour.

w_y the fitness of y. In general, w_y will depend on the strategy used by a large number of individuals x:

$$w_y = F(\text{many } \alpha_x)\,. \tag{1.2}$$

$w_y^x = \partial w_y/\partial \alpha_x$ is called the fitness effect of x on y, and is the rate at which deviations in the strategy of x affect the fitness of y. Here, the superscript x denotes the partial derivative with respect to α_x.

The following notation applies to the genetic model.

g_y the genotypic value of y, defined as the frequency of the mutant allele in y.

$Q = E(g_y)$ the population-wide frequency of the mutant allele.

2 The stability conditions

I begin by repeating that I restrict attention here to a one-dimensional parameter space, the possible strategies α lying in the unit interval [0, 1]. By an α-population, I mean a population in which the average strategy used is α, and I assume that the range of strategies used is distributed closely around α.

A fundamental assumption is that the expected fitness of an individual will depend upon his strategy and the average strategy in the population, and I define $W(\beta, \alpha)$ as the fitness of a β-strategist in an α-population.

Different models will have different ways of constructing this fitness function; for example, in a one-locus genetic model, $W(\beta, \alpha)$ is the change in frequency over one generation of a mutant allele coding for β, and in an inclusive fitness model, $W(\beta, \alpha)$ is the inclusive fitness of a β-individual. I will formulate the stability conditions in a general manner, so that they can be applied to a number of models.

In this section we formulate conditions for a population strategy α to be stable. In fact there is a rich and often confusing array of definitions and terminology in the literature, and I shall try to draw a basic map of some of the territory. I find it helpful to identify two general types of stability conditions in use, which roughly correspond to the two independent variables of W: the mutant variable β and the population variable α. Notions of *mutant stability* hold the population strategy α fixed and require that values of β that are different from (but perhaps near to) α be less fit. And notions of *population stability* alter α and ask whether the selective forces which then arise on β will tend to move the average population strategy towards the stable candidate. In fact, there is considerable overlap between the notions, but I find this basic distinction to be of conceptual help.

2.1 Mutant stability

In an α-population, $W(\alpha, \alpha)$ will measure what we call normal fitness . We say that α is a *Nash Equilibrium* if no strategy has greater than normal fitness: that is, for all β, $0 \leqq \beta \leqq 1$,

$$W(\beta, \alpha) \leqq W(\alpha, \alpha) . \tag{2.1}$$

A local version of this condition requires that (2.1) hold only for β in some neighbourhood of α. An important consequence of (2.1) is the *equilibrium condition*

$$\frac{\partial W}{\partial \beta}(\beta, \alpha)|_{\beta=\alpha} = 0 \tag{2.2}$$

which is typically used to find candidates for stable equilibria.

For α to be stable, in an evolutionary sense, to invasion by alternative strategies β, we need more than (2.1). One obvious idea is to ask for strict inequality in (2.1):

$$W(\beta, \alpha) < W(\alpha, \alpha) \tag{2.3}$$

for $\beta \neq \alpha$. In fact, (2.3) sometimes turns out to be too restrictive. For example, in an infinite random mating population, the stable sex ratio is $\alpha = 1/2$, but in a population with average sex ratio 1/2, all sex ratio strategies are equally fit, and (2.3) fails. Nevertheless, it is an important sufficient condition for mutant

stability, particularly in the form of the second derivative version:

$$\frac{\partial^2 W}{\partial \beta^2}(\beta, \alpha)|_{\beta=\alpha} = \ < 0 \ . \tag{2.4}$$

Conditions (2.2) and (2.4) are together stronger than (2.3).

2.2 Population stability

The condition that α be population stable is that if we consider a γ-population, for γ near α, then for β sufficiently close to γ, β-fitness should increase with β when γ is below α and should decrease with β when γ is above α.

$$\begin{aligned} &\frac{\partial W}{\partial \beta}(\beta, \gamma)|_{\beta=\gamma} > 0 \quad \text{for } \gamma < \alpha \\ &\frac{\partial W}{\partial \beta}(\beta, \gamma)|_{\beta=\gamma} < 0 \quad \text{for } \gamma > \alpha \ . \end{aligned} \tag{2.5}$$

The second-derivative version of (2.5) is

$$\frac{\partial}{\partial \gamma}\left[\frac{\partial W}{\partial \beta}(\beta, \gamma)|_{\beta=\gamma}\right]_{\gamma=\alpha} < 0 \ , \tag{2.6}$$

which can be written

$$\frac{\partial^2 W}{\partial \beta^2}(\beta, \alpha) + \frac{\partial^2 W}{\partial \alpha \partial \beta}(\beta, \alpha)|_{\beta=\alpha} < 0 \ . \tag{2.7}$$

Of the two last equivalent formulations, (2.6) is conceptually more transparent, but (2.7) is easier to verify. In conjunction with the equilibrium condition (2.2), (2.6) is slightly stronger than (2.5), but it tends to be mathematically easier to verify.

A number of papers have explored different formulations of the population stability notion (2.5). The idea was introduced by Eshel and Motro (1981) and called "strong stability." Subsequently Eshel (1983) defined a "continuously stable strategy" (CSS) to be an ESS that satisfies condition (2.5). In Taylor (1989), I called condition (2.7) "*m*-stability," but I was not happy with the terminology, and not long afterwards, Christiansen (1991) called (2.7) "covergence stability," and focused on the importance of this condition for the equilibrium to be "attainable" according to any reasonable evolutionary dynamic.

2.3 The relationship between mutant and population stability

Condition (2.5) implies the equilibrium condition (2.2) but not the Nash condition (2.1), and therefore certainly not the stronger condition (2.3). Van Tienderen and De Jong (1986 – discussed by Metz et. al. 1992), Christiansen

(1991), and Abrams et al. (1993) all give biological examples in which (2.7) holds but not (2.1). Abrams et al. (1993) provides the simplest mathematical example of this in a model of the intensity with which a resource is exploited. Their fitness function has the form:

$$W(\beta, \alpha) = \left[\frac{\beta}{a + \alpha}\right]^2 - b\beta .$$

For example, for the parameter values $a = 1$, $b = 3/8$, it is easily checked that $\alpha = 3$ is an equilibrium at which (2.7) holds but (2.1) fails. In fact, the second derivative (2.4) is positive.

In this case, a monomorphic population that starts near the equilibrium may converge, but once it has attained equilibrium, selective forces will favour alternative mutant strategies, and there is the possibility of the emergence of a stable polymorphism. Such population states are beyond the scope of the present work, but Metz (this volume) has a discussion of this phenomenon. Also in this volume, Eshel provides an extensive discussion of the general concept of stability in evolutionary modelling, and reviews the many results which tie the game theoretic notions to the selective action of genotype frequencies.

My preferred candidate for the "compleat" monomorphic stability condition in the context of a one-parameter strategy set, would be the Nash condition (2.1) together with the population stability condition (2.5) or (2.6). The importance of (2.5) in one-locus genetic models is that it involves only the β-derivative of fitness evaluated at $\beta = \alpha$, and this is the quantity that can be measured by inclusive fitness (see 7.1).

The ESS definition

I should tie these stability notions to the now classic definition of ESS. According to this definition, α is an evolutionarily stable strategy (ESS) if whenever an α-population is invaded by a rare β-mutant (of frequency ε), β has lower fitness than α *in the perturbed population.* Formally, for $\beta \neq \alpha$, we require

$$W(\beta, (1 - \varepsilon)\alpha + \varepsilon\beta) < W(\alpha, (1 - \varepsilon)\alpha + \varepsilon\beta) \tag{2.8}$$

for sufficiently small $\varepsilon > 0$. Note that this condition has elements of both mutant and population stability. Note also that by taking the limit as ε approaches zero, (2.8) implies the Nash condition (2.1), but not (2.3) – in the limit, the inequality need no longer be strict. This condition also does not imply (2.5).

In the original formulation of Maynard Smith and Price (1973) and Maynard Smith (1974), and in many of the subsequent treatments, $W(\beta, \alpha)$ is affine in the population strategy α (the term "linear" is generally used, though "affine" is technically correct; an affine function is a linear function plus a constant). Let me first give a general example of how this might arise.

Suppose there are two alternative activities, 0 and 1, and an individual's strategy is his probability of playing 1, as opposed to 0. Now suppose a β-individual's fitness is determined by random encounters in the population, gaining $w_k(\beta)$ in an encounter with a k-strategist ($k = 0, 1$). Since he will encounter a 1-strategist a proportion α of the time, his average fitness is

$$W(\beta, \alpha) = (1 - \alpha)w_0(\beta) + \alpha w_1(\beta)$$

which is affine in α.

With this affine assumption, (2.8) can be written

$$(1 - \varepsilon)W(\beta, \alpha) + \varepsilon W(\beta, \beta) < (1 - \varepsilon)W(\alpha, \alpha) + \varepsilon W(\alpha, \beta) \tag{2.9}$$

and this is easily seen to be equivalent to the condition:

$$\begin{aligned} &\text{either} && W(\beta, \alpha) < W(\alpha, \alpha) \\ &\text{or} && W(\beta, \alpha) = W(\alpha, \alpha) \quad \text{and} \quad W(\beta, \beta) < W(\alpha, \beta)\,. \end{aligned} \tag{2.10}$$

which is how the original definition was formulated. I prefer the form (2.8) because it is more general and it explicitly formulates the original intention of the definition. In the applications which I have in mind in the present paper (sex ratio, dispersal rates, parental investment, time of emergence, probability of altruism, etc.), $W(\beta, \alpha)$ is certainly not typically affine in α. I remark that Eshel (this volume) notes that (2.8) and (2.5) are equivalent when $W(\beta, \alpha)$ is affine in α.

Finally I suggest that in most biological situations, the two notions of stability, mutant and population, may not be so far apart. A stronger version of (2.5) would require that if we change the population strategy α to a nearby strategy γ, then mutants which tend to move the strategy back towards α are more fit than mutants which tend to move the strategy further away from α. More precisely, let me call α a *strong ESS* if for β and γ in some neighbourhood of α,

$$\begin{aligned} &W(\beta, \gamma) > W(\gamma, \gamma) \quad \text{if } \beta \text{ is between } \alpha \text{ and } \gamma \\ &W(\beta, \gamma) < W(\gamma, \gamma) \quad \text{if } \gamma \text{ is between } \alpha \text{ and } \beta\,. \end{aligned} \tag{2.11}$$

Although (2.11) does not seem to be an unreasonable strengthening of (2.5), it is strong enough to imply the ESS condition (2.8). To see this, first note that the first condition of (2.11) implies $W(\alpha, \gamma) \geqq W(\gamma, \gamma)$ by taking the limit as β approaches α. Now to obtain (2.8), take $\gamma = (1 - \varepsilon)\alpha + \varepsilon\beta$, and then (2.11) gives us

$$W(\beta, \gamma) < W(\gamma, \gamma) \leqq W(\alpha, \gamma)$$

which is (2.8).

3 The one-locus genetic model

For the one-locus genetic model, I suppose there are two alleles at a single locus: a normal allele and a mutant allele, and I will take mutant fitness

$W(\beta, \alpha)$ to be the change in frequency ΔQ of the mutant allele over one generation. I do not assume that the mutant allele is rare.

The mutant frequency Q changes from one generation to the next if individuals with more mutant genes tend to have greater or less fitness than individuals with fewer mutant genes, that is, if there is some correlation between fitness and genotypic value. To be precise, the mutant frequency next generation $Q^\wedge$ is the fitness-weighted average of the current genotypic values:

$$Q^\wedge = E(w_y\, g_y)/E(w_y)\,. \tag{3.1}$$

It is important to note that (3.1) assumes Mendelian segregation – that the genetic contribution of an individual to her offspring has the same average frequency of the mutant allele as does she. Then the change in mutant frequency is

$$\Delta Q = Q^\wedge - Q = E(w_y g_y)/E(w_y) - E(g)$$

$$= \frac{1}{E(w_y)}[E(w_y g_y) - E(w_y)\,E(g)] = \frac{1}{E(w_y)}[\mathrm{Cov}(w_y, g_y)] \tag{3.2}$$

This is Price's (1970) covariance formula for gene frequency change.

In a one-locus genetic model, ΔQ is our candidate for $W(\beta, \alpha)$, and so to apply the equilibrium condition (2.2) and the stability condition (2.5), we need to calculate the derivative of ΔQ with respect to β. Differentiating (3.2), using the superscript β to denote derivatives with respect to β evaluated at $\beta = \alpha$:

$$\left.\frac{\partial \Delta Q}{\partial \beta}\right|_{\beta=\alpha} = \frac{1}{E(w_y)^2}[\mathrm{Cov}(w_y^\beta, g_y)\,E(w_y) - \mathrm{Cov}(w_y, g_y)\,E(w_y^\beta)]$$

$$= \frac{1}{E(w_y)}[\mathrm{Cov}(w_y^\beta, g_y)] \tag{3.3}$$

Here I have used the fact that when $\beta = \alpha$, the mutant allele has no effect on behaviour, and fitness and genotypic value are uncorrelated, that is, $\mathrm{Cov}(w_y, g_y) = 0$. Note also that the covariance which remains, $\mathrm{Cov}(w_y^\beta, g_y)$, is also calculated at $\beta = \alpha$, that is, when the mutant allele is neutral. This is computationally important: when $\beta \neq \alpha$, the effect of the mutant allele will typically be to alter the distribution of genotypes in the population, and statistical calculations in this altered distribution can be difficult. But there are simple approaches (e.g. identity by descent) that apply to the neutral distribution.

We get an expression for the derivative of w_y with respect to β from (1.1) and (1.2):

$$w_y^\beta = \sum_x \partial w_y/\partial \alpha_x \cdot \partial \alpha_x/\partial \beta = \sum_x w_y^x h_x \tag{3.4}$$

and hence the derivative of ΔQ, from (3.3), expressed in terms of the phenotypes, genotypes, and fitness effects is given by

$$\left.\frac{\partial \Delta Q}{\partial \beta}\right|_{\beta=\alpha} = \frac{1}{E(w_y)}\mathrm{Cov}\left(\sum_x w_y^x h_x, g_y\right). \tag{3.5}$$

4 The inclusive fitness model

The *inclusive fitness effect* of the strategy α is denoted $W^I(\alpha)$ and is calculated by taking a random "actor" x in an α-population and adding up the fitness effects of x over all individuals y in the population (including x), though of course we need only include the non-zero terms, each effect weighted by r_{xy}, the relatedness of x to y. That is,

$$W^I(\alpha) = \sum_y r_{xy} w_y^x \tag{4.1}$$

Here we use the pedigree definition of relatedness (Hamilton 1964, 1972; Michod and Hamilton 1980):

$$r_{xy} = r_{x \to y} = \frac{f_{xy}}{f_{xx}}, \tag{4.2}$$

where f_{xy} is the coefficient of consanguinity between x and y defined as the probability that random alleles chosen from x and y are identical by descent (IBD) at a selectively neutral locus. In a diploid population, $f_{xx} = (1 + F)/2$, where F is the inbreeding coefficient of x.

Seger (1981) and Pamilo and Crozier (1982) provide valuable discussions of the different definitions of relatedness that have appeared. Both papers emphasize the importance of understanding what the coefficient is meant to describe, and this will be important for us in Sect. 7. Seger pays particular attention to the effects of inbreeding, and Pamilo and Crozier discuss ways in which the coefficients can be measured. Grafen (1984 and 1985a) provides a valuable simple introduction to the ideas of inclusive fitness and its relationship to old and new ideas of group selection in models of altruistic behaviour.

Often the fitness effects are specified multiplicatively, that is, we are told that a small δ-change in the behaviour of x changes the fitness of y by the *factor* $\phi_y \delta$. In this case, ϕ_y is the logarithmic derivative of fitness: $\phi_y = w_y^x / w_y$, and (4.1) becomes

$$W^I(\alpha) = \sum_y r_{xy} w_y \phi_y \,. \tag{4.3}$$

The stability conditions are obtained by letting the inclusive fitness effect $W^I(\alpha)$ play the role of the derivative $\partial W / \partial \beta$ in (2.2) and (2.5). [Indeed, it is interesting to note that W^I can actually be interpreted as a derivative. Take a population in which all individuals use strategy α except the actor x who uses β. Then $W^I(\alpha)$ is the derivative with respect to β of the weighted (by relatedness) sum of all fitnesses in the population:

$$W^I(\alpha) = \frac{\partial}{\partial \beta} \sum_y r_{xy} w_y \tag{4.4}$$

evaluated at $\beta = \alpha$.]

Thus the equilibrium condition (2.2) is

$$W^I(\alpha) = 0 \tag{4.5}$$

and the population stability condition (2.5) is that for γ near α,

$$\begin{aligned} W^I(\gamma) &> 0 \quad \text{for } \gamma < \alpha \\ W^I(\gamma) &< 0 \quad \text{for } \gamma > \alpha \,. \end{aligned} \tag{4.6}$$

There is another piece of notation which frequently arises in inclusive fitness arguments. Often, α measures the allocation of time or energy between two activities A and B; specifically, α is the allocation to activity A or the probability of engaging in A and $1 - \alpha$ measures the same for B. For example, if α is the sex ratio, A is the production of males, and B is the production of females, or A might represent offspring dispersal and B philopatry. In this case, when α is increased, two things happen: there is a greater allocation to A and a smaller allocation to B. Often, when calculating fitness changes, it is convenient to separate these two effects out. We define $W_A^I(\alpha)$ by the formula (4.1) except when calculating the fitness effect w_y^x, we allow x to increase his allocation to A without having to reduce his allocation to B. Similarly, $W_B^I(\alpha)$ is calculated by allowing x to increase his allocation to B without having to reduce his allocation to A.

With this notation, the inclusive fitness of α is

$$W^I = W_A^I - W_B^I \tag{4.7}$$

and the equilibrium condition (4.5) becomes

$$W_A^I(\alpha) = W_B^I(\alpha) \,. \tag{4.8}$$

The population stability condition (4.6) is that for γ near α,

$$\begin{aligned} W_A^I(\gamma) &> W_B^I(\gamma) \quad \text{for } \gamma < \alpha \\ W_A^I(\gamma) &< W_B^I(\gamma) \quad \text{for } \gamma > \alpha \,. \end{aligned} \tag{4.9}$$

This makes sense: (4.9) says that in a population which allocates less to A ($\gamma < \alpha$), allocation to A will be more fit than allocation to B, and viceversa for a population with a greater allocation to A.

By analogy with (2.6), the following "second derivative" conditions are usually easier to verify than (4.9), and they are both (separately) stronger than (4.9). Thus they each imply stability.

$$\frac{\mathrm{d}}{\mathrm{d}\gamma}[W_A^I(\gamma) - W_B^I(\gamma)]_{\gamma=\alpha} < 0 \tag{4.10}$$

$$\frac{\mathrm{d}}{\mathrm{d}\gamma}[W_A^I(\gamma)/W_B^I(\gamma)]_{\gamma=\alpha} < 0 \tag{4.11}$$

Condition (4.11) is useful when we are working with fitness expressions which are unnormalized, because the normalization constant (which might depend on γ) will drop out of the quotient. This is illustrated in the sex ratio example of Sect. 6.

5 Inclusive fitness in a class-structured population

I suppose that the population has a class structure of unspecified type, such that every individual belongs to some class and may have "offspring" belonging to any class. For example the classes might be the sexes (male, female, hermaphrodite) or age classes or size classes. It is simplest to assume that generations are discrete and non-overlapping, so that an individual who survives to the next generation with probability p is regarded as dying and contributing p "offspring" to the appropriate class.

To calculate fitness it may no longer be reasonable simply to count total numbers of offspring, because, for example, a class 1 offspring might be more valuable than a class 2 offspring. The notion of reproductive value (RV) allows us to compare offspring of different classes; roughly speaking the RV of a class i offspring is a measure of his average longterm genetic contribution to future generations.

An interesting example of class structure is found in a partial sib-mating model in a haplodiploid population with an inbreeding cost to daughters of sib-mated parents. (The cost is due to increased homozygosity, and is not suffered by sons who are haploid.) Then with a fixed probability that a female will sibmate, sons of sibmated parents have a smaller RV than sons of outbred parents because they have fewer sisters to mate with. Thus, in this model, we require three classes, one female class and two male classes (Taylor and Getz, 1993).

I summarize the notation. A "prime" on a variable will denote its value next generation. I assume we have a genetically uniform population with equilibrium class frequencies.

n_j is the number of individuals in class j

$N = \sum_j n_j$ is the total number of individuals.

$r = N'/N$ is the growth rate of the population.

$u_j = n_j/N$ is the proportion of individuals in class j.

w_{ij} is the average i-fitness of class j, defined as the average number of class i offspring per class j individual, where offspring must be weighted according to genetic contribution. With age classes, this is the Leslie matrix.

$w_i = \sum_j w_{ij} u_j$ is average i-fitness over the population.

$p_{ij} = w_{ij} u_j / w_i$ is the probability a random class i offspring allele comes from class j.

c_j is the reproductive value of class j, defined as the probability that a random allele in the future gene pool will derive from a class j allele in the current generation. Note that $\sum_j c_j = 1$.

$v_j = c_j/u_j$ is the average reproductive value of a class j individual, defined as the relative (to other classes) contribution of a class j individual to the future gene pool of the population, normalized so that average reproductive value is one:

$$\sum_j u_j v_j = 1 \,. \tag{5.1}$$

There is a lot of notation here; in particular, I have defined two types of reproductive value: individual RV v_j and class RV c_j. The advantage of the notation is that there's an elegant piece of mathematics which ties it all together. The idea is to look at the dominant eigenvalues of the matrices (p_{ij}) and (w_{ij}). In economic terms these are "input-output" matrices; the columns being inputs and the rows being outputs, and in such a case, the dominant eigenvalue typically has the interpretation of growth rate, the corresponding right eigenvector is the equilibrium class-frequency vector, and the corresponding left eigenvector is the vector of individual future values at equilibrium.

5.1 The right and left eigenvector result (Leslie 1948; Charlesworth 1980b)

The matrix (p_{ij}) has dominant eigenvalue 1 and the matrix (w_{ij}) has dominant eigenvalue r the growth rate of the population. The dominant right eigenvector for (p_{ij}) is the constant vector 1, and the dominant left eigenvector is the vector (c_j) of class reproductive values:

$$1 = \sum_j p_{ij} \tag{5.2}$$

$$c_j = \sum_i c_i p_{ij} \tag{5.3}$$

The dominant right eigenvector for (w_{ij}) is the stable asymptotic value of the class frequency vector (u_i) and the dominant left eigenvector is the vector (v_j) of individual reproductive values:

$$ru_i = \sum_j w_{ij} u_j \,. \tag{5.4}$$

$$rv_j = \sum_i v_i w_{ij} \tag{5.5}$$

Equation (5.2) follows from the probabilistic definition of p_{ij}. To derive (5.4) we write the dynamic equations for population change:

$$n_i' = \sum_j w_{ij} n_j \tag{5.6}$$

and divide by N to get

$$ru_i' = \sum_j w_{ij} u_j \tag{5.7}$$

which is the equation for class frequency change. At equilibrium, $u_i' = u_i$ and we get (5.4). We get (5.5) by noting that the asymptotic genetic contribution of a class j individual is made through her offspring of all classes. Since she has, on average, w_{ij} class i offspring, her reproductive value v_j must be proportional to the value of her offspring $\sum_i v_i w_{ij}$. If we hit both of these quantities with u_j and sum over j, using equations (5.1) and (5.4), we see that the constant

of proportionality must be $1/r$, and that gives us (5.5). Finally, to get equation (5.3), we multiply equation (5.5) by u_j:

$$rv_j u_j = \sum_i v_i w_{ij} u_j \tag{5.8}$$

which is written

$$rc_j = \sum_i v_i w_i p_{ij} \tag{5.9}$$

using the definition of p_{ij}. We get (5.3) using the fact that $c_i = v_i w_i$.

In practice, our reproductive value calculation follows the simplest or most natural route. In an age-structured population, the most natural matrix to calculate is the Leslie matrix (w_{ij}), and from this we obtain the individual values v_j. In a sex-structured population, the most natural matrix is often the gene-flow matrix (p_{ij}), and from this we obtain the class values c_j. As an example of the latter, in a haplodiploid population with standard pattern of inheritance, the p-matrix (male = 1, female = 2) is

$$(p_{ij}) = \begin{bmatrix} 0 & 1 \\ 1/2 & 1/2 \end{bmatrix}$$

and the left eigenvector for 1 is $(c_j) = (1/3, 2/3)$. This provides a "proof" of the standard result (Price 1970) that the female population makes twice the future genetic contribution of the male population.

How should the expression (4.1) for inclusive fitness be modified in a class-structured population? The answer is that we have to be careful to identify the nature of the fitness change, e.g. fecundity, or survival or both together, and choose the reproductive value accordingly. For example, if the effect of an actor is to increase the entire fitness of y by a factor ϕ_y, then equation (4.3) becomes

$$W^I = \sum_y r_{xy} v_y \phi_y \,. \tag{5.10}$$

To take an example, in a sex allocation problem, if W_m^I and W_f^I are the fitness effects of a "free" extra allocation to male and female offspring, then the inclusive fitness (4.7) is

$$W^I = W_m^I - W_f^I \tag{5.11}$$

with

$$W_m^I = r_m v_m \phi_m \tag{5.12}$$

and

$$W_f^I = r_f v_f \phi_f \tag{5.13}$$

where r_i is the relatedness of the actor to the sex-i offspring, ϕ_i is the rate at which extra allocation is converted into sex i offspring, and v_i is the RV of an individual offspring of sex i.

For another example, which mixes fecundity and survival, suppose extra allocation of time to parental care by the actor x reduces his viability by 2%

but increases the fecundity of his mate y by 3%. Then do we write the inclusive fitness effect as

$$W^I = (0.03)r_{xy}v_y - (0.02)r_{xx}v_x \tag{5.14}$$

where r_{xx} (= 1) and r_{xy} are the relatednesses of x to x and to y? The answer is no – the y-term is wrong. The extra fecundity results in extra offspring so that it is not r_{xy} we want but the relatedness of x to these extra offspring, and this will depend on the extent to which x or some relative of x has paternity. Also we do not want to use v_y as this represents the life-time fitness of y, and it is only the current brood we are interested in (and that is what the 3% applies to). Thus the inclusive fitness should be written

$$W^I = (0.03)r_0v_0 - (0.02)r_{xx}v_x \tag{5.15}$$

where r_0 is the average relatedness of x to y's offspring, and v_0 is the total RV of the offspring in the current brood. [And if there is more than one class of offspring involved (e.g. two sexes) then instead of r_0v_0 we would use the appropriate average value of the rv product.] A number of more challenging examples can be found in Taylor and Frank (1996).

6 A sex ratio example

Colonies of the hymenopteran ant *Apterostigma dentigerum* are initiated by a queen who produces (female) workers and (male and female) reproductives. If the queen dies or fails to function, at least one worker will lay unfertilized (haploid) male eggs. Forsyth (1981) studied a population of this species and found that in colonies with a functioning queen, the sex ratio among reproductives was female biased, and he suggested that this bias might have evolved to compensate for the all-male production of workers in colonies which had lost their queen. A simple inclusive fitness model can investigate this idea, and see just how much "compensation" is expected. We suppose that in "queen-right" colonies, the queen lays all the eggs and has control of the offspring sex ratio.

Let k denote the worker/queen ratio of reproductive output; that is, in the whole population, there are k worker-laid eggs for every queen-laid egg. Let the sex ratio for the queen be s. Then the relative numbers of males and females in the offspring population are

$$\begin{aligned} u_m &\approx k + s \\ u_f &\approx 1 - s\,. \end{aligned} \tag{6.1}$$

These expressions for the u_i must be normalized by dividing by $1 + k$. The gene-flow matrix is

$$\mathbf{p} = \begin{matrix} \\ m \\ f \end{matrix} \begin{matrix} \begin{matrix} m & \quad f \end{matrix} \\ \begin{bmatrix} \frac{k/2}{s+k} & \frac{s+k/2}{s+k} \\ 1/2 & 1/2 \end{bmatrix} \end{matrix}. \tag{6.2}$$

To find the class reproductive values we use the first column of **p** to get

$$c_m \frac{k/2}{s+k} + c_f/2 = c_m \tag{6.3}$$

which solves to give

$$\begin{aligned} c_m &\approx s + k \\ c_f &\approx 2s + k \end{aligned} \tag{6.4}$$

up to a normalizing constant. Note that if $k = 0$, there is no worker production, and we get the standard 1:2 ratio of class RV for haplodiploidy. Finally the relatednesses of mother to offspring in an outbred haplodiploid population are $r_m = 1$ and $r_f = 1/2$. The (unnormalized) fitness effects are given by equations (5.12) and (5.13) with units chosen so that $\phi_1 = 1$:

$$W_m^I = r_m v_m = r_m c_m / u_m \approx \frac{s+k}{s+k} \tag{6.5}$$

$$W_f^I = r_f v_f = r_f c_f / u_f \approx \frac{2s+k}{2(1-s)} \tag{6.6}$$

If these are equated as in equation (4.8), we get the equilibrium sex ratio

$$s = \frac{2-k}{4}. \tag{6.7}$$

Check that if $k = 0$ (no worker production), we get the expected ratio $s = 1/2$.

Since the fitnesses are unnormalized, we check stability with (4.11). The derivative

$$\frac{d}{ds}[W_m^I(s)/W_f^I(s)] = \frac{d}{ds}\left[\frac{2(1-s)}{2s+k}\right] = \frac{-2(2+k)}{(2s+k)^2} \tag{6.8}$$

is clearly negative, and the sex ratio is stable.

A naive conjecture might be that the queen should choose a sex ratio which produces an unbiased overall population ratio, that is, which makes u_m equal to u_f. From (6.1), this would require

$$s = \frac{1-k}{2} = \frac{2-2k}{4}. \tag{6.9}$$

Comparison of the last expression in (6.9) with (6.7) shows that the ESS queen strategy "splits the difference" between an unbiased ratio among her own offspring and an unbiased population ratio. Equation (6.7) has been obtained by Taylor (1981) and, as part of a more general model with the queen laying two successive broods, by Iwasa (1981 – his eq. (22) with $t = 1$ and $k = N_{10}/N_2$).

7 The equivalence between one-locus genetic models and inclusive fitness

What I show here is that, under simple genetic assumptions (weak selection and additive gene action), the derivative of the change in mutant frequency given by (3.5) is equivalent to the inclusive fitness defined by (4.1):

$$\left.\frac{\partial \Delta Q}{\partial \beta}\right|_{\beta=\alpha} = KW^I(\alpha) \tag{7.1}$$

for some positive constant $K = K(\alpha)$. The importance of (7.1) is that it allows us to use inclusive fitness to check population stability (2.5) in a one-locus genetic model.

The argument is given for a one-class population. In the discussion (8.1) I mention how the analysis is modified in a class-structured population.

I begin by rewriting the expression (3.5) in a sequence of steps which I justify below.

$$\begin{aligned}
\left.\frac{\partial \Delta Q}{\partial \beta}\right|_{\beta=\alpha} E(w) &= \mathrm{Cov}\left(\sum_x w_y^x h_x, g_y\right) \\
&= N\,\mathrm{Cov}(E_y w_y^x h_x, g_y) && (7.2)\\
&= N\,\mathrm{Cov}(w_y^x h_x, g_y) && (7.3)\\
&= N\,[E\,\mathrm{Cov}_k(w_y^x h_x, g_y) + \mathrm{Cov}(E_k(w_y^x h_x), E_k(g_y))] && (7.4)\\
&= N\,[E w_k^x\,\mathrm{Cov}_k(h_x, g_y)] && (7.5)\\
&= \frac{N}{N^2}\left[\sum_k n_k N w_k^x\,\mathrm{Cov}_k(h_x, g_y)\right] && (7.6)\\
&= \left[\sum_k n_k w_k^x r_k\,\mathrm{Cov}(g_x, h_x)\right] && (7.7)
\end{aligned}$$

In (7.2) the average E_y is taken over all x for each y, and the covariance is over all y. In (7.3), the covariance is now taken over all pairs (x, y). This last step uses a special case of the covariance decomposition theorem (A2) where the classes are indexed by y, and g_y is constant over each class.

To get (7.4) we note that a typical individual x is engaged in different relatedness classes of interactions, e.g. brother-brother, or father-daughter, or nearest neighbour etc. such that the relatedness of x to y is constant within any class k, and we apply the covariance decomposition to this class structure. Now fasten attention on (7.4). If $\beta = 0$, then $E_k(g_y)$ is independent of k, which is to say that the genotype of an individual y has no effect of his likelihood of appearing in any particular relatedness class and the second term of (7.4) is zero. This requires, for example, that individuals with more mutant genes are not more likely to be brothers (or are not likely to have more brothers) than normal, and this will be the case if the mutant allele is neutral.

In (7.5), w_y^x can be taken out of the covariance as its average w_k^x over all class k interactions because, within class k, the fitness effect w_y^x is uncorrelated

with genotype or phenotype. [Again this might be strictly true only when the mutant allele is neutral ($\beta = 0$).]

In (7.6) I have replaced the expectation by the appropriate weighted average, where n_k is the average number of class k relatives per individual, so that $n_k N$ is the size of class k. Note that $\sum_k n_k N = N^2$ since it must equal the total number of sample points. Finally, in (7.7), I have introduced

$$r_k = \frac{\mathrm{Cov}_k(g_y, h_x)}{\mathrm{Cov}(g_x, h_x)} \tag{7.8}$$

which I will call the average relatedness of x to y over class k interactions.

Equation (7.7) can be written

$$\left.\frac{\partial \Delta Q}{\partial \beta}\right|_{\beta=\alpha} = K \sum_k n_k w_k^x r_k \tag{7.9}$$

where $K = \mathrm{Cov}(g_x, h_x)/E(w)$ depends on α and is positive if genotype and phenotype are positively correlated. I show below that, provided allelic effects are additive (i.e. the mutant allele is semidominant), the two definitions of relatedness coincide, that is, r_k defined in (7.8) is the same as the pedigree definition $r_{x \to y}$ in (4.2) for a class k interaction. This establishes the equivalence (7.1) between the two modelling approaches.

7.1 The identity of the relatedness coefficients

Let the mutant and normal alleles have *genic values* 1 and 0, and frequencies p and $q = 1 - p$. Note that if a and b are the genic values of the two alleles belonging to x, then the genotypic value of x is

$$g_x = \frac{a + b}{2}, \tag{7.10}$$

which is the average genic value of x. Also, if a is the value of a random allele in the population, then

$$E(a) = E(a^2) = p$$

and

$$\mathrm{Var}(a) = \mathrm{Cov}(a, a) = E(a^2) - E(a)^2 = p - p^2 = pq\ . \tag{7.11}$$

I consider a fixed relatedness class k of interactants, e.g. two brothers, and I remain inside this class for the rest of this section. In Table 1, I classify all the pairs (x, y) in this relatedness class according to the IBD pattern of their alleles, and for each pattern, I calculate the covariance between genotypic values and show that, in each row,

$$\mathrm{Cov}_i(g_x, g_y) = pq g_{xy} \tag{7.12}$$

where the covariance is over all pairs belonging to row i. The classification scheme of Table 1 follows Jacquard (1974) and has been employed for the same purpose by Michod and Hamilton (1980) and Grafen (1984). Now

Table 1. Classification of pairs (x, y) of interactants in a fixed relatedness class k according to the IBD pattern of their alleles. In the covariance calculation I use (7.10) and (7.11) and I assume that two random alleles a and b must either be IBD or independent, and in the latter case $\mathrm{Cov}(a, b) = 0$. [Crow and Kimura 1970, p. 69 – it seems to me that this assumption requires that mating be non-assortative, that is that no component of mate choice is based on phenotypic value at the mutant locus.]

x	y	$\mathrm{Cov}(g_x, g_y)$	g_{xy}
·	·		
·	·		
⎥	⋮		
		0	0
⋮	⎥		
⎥	⎥		
———		$\mathrm{Cov}\left(\frac{a+b}{2}, \frac{a+c}{2}\right) = \frac{\mathrm{Cov}(a,a)}{4} = \frac{pq}{4}$	$\frac{1}{4}$
———	·		
———	———	$\mathrm{Cov}\left(\frac{a+b}{2}, \frac{a+b}{2}\right) = \frac{\mathrm{Cov}(a,a)+\mathrm{Cov}(b,b)}{4} = \frac{pq}{2}$	$\frac{1}{2}$
◺	·	$\mathrm{Cov}\left(a, \frac{a+b}{2}\right) = \frac{\mathrm{Cov}(a,a)}{2} = \frac{pq}{2}$	$\frac{1}{2}$
⊠		$\mathrm{Cov}(a, a) = pq$	1

I decompose the covariance (Appendix) over the entire relatedness class k according to the rows of the Table 1:

$$\mathrm{Cov}(g_x, g_y) = E\mathrm{Cov}_i(g_x, g_y) + \mathrm{Cov}(E_i g_x, E_k g_y) = pqg_{xy} \tag{7.13}$$

where the second term vanishes because $E_i g_x = p$ and is independent of i. This result also applies in case $y = x$, and we have that, under additive gene action $(h_x = g_x)$:

$$r_k = \frac{\mathrm{Cov}_k(g_y, h_x)}{\mathrm{Cov}(g_x, h_x)} = \frac{g_{xy}}{g_{xx}} = r_{x \to y} \tag{7.14}$$

and this demonstrates the equivalence of the relatedness forms. I have done the analysis for diploidy, but it also holds for haplodiploidy.

8 Discussion

The main result of Sect. 7 states that under certain conditions (discussed below), an inclusive fitness analysis will provide the same equilibrium condition (2.2) and the same population stability condition (2.5) as a one-locus

genetic model. Mathematically, the reason for this is that the derivative of mutant fitness with respect to the mutant strategy β (at $\beta = \alpha$) has the same sign in both models. A number of authors have explored this question of the equivalence of the two approaches. Hamilton (1975) was the first to do an analysis of Price's equation in a group-structured population, and to construct from there the condition for the spread of an altruistic trait. Since that time, particular models, and versions of the general result, have been obtained by Charnov (1977), Charlesworth (1980a), Michod and Hamilton (1980), Seger (1981), Grafen (1985a) and Taylor (1989, 1990).

To generalize this equivalence result to a class-structured population, it is first necessary to ask how the mutant frequency should be defined. The most natural definition is to weight each individual by his reproductive value v_j. This gives us the definition

$$Q = \sum v_j Q_j u_j = \sum c_j Q_j \tag{8.1}$$

where Q_j is the mutant frequency among class j individuals. With this definition, and with inclusive fitness defined as in Sect. 5, the equivalence result of Sect. 7 continues to hold (Taylor 1990).

The equivalence result is important because genetic models are generally regarded as the most realistic approaches to the modelling of simple kinds of evolutionary behaviour, whereas inclusive fitness arguments are often computationally simpler and offer a powerful conceptual heuristic which can aid our understanding, particularly in the study of social interactions. Indeed, in my work over the past few years, I have studied a number of examples in which the genetic model is intractible, even for a rare mutant (typically because the matrix is too large), but the inclusive fitness model is quite accessible. For example, this occurs in models of sex ratio and dispersal in a patch structured population with partial dispersal of offspring (Taylor 1988a, b) and in a recent model of sibmating with inbreeding depression (Taylor and Getz, 1994).

There are two important assumptions behind the equivalence result (7.1): that the mutant gene have small effect (weak selection), and that fitness interactions between genes be additive. I will discuss each of these.

The essential problem with large effects (strong selection) is that the mutant gene, by its deviant behaviour, can alter its own distribution, and so, for example, change the probability that the bearer will engage in a certain type of interaction (e.g. alter his number of brothers) or even change his relatedness to a fixed class of interactants (for example, a gene affecting mate choice might alter the relatedness between brothers). The first factor figured in our derivation of (7.7), and the second in our argument that the two types of relatedness coefficients were identical. And in the extension of the argument to class-structured populations, it is necessary that the mutant gene have negligible effect on the class frequencies u_j and reproductive values v_i. Mathematically, the equivalence is obtained by ignoring higher (than linear) powers in the mutant deviation $(\beta - \alpha)$ and this is the reason it is difficult to find an equivalence for mutant stability (2.4). The possibilities for an equivalence

under strong selection have been discussed by Charlesworth (1980a); he reports that there is little hope unless the mutant allele is rare (because of frequency dependent effects) and even then, there are additional conditions that must be met.

The assumption of additive gene action has components both between and within individuals. Between individuals, it is necessary that fitness effects be additive, that is, that the fitness functions (1.2) be linear. In fact, this rarely happens, but an important dividend of weak selection is that the fitness effect (3.4) *is* linear. [Indeed, what the derivative is all about, is that it linearizes non-linear functions.] Queller (1985) has a nice discussion of a particularly important non-additivity (synergistic interactions between altruist and recipient) and in the same issue Grafen (1985b) observes that weak selection allows additivity to be restored. The within-individual component is a requirement of semidominance of the mutant allele. In fact, this assumption is often not required and even when it is, the effect of dominance is slight. For example I analyzed (Taylor 1981) the sex ratio model considered above (sex ratio bias due to worker laying) with a one locus genetic model with a dominant mutant allele, and obtained the same result. On the other hand, a sex ratio model for a patch structured population with partial dispersal of offspring considered by Bulmer (1986) with a genetic model, and by Frank (1986) and Taylor (1989) with an inclusive fitness model, showed deviations in the ESS sex ratio depending on the level of dominance, but these were slight – for three breeding females per patch, the variation from the semidominance result was at most 0.2%. Taylor and Getz (1994) compared the two modelling approaches in a model of the advantage of sibmating under inbreeding cost, and found that dominance had a negligible effect under weak selection, but a noticeable effect for large values of β.

In this paper, I have restricted attention to stability of pure strategies, that is, to populations with a uniform value of α. A natural extension of these results would study genetic polymorphisms – stable mixtures of two or more strategies.

Appendix: Covariance results

For simplicity, I assume all random variables are defined on a discrete set of N points (the sample space). Suppose the sample space is partitioned into classes, with class i of size n_i, and for any random variable a, let a_{ij} denote the a-value of the jth sample point in class i. Then if a and b are two random variables, their covariance over the whole space can be decomposed along these classes:

$$\mathrm{Cov}(a_{ij}, b_{ij}) = E(\mathrm{Cov}_i(a_{ij}, b_{ij})) + \mathrm{Cov}(E_i(a_{ij}), E_i(b_{ij})) \qquad \text{(A1)}$$

The first term in (A1) is the average within class covariance, and the second term is the covariance of the class averages. Here the notation cov_i and E_i indicates that the covariance and the expectation are taken for fixed i over the

sample points in class i. For example, $E_i(a_{ij})$ is the average value of a over class i. Note that the E in the first term and the Cov in the second term represent *weighted* averages over the classes, where class i has weight n_i/N.

An important special case arises when the points in class i all have the same a-value a_i. Indeed, the random variable a might have been used to define the classes, the classes being the level sets of a. In this case, covariance of a and b over class i is zero for all i, and we have only the second term:

$$\mathrm{Cov}(a_{ij}, b_{ij}) = \mathrm{Cov}(E_i(a_{ij}), E_i(b_{ij}))\ . \tag{A2}$$

Acknowledgements. This work was partially supported by a grant from the Natural Sciences and Engineering Research Council of Canada. I am grateful to Ilan Eshel and to an anonymous referee for a number of important comments.

References

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

Bulmer, M. G. 1986. Sex ratio theory in geographically structured populations, Heredity **56**: 69–73

Charlesworth, B. 1980a. Models of kin selection. In: Evolution of Social Behaviour: Hypotheses and Empirical Tests (ed. H. Markl), Verlag Chemie, Weinheim

Charlesworth, B. 1980b. Evolution in age-structured populations. Cambridge Studies in Mathematical Biology, Cambridge Univ. Press

Charnov, E.L. 1977. An elementary treatment of the genetical theory of kin selection, J. Theor. Biol. **66**: 541–550

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

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

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

Eshel, I and Motro, U. 1981. Kin selection and strong evolutionary stability of mutual help, Theor. Pop. Biol. **19**: 420–433

Forsyth, A. 1981. Sex ratio and parental investment in an ant population. Evolution **36**: 1252–1253

Frank, S. A. 1986. Hierarchical selection theory and sex ratios. I. General solutions for structured populations, Theor. Pop. Biol. **29**: 312–342

Grafen, A. 1984. Natural, kin and group selection, in Behavioural Ecology, An Evolutionary Approach (J. R. Krebs and N. B. Davies, eds) 62–84. Sinauer

Grafen, A. 1985a. A geometric view of relatedness, Oxford Surveys in Evolutionary Biology **2**: 28–89

Grafen, A. 1985b. Hamilton's rule OK, Nature **318**: 310–311

Hamilton, W. D. 1964. The genetical evolution of social behaviour, I and II, J. Theor. Biol. **7**: 1–52

Hamilton, W. D. 1970. Selfish and spiteful behaviour in an evolutionary model, Nature (Lond.) **228**: 1218–1220

Hamilton, W. D. 1972. Altruism and related phenomena, mainly in social insects, Ann. Rev. Ecol. Syst. **3**: 192–232

Hamilton, W. D. 1975. Innate social aptitudes of man: an approach from evolutionary biology. In: Biosocial Anthropology (R. Fox, ed.). pp 133–155. New York: John Wiley and sons

Iwasa, Y. 1981. Role of sex ratio in the evolution of eusociality in haplodiploid social insects, J. theor. Biol. **93**: 125–142

Jacquard, A. 1974. The Genetic Structure of Populations (trans. D. and B. Charlesworth) Biomathematics Series 5, Springer, New York
Leslie, P. H. 1948. Some further remarks on the use of matrices in population mathematics, Biometrika **35**: 213–245
Maynard Smith, J. 1974. The theory of games and the evolution of animal conflicts, J. theor. Biol. **47**: 209–221
Maynard Smith, J. and Price, G. R. 1973. The logic of animal conflict. Nature **246**: 15–18
Metz, J. A. J., R. M. Nisbet and Geritz, S. A. H. 1992. How should we define 'fitness' for general ecological scenarios? TREE **7**: 198–202
Michod, R. E. and Hamilton, W. D. 1980. Coefficients of relatedness in sociobiology, Nature **288**: 694–697
Pamilo, P. and Crozier, R. H. 1982. Measuring genetic relatedness in natural populations: methodology. Theor. Pop. Biol. **21**: 171–193
Price, G. R. 1970. Selection and covariance, Nature **227**: 520–521
Queller, D. C. 1985. Kinship, reciprocity and synergism in the evolution of social behaviour, Nature **318**: 366–367
Seger, J. 1981. Kinship and covariance, J. theor. Biol. **91**: 191–213
Taylor, P. D. 1981. Sex ratio compensation in ant populations. Evolution **35**: 1250–1251
Taylor, P. D. 1988a. An inclusive fitness model for dispersal of offspring, J. theor. Biol. **130**: 363–378
Taylor, P. D. 1988b. Inclusive fitness models with two sexes. Theor. Pop. Biol. **34**: 145–168
Taylor, P. D. 1989. Evolutionary stability in one-parameter models under weak selection, Theor. Pop. Biol. **36**: 125–143
Taylor, P. D. 1990. Allele frequency change in a class-structured population, American Naturalist **135**: 95–106
Taylor, P. D. and Getz, W. M. 1994. An inclusive fitness model for the evolutionary advantage of sib-mating. Evol. Ecol. 8: 61–69
Taylor, P. D. and Frank, S. A. 1996. How to make a kin selection model. J. theor. Biol. (in press)
van Tienderen, P. H. and De Jong, D. 1986. Sex ratio under the haystack model: polymorphism may occur. J. theor. Biol. **122**: 69–81