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# Local Stability in a Seed Provisioning Model

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A model of seed provisioning is used to illustrate the relationship between an inclusive fitness and a one locus genetic model, with particular attention to the two basic local stability conditions: $\delta$-stability and $m$-stability The model is able to illustrate the difference between these two conditions, and provide an example in which the first holds but not the second.

## 1. Introduction

The "calculus" of inclusive fitness (Hamilton, 1964, 1970, 1972) provides a powerful method, both conceptually and computationally, of modeling social behavior. An important question concerns the extent to which inclusive fitness models are able to give the same results as genetic models. There is quite a general result, various versions of which have been discussed by Hamilton (1970, 1979), Charlesworth (1980), Seger (1981), Grafen (1985), and Taylor (submitted), which provides a partial equivalence between inclusive fitness conditions and one-locus genetic models when selection is weak. The purpose of this paper is to illustrate this equivalence result with a seed provisioning model of Queller (1983, 1984), paying particular attention to the extent to which the inclusive fitness model is able to diagnose the local stability of the equilibrium. The model will also serve as a good example of the relationship between two different types of local stability.

I begin with a simple description of an ESS model of behavior, general enough to embrace both an inclusive fitness model, and a one-locus genetic model. I assume that the range of possible behaviors is described by a continuous parameter $m$ which can be regarded as the probability of engaging in a certain activity, or the proportion of resources invested in one activity instead of another. I let $m$ stand for "normal" behavior, and consider a rare "deviant" behavior $m+\delta$. The objective of the model is to produce an expression $W(m, \delta)$ for the incremental fitness of the deviant behavior. If $\delta = 0$, deviant behavior is the same as normal behavior, and $W$ will be zero. A "weak-selection" model is interested in the form of $W$ for values of $\delta$ close to zero, and looks at the Taylor series expansion of $W$ about $\delta = 0$:

$$W(m, \delta) = \delta F_1(m) + (\delta^2/2) F_2(m) + O(\delta^2), \tag{1.1}$$

where $F_1 = \partial W/\partial \delta$ and $F_2 = \partial^2 W/\partial \delta^2$. If $F_1(m) \neq 0$, the story is told by the first term in eqn (1.1). If $F_1(m) > 0$, then $W$ will be positive for $\delta > 0$, and selection will favour an increase in $m$. If $F_1(m) < 0$, then $W$ will be positive for $\delta < 0$, and selection will



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favour a decrease in $m$. The eqn $F_1(m)=0$, which can be written

$$\frac{\partial W}{\partial m}(m,0)=0 \tag{1.2}$$

is called the equilibrium condition, and any solution $m^*$ gives an equilibrium point for the model.

Equation (1.1) provides two formal ways in which an equilibrium $m^*$ might be called locally stable. Stability to changes in the normal strategy $m$, will be called "$m$-stability", and stability to changes in the deviant strategy $\delta$ will be called "$\delta$-stability". Under $m$-stability, if $m>m^*$, slection should favour mutants with $\delta<0$, so we want $F_1(m)<0$, but if $m<m^*$, selection should favour $\delta>0$, so $F_1(m)>0$. The first-order condition in $F_1$ for this is

$m$-stability $$\mathrm{d}F_1/\mathrm{d}m<0 \text{ at } m=m^*. \tag{1.3}$$

Under $\delta$-stability, at $m=m^*$, we want $W<0$ for all sufficiently small $\delta$, which requires

$\delta$-stability $$F_2(m^*)<0. \tag{1.4}$$

As second-order conditions in $W$, these can be written

$m$-stability $$\frac{\partial^2 W}{\partial m\,\partial \delta}(m^*,0)<0 \tag{1.3}'$$

$\delta$-stability $$\frac{\partial^2 W}{\partial \delta^2}(m^*,0)<0. \tag{1.4}'$$

Eshel & Motro (1981) and Eshel (1983) call condition (1.4)′ the ESS condition (evolutionarily stable strategy), and when both conditions hold, the ESS is called continuously stable or CSS, so named because condition (1.3) only makes sense when $m$ is a continuous variable. My preference is to make both conditions part of what is generally called evolutionary stability for one-parameter models. Elsewhere (Taylor, submitted) I argue that condition (1.3) is closely related to Fisher's classical argument (1930) for the stability of the unbiased sex ratio, and I also show, in the context of linear games between relatives (Hines & Maynard Smith, 1979; Grafen, 1979), that conditions (1.3) and (1.4) are each equivalent to Maynard Smith's (1974) definition of ESS (see also Maynard Smith & Price, 1973). Eshel & Motro (1981) give a mathematical example in which condition (1.3) is stronger than condition (1.4), and the seed provisioning model discussed here provides a biological example of the same type.

Two important examples of this general modeling approach are found in inclusive fitness models, and population genetics models with two alleles at a single locus. The inclusive fitness argument typically considers a single deviant individual (playing $m+\delta$) in a normal population (playing $m$) and takes as $W$ the inclusive fitness increment $w_I$ of the deviant individual, defined as the change in fitness of the deviant individual plus the sum of all changes in fitness of other individuals in the population, due to the behavioral deviation, each such change weighted by coefficient of relatedness of the affected individual to the deviant individual. The population

genetics model considers a normal allele (coding for $m$) and a rare mutant allele (coding for $m+\delta$) and takes as $W$ the change $\Delta Q$ in the frequency $Q$ of the mutant allele over a single generation.

The equivalence result referred to above is the following. Provided the assortment of gametes among offspring is Mendelian and the correct relatedness coefficient is used, the inclusive fitness $w_I$ and the allele frequency change $\Delta Q$ have the same first-order coefficient $F_1(m)$ in eqn (1.1) (up to a positive multiplicative constant), and hence they have the same equilibrium points [the solutions of eqn (1.2)], and the same $m$-stability condition [condition (1.3)] for these points. However, the $\delta$-stability condition will not, in general, be the same.

In section 2, I construct an inclusive fitness model for seed provisioning, under two different assumptions on how extra resources to one embryo affect the fitness of its sibs, and in section 3, I construct a one-locus genetic model under one of these two assumptions, and compare the different local stability results obtained. I also recall just what the relatedness coefficient has to be for the equivalence result to hold.

## 2. Inclusive Fitness Model of Seed Provisioning

When a plant produces multiple seeds, a decision must be made concerning the amount of resources to be given to each seed. The more a particular seed gets, the less will be available for other seeds, contemporary or future, of the same maternal plant. Thus there is a trade-off between the fitness of each seed, and the total fitness of all its sibs (who may only be half-sibs, depending on paternity). This trade-off may lead to a conflict between the various agents who may have (partial) control over the allocation of resources to each embryo, such as the mother, the embryo itself, or certain intermediate structures such as the endosperm in angiosperms.

The optimum allocation from the point of view of each agent depends on the precise way in which extra resources to one seed affect the quantity or quality of its sibs. In fact, roughly speaking, these two modes of effect, quantity and quality describe two rather natural special cases. The first case, in which extra allocation to one seed reduces the number of sibs, but leaves their fitness unaltered, is apt to describe the case in which offspring are produced sequentially with a fixed level of resource supply to the mother, and the effect of a greedy embryo is to end the mother's reproductive life after fewer total seeds have been produced. The second case, in which extra allocation reduces the fitness of sibs, but leaves their number unaltered, may describe the case in which seeds are produced in batches of fixed size, and what one embryo gains, the others will lose.

Models of the first, the quantitative case, have been considered by Trivers (1974), Parker & Macnair (1978), Macnair & Parker (1978), Charnov (1979), Westoby & Rice (1982), Queller (1983, 1984), and Bulmer (1986), and of the second, the qualitative case, by Macnair & Parker (1979), and Law & Cannings (1984). To illustrate the contrast between the two cases, I construct a simple inclusive fitness model which assumes a fixed total resource $M$ to the mother, which must equal the sum of her allocation to all seeds. I also assume no cost to any party for a differential modification of the allocation.

The model requires the specification of the function $f(m)$ describing the expected fitness of a seed receiving $m$ resource units. I assume the graph of $f(m)$ has the sigmoid form of Fig. 1. If $m$ is the normal population-wide level of resource allocation per embryo, then a deviant embryo which receives $m+\delta$ will have fitness increment $f(m+\delta)-f(m)$.

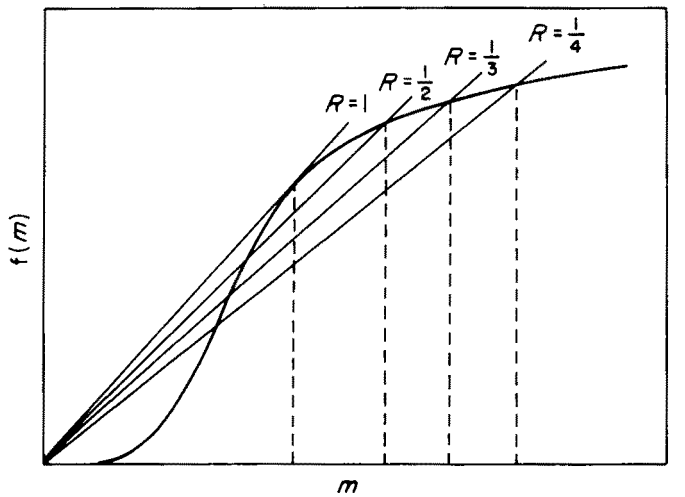


FIG. 1. Graph of expected fitness $f(m)$ of a seed receiving $m$ resource units. Solutions to the equilibrium eqn (2.5), for the quantitative case, are illustrated for the different values of $R$ tabulated in eqn (2.9).

On the other hand, its loss through reduced fitness of sibs will have a different expression in the two cases. In the quantitative case, each seed receives $m$ units, and this loss of $\delta$ units will result in a loss of $\delta/m$ seeds with fitness $f(m)$. This must be weighted by the relatedness $R$ of these other seeds to the embryo, so the inclusive fitness increment of the deviant embryo is

$$w_I = f(m+\delta) - f(m) - Rf(m)(\delta/m). \tag{2.1}$$

In the qualitative case, if there are a total of $S+1$ seeds, each of the $S$ sibs will lose $\delta/S$ units, and will have fitness $f(m-\delta/S)-f(m)$, so the inclusive fitness increment of the deviant embryo is

$$w_I = f(m+\delta) - f(m) + RS[f(m-\delta/S) - f(m)]. \tag{2.2}$$

In both cases, the calculation of $R$ requires specification of the controlling agent and the mode of gene action (Michod & Hamilton, 1980; Taylor, 1988 submitted).

If we differentiate $w_I$ with respect to $\delta$, we get the form (1.1) with

$$\begin{aligned} F_1(m) &= f'(m) - Rf(m)/m \\ F_2(m) &= f''(m) \end{aligned} \tag{2.3}$$

in the quantitative case, and

$$F_1(m) = (1-R)f'(m)$$
$$F_2(m) = (1+R/S)f''(m) \tag{2.4}$$

in the qualitative case.

In the quantitative case eqn (2.3), the equilibrium eqn (1.2) is

$$f'(m) = Rf(m)/m \tag{2.5}$$

and a number of solutions are illustrated in Fig. 1 for different values of $R$. The condition (1.3) for $m$-stability for any such solution is

$$f''(m) < -R(1-R)f(m)/m^2 \tag{2.6}$$

and the condition (1.4) for $\delta$-stability is

$$f''(m) < 0 \tag{2.7}$$

at $m = m^*$. Condition (2.6) is stronger and says that for $m$-stability it is not in general enough that $f''(m^*)$ be negative; it is necessary that it be not too close to zero.

For the qualitative case, whenever $R < 1$, equilibrium eqn (1.2) is attained only when $f'(m^*) = 0$, and both types of stability conditions require $f''(m^*) < 0$, which means $m^*$ is a local maximum of $f(m)$. This implies that the embryo will be selected to take as much of the maternal resource as it can effectively get. Our assumption of no cost for a differential reallocation will be unreasonable for large allocations to each seed, and a more realistic model of this second case will have to build this in.

However, we can see that there is an important difference between the quantitative and qualitative cases. In the case of eqn (2.2), the embryo is selected to behave selfishly, no matter how closely it is related to its sibling embryos, but in eqn (2.1) it will want to take less for itself when $R$ is closer to 1. This distinction has been noted and discussed by Macnair & Parker (1979) and Bulmer (1986).

The relatedness coefficient $R$ to be used in the above equations is "the relatedness of $y$ to $x$ under the control of $z$" which is

$$R = R^z_{x \to y} = \frac{f_{yz}}{f_{xz}}, \tag{2.8}$$

where $f_{xz}$ is the coefficient of consanguinity between $x$ and $z$, and is defined as the probability that random alleles from $x$ and $z$, at the locus in question, are identical by descent.

The relatedness, eqn (2.8), is not hard to calculate in the case in which $x$ and $y$ are half sibs of an outbred diploid mother, under a number of control agents: the mother, the embryo itself, the gametophyte (a haploid entity genetically identical to the maternal contribution to the embryo), and the endosperm (a triploid entity with two doses of the maternal and one of the paternal contributions to the embryo).

The results are tabulated below (Westoby & Rice, 1982; Queller, 1983, 1984).

| Control $z$ | $R^z_{x\to y}$ |
|---|---|
| mother | 1 |
| gametophyte | $\frac{1}{2}$ |
| endosperm | $\frac{1}{3}$ |
| embryo | $\frac{1}{4}$ |

(2.9)

The corresponding solutions of eqn (2.5) are depicted in Fig. 1. Bulmer (1986) tabulates relatedness coefficients for a number of cases in which the endosperm is genetically more complex, but it is important to note that Bulmer uses the coefficient $r_{xy}$ defined as the probability that a random allele from $y$ has an IBD copy in $x$, and this is not in general the same as the coefficient $R$ in eqn (2.8).

We see that the case $R = 1$ does occur under maternal control. From the point of view of the mother, there is no reason to prefer $x$ over $y$. In general the relatedness $R$ decreases as the control agent gets genetically "closer" to $x$.

## 3. Genetic Model of the Quantitative-effect Case

For the rest of the paper I restrict attention to the more interesting case in which a greedy embryo affects the quantity but not the quality of its sibs. I construct a one-locus diploid genetic model for this case and formulate the equilibrium and stability conditions. The expression for $\Delta Q$ is obtained using Price's elegant and simple covariance formula (1970).

What the genetic model requires is an expression for the change $\Delta Q$ in mutant allele frequency over one generation. The way to find this is to relate the genetic composition (the genotype) of a random embryo to its fitness, and to find its fitness we have to know its behavior (its phenotype), and the behavior of its sibs. We define the genotypic value $G_x$ of an embryo $x$ to be the frequency of the mutant allele in its genotype, and, for fixed $m$ and $\delta$, we define the phenotypic value $H_x$ to be such that the allocation of resources to $x$ is $m + H_x\delta$. I will focus attention on a random embryo $x$, and denote by $H_y$ the average phenotypic value of all the offspring of $x$'s mother.

Recall that each maternal plant has a fixed total resource $M$ to divide among her ovules. Hence the probability that a random embryo $x$ will be developed is inversely proportional to its mother's average allocation per embryo, and if developed, $x$ will have fitness $\mathrm{f}(m + H_x\delta)$, so the fitness of $x$ is

$$W_x = \frac{\mathrm{f}(m + H_x\delta)}{m + H_y\delta},$$

which expands in powers of $\delta$ as

$$W_x = \frac{\mathrm{f}(m)}{m}\left\{1 + \delta\left[H_x\frac{\mathrm{f}'(m)}{\mathrm{f}(m)} - H_y\frac{1}{m}\right] + \delta^2\left[H_y^2\frac{1}{m^2} + H_x^2\frac{\mathrm{f}''(m)}{2\mathrm{f}(m)} - H_xH_y\frac{\mathrm{f}'(m)}{m\mathrm{f}(m)}\right]\right\} + o(\delta^2). \tag{3.1}$$

Provided inheritance of parental alleles is Mendelian, the change in mutant allele frequency is then the co-variance between the genotypic value and the normalized fitness of $x$ (Price, 1970):

$$\Delta Q = \mathrm{Cov}\,(G_x, W_x)/E(W_x), \tag{3.2}$$

where $E(W_x)$ is the population-wide average fitness.

Note that since the $\delta$ term of $W_x$ in eqn (3.1) is proportional to phenotypic value, it will be first order in the mutant allele frequency $Q$, and hence so will the $\delta$ term in $E(W_x)$. Since I have assumed a rare mutant allele, we are interested only in the terms in $\Delta Q$ which are first order in $Q$, and since $G_x$ is already first order in $Q$, we can replace $E(W_x)$ in eqn (3.2) by its zeroth-order term $\mathrm{f}(m)/m$. If we then put eqn (3.1) into eqn (3.2), we get the form of eqn (1.1)

$$\Delta Q = \delta F_1(m) + (\delta^2/2)F_2(m) + o(\delta^2), \tag{3.3}$$

where

$$F_1 = \mathrm{Cov}\,(G_x, H_x)\frac{\mathrm{f}'(m)}{\mathrm{f}(m)} - \mathrm{Cov}\,(G_x, H_y)\frac{1}{m} \tag{3.4}$$

$$F_2 = \mathrm{Cov}\,(G_x, H_y^2)\frac{2}{m^2} + \mathrm{Cov}\,(G_x, H_x^2)\frac{\mathrm{f}''(m)}{\mathrm{f}(m)} - \mathrm{Cov}\,(G_x, H_xH_y)\frac{2\mathrm{f}'(m)}{m\mathrm{f}(m)}.$$

This appears to give the expansion of $\Delta Q$ in powers of $\delta$, but care must be taken at this point. The covariances themselves can be expected to depend on $\delta$, and if this is the case, the covariances in $F_1$ may contribute some additional $\delta^2$ terms to $\Delta Q$. In our simple model, this does not happen; to first order in $Q$, the covariances are independent of $\delta$. Indeed, to first order in $Q$, a mutant embryo must have (with probability 50% each) either a singly mutant mother or father (but not both), and in each case, the distribution of the sib phenotypes is fixed and independent of $\delta$. This would fail to be true in the presence of homozygose mutants, and this would occur if the mutant allele were not rare, or if there were inbreeding.

Now I remark that $F_1$ in eqn (3.4) differs from eqn (2.3) by a positive multiplicative constant, if $R$ is given by

$$R^z_{x\to y} = \frac{\mathrm{Cov}\,(G_y, H_x)}{\mathrm{Cov}\,(G_x, H_x)}. \tag{3.5}$$

Actually, this assumes $\mathrm{Cov}\,(G_x, H_x)$ is positive, and requires the observation that, by symmetry, $\mathrm{Cov}\,(G_x, H_y) = \mathrm{Cov}\,(G_y, H_x)$. In this case, to say that allocation of resources is under the control of $z$, is to say that $H_x$ is a function of the genotypic value of $z$, $H_x = F(G_z)$, and this function, which will be determined by the mode of gene action, must be specified for $R$ in eqn (3.5) to be calculated. In case the mutant allele is neutral, and gene action in $z$ is additive (that is, $H_x$ depends affinely on $G_z$) or $z$ is outbred, the coefficient defined by eqn (3.5) coincides with the standard inclusive fitness form eqn (2.8) (Michod & Hamilton, 1980; Seger, 1981; Grafen, 1985; Taylor, 1988 submitted).

It follows that, provided this more general form of $R$ is used, the equilibrium condition (1.2) of the genetic model, $F_1 = 0$, is equivalent to eqn (2.5), obtained by the inclusive fitness argument.

I now do the stability analysis. Since the $F_1$ in eqns (2.3) and (3.4) are proportional, the condition (1.3) for $m$-stability of the genetic model, that $dF_1/dm$ be negative, is equivalent to the $m$-stability condition (2.6) for the inclusive fitness model. For my purpose here, I write it in the form

$$f''(m) < -\left[\frac{f(m)}{m^2}\right] K_m \tag{3.6}$$

where

$$K_m = R(1-R). \tag{3.7}$$

However, the condition (1.4) for $\delta$-stability of the equilibrium is quite different from the inclusive fitness condition (2.7), and can be written as

$$f''(m) < -\left[\frac{f(m)}{m^2}\right] K_\delta \tag{3.8}$$

where

$$K_\delta = \frac{2[R\,\mathrm{Cov}\,(G_x, H_x H_y) - \mathrm{Cov}\,(G_x, H_y^2)]}{\mathrm{Cov}\,(G_x, H_x^2)}. \tag{3.9}$$

It turns out that for additive gene action, under all four control agents tabulated in condition (2.9), $K_m$ exceeds $K_\delta$, and $m$-stability is stronger than $\delta$-stability. I am unable to show that this is true in general, but I present the calculations for the four cases mentioned.

For the calculation of the covariances, note that $E(G_x) = Q$, the mutant allele frequency, and all covariance terms can be written

$$\mathrm{Cov}\,(G_x, L) = E[(G_x - Q), L] \simeq E(G_x L)$$

to first order in $Q$, and so for a rare mutant, we can ignore the cases in which $G_x = 0$, which means we only have to consider mutant embryos. Now a proportion $Q$ of all embryos are mutant with a mutant mother, and a proportion $Q$ are mutant with a mutant father. If, in the first case, we let $H_x = h_2$ and $H_y = h_0$ and, in the second, we let $H_x = h_1$ and $H_y = 0$, then

$$\begin{aligned}
\mathrm{Cov}\,(G_x, H_x) &= Q(h_1 + h_2)/2 \\
\mathrm{Cov}\,(G_x, H_y) &= Qh_0/2 \\
\mathrm{Cov}\,(G_x, H_x^2) &= Q(h_1^2 + h_2^2)/2 \\
\mathrm{Cov}\,(G_x, H_x H_y) &= Qh_0 h_2/2 \\
\mathrm{Cov}\,(G_x, H_y^2) &= Qh_0^2/2
\end{aligned} \tag{3.10}$$

Putting these into eqns (3.7) and (3.9), we get

$$K_m = \frac{h_0(h_1 + h_2 - h_0)}{(h_1 + h_2)^2}, \tag{3.11}$$

$$K_\delta = \frac{2h_0^2 h_1}{(h_1 + h_2)(h_1^2 + h_2^2)}. \tag{3.12}$$

The results, for the case of additive gene action, are presented in Table 1. Under maternal control, the stability conditions are the same, and require only that $f(m^*)$ be negative. But for the other three control agents, $m$-stability is the stronger condition.

In particular, for these three control agents, $m$-stability will fail if $f''(m^*)$ is too close to zero. To illustrate what happens in such a case, I present, in Fig. 2, an example of a fitness function, in which the equilibrium point $m^*$ occurs at a point at which the graph is a linear segment between the two endpoints $m_1$ and $m_2$. Using the inclusive fitness form eqn (2.3) of $F_1$, which is equivalent to eqn (3.4),

$$F_1(m) = c\left[f'(m) - R\frac{f(m)}{m}\right],$$

where $c$ is a positive constant, and we see that the equilibrium point $m^*$ is a point at which the slope of the graph is $R$ times the slope of the line from the origin.

TABLE 1

*Calculation of stability coefficients in eqns (3.7) and (3.9) for m-stability and δ-stability, under four different control agents, assuming additive gene action.*

| Control Agent | | $R$ | $K_m$ | $K_\delta$ |
|---|---|---|---|---|
| Mother | $h_1 = 0,\ h_2 = h_0$ | 1 | 0 | 0 |
| Gametophyte | $h_1 = 0,\ h_2 = 2h_0$ | 1/2 | 1/4 | 0 |
| Endosperm | $h_1 = h_0 = h_2/2$ | 1/3 | 2/9 | 2/15 |
| Embryo | $h_1 = h_2 = 2h_0$ | 1/4 | 3/16 | 1/8 |

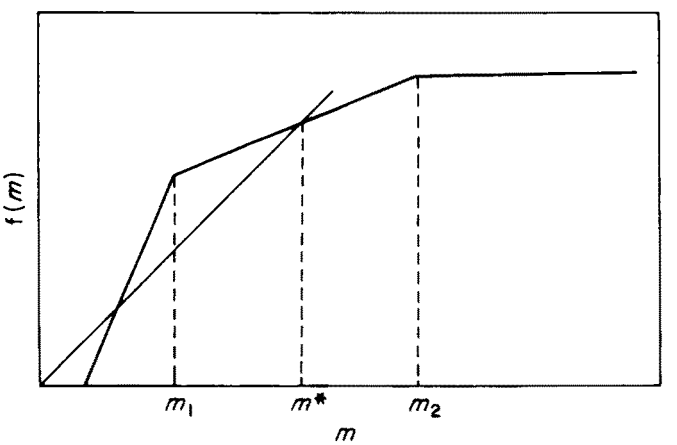


FIG. 2. Example of an equilibrium point [a solution of condition (2.5)], for the quantitative case, which is genetically not $m$-stable [condition (3.6) fails because $f''(m^*) = 0$]. If we gave the line segment a slight curve, we would get a point which, for the case of gametophyte control, was $\delta$-stable but not $m$-stable. [If we make $f''(m^*)$ slightly negative, condition (3.6) will continue to fail, since $K_m = 1/4$, but condition (3.8) will hold because $K_\delta = 0$—see Table 1.]

Also it is clear that, since $f'(m)$ stays constant for $m$ just above $m^*$, $F_1(m)$ will be positive, and for $m$ just below $m^*$, $F_1(m)$ will be negative. Equation (3.3) tells us that in the first case, selection will favour $\delta > 0$, and in the second, $\delta < 0$. Thus $m$ will move away from $m^*$ in either direction, until it reaches either $m_1$ or $m_2$. Now, neither of these points can be called equilibrium points, because $f(m)$, and therefore $F_1(m)$, fail to be differentiable there, but both these points are $m$-stable. Indeed, I have just argued that $m_2$ is stable from below and $m_1$ from above. But also, if $m$ is above $m_2$, $f'(m) = 0$ and $F_1(m)$ will be negative, and if $m$ is below $m_1$, $f'(m)$ exceeds $f(m)/m$, the slope of the line from the origin, and $F_1(m)$ is certainly positive, with eqn (3.3) telling us that, in both cases, the action of selection will be to move $m$ back towards $m_i$.

To get a differentiable example, the curve can be made smooth by rounding the corners, and we will get equilibrium points at each corner. For example, at $m_1$, as I have argued above, $F_1(m)$ is negative for $m$ just above the corner, and $F_1(m)$ is positive for $m$ just below the corner, so somewhere in the (rounded) corner $F_1(m)$ will vanish. This will be stable if we make the corner sharp enough to give $f''(m)$ a large enough negative value. This illustrates a general phenomenon: an unstable equilibrium point will tend to have stable equilibria on either side.

Curves which are qualitatively similar to Fig. 2, in which the slope changes slowly on some intervals and quickly on others, will tend to concentrate their stable equilibria at points at which the slope changes quickly, thus reducing the potential conflict between different agents of control.

## 4. Discussion

I have begun with a general discussion of local stability in an evolutionary model of behavior in which two natural conditions arise, $m$-stability and $\delta$-stability. The first focuses on the effect of departures from equilibrium in the population-wide or normal value of the behavioral parameter, while the second focuses on changes in the mutant value when the population is at equilibrium.

This general discussion provides a framework to study local stability for both inclusive fitness and one-locus genetic models. Both types of models are useful: inclusive fitness models are simpler and provide a powerful heuristic, both conceptually and computationally, whereas population genetic models follow more closely the mechanisms of the evolutionary process. We are particularly interested in cases in which the two types of models can be predicted to give the same results, because then the simplicity of the inclusive fitness approach can give some insight into the process of natural selection.

In all the particular genetic models I have looked at, $m$-stability has been the stronger of the two conditions, but I do not have a general result of this type. Even in the simple genetic model of seed provisioning treated in section 3, it is difficult to show that for all possible control agents, the $m$-stability condition (3.6) is stronger than the $\delta$-stability condition (3.8). A result which could show that, for genetic models under a wide range of conditions, $m$-stability was stronger than $\delta$-stability

would be of great value because, in most cases, $m$-stability is considerably easier to check than $\delta$-stability. The reason for this is that the $m$-stability condition is first-order in $\delta$, and coincides, in fact, with $m$-stability in the inclusive fitness model (if $R$ is chosen correctly), whereas the $\delta$-stability condition is second-order in $\delta$, and requires the calculation of covariances between genotypic value and second-order terms in phenotypic value.

The model of seed provisioning in angiosperms provides a good illustration of this general discussion. I have identified two different ways in which extra allocation to one embryo can affect is sibs: quantitatively, by reducing their numbers, and qualitatively, by reducing their fitness. Inclusive fitness models for each of these cases show that there is an important difference between them with regard to sib altruism: in the quantitative case the developing embryo is selected to consider the needs of its sibs, increasingly as the relatedness between them is closer, whereas in the qualitative case an embryo is selected to be selfish no matter how closely related are its sibs.

When selection is weak, the inclusive fitness model will always give the same equilibrium condition eqn (1.2) as the one-locus model provided the relatedness $R$ is taken as eqn (3.5). There are two important cases in which this $R$ is equal to the simple inclusive fitness form eqn (2.8): when gene action is additive ($H_x$ is affinely related to $G_z$), and when $z$ is outbred (Michod & Hamilton, 1980; Seger, 1981; Grafen, 1985; Taylor, submitted). But if both these conditions fail, $R$ must be calculated from the covariances, as in eqn (3.10), and $R$ can then change with different assumptions on the dominance of the mutant allele, possibly causing the equilibrium point to change. This can be seen to occur for the seed provisioning model with endosperm control, and is discussed by Queller (1984), Grafen (1985) and Bulmer (1986).

In the genetic model of seed provisioning, in section 3, I have calculated both the $m$-stability and the $\delta$-stability conditions, under additive gene action, for four different control agents, and found that the $m$-stability condition is stronger, except in the case of maternal control, when they are the same. The results are presented in Table 1. This allows us to construct an example of a $\delta$-stable point which is not $m$-stable. Take, for example, the graph in Fig. 2, and give the straight line segment a slight curvature, so that $\mathrm{f}''(m)$ becomes negative but stays greater than $-\mathrm{f}(m)/4m^2$ at the point $m^*$. If, at $m = m^*$, the slopes are such that $f'(m) = \mathrm{f}(m)/2m$, then the point $m^*$ will be at equilibrium under gametophyte control (see Table 1) and will be $\delta$-stable, but will not be $m$-stable. Selection acting on $m$ will cause the population allocation to move either up or down, until an $m$-stable equilibrium is encountered. Similar examples could be constructed for endosperm or embryo control.

Queller (1984) has constructed genetic models for all the cases I have considered, not with the use of Price's formula, but by direct calculation of frequency change in the mutant allele, and it is interesting to compare the two approaches. Queller also has an interesting discussion of endosperm control when the mutant allele is no longer rare, and there are frequency dependent effects. When Queller checks the stability of his equilibria, it is $\delta$-stability he is using, though in the case of endosperm and offspring control he has reached the conclusion that the equilibrium is stable

as long as $f''(m^*)$ is negative, whereas the condition in Table 1 is stronger than this. This seems to be due to an error in differentiating the expressions for his difference function $D$, for these expressions are correct.

The discussions of Queller (1983, 1984) and Bulmer (1986) contain a fuller account of the interesting question of the role of the endosperm in seed provisioning.

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