

## **Polymorphism in a varied environment: how robust are the models?**

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*(Received 22 April 1979)*

### SUMMARY

This paper shows that a number of models of the maintenance of polymorphism in a heterogeneous environment, including those of Levene and Dempster, can be derived from a simple assumption about the way in which the numbers and kinds of individuals emerging from a niche depend on the number of eggs laid in it. It is shown that for such models, unless selective advantages per locus are large, protected polymorphism requires that the relative niche sizes lie in a narrow range. This lack of robustness applies also to models of stable polymorphism proposed by Clarke and by Stewart & Levin. Excluding models relying on habitat selection or restricted migration, the only models which may escape this criticism are diploid models with partial dominance with respect to fitness, such as one proposed by Gillespie, in which in all niches the fitness of heterozygotes is higher than the arithmetic mean of the homozygotes.

### 1. INTRODUCTION

It has been known since Levene (1953) that a stable genetic polymorphism can be maintained in a spatially heterogeneous environment, although this depends critically on the way selection is supposed to act (Dempster, 1955). It has less often been realized that, unless selective advantages per locus are large, the fitness must be nicely adjusted to the niche sizes if polymorphism is to be maintained (Maynard Smith, 1966). To put the matter in another way, fitnesses which will maintain polymorphism for one set of niche sizes will in general not do so if the relative niche sizes are varied. This fact casts serious doubts on the plausibility of this type of mechanism as an important cause of extensive polymorphism.

In section 2 of this paper we derive a range of models, including those of Levene and Dempster, from a simple and plausible model of the 'input-output' relations of a niche; that is, of the numbers of individuals of different genotypes emerging from a niche as a function of the numbers of eggs laid in the niche. Although some versions of this model do give polymorphism for a somewhat wider range of niche

sizes than the simple Levene model, the gain in robustness is not great. In section 3 we consider a related model due to Clarke (1972) and in section 4 a model due to Stewart & Levin (1973); neither model escapes the difficulty.

In sections 2–4 we consider models with only two phenotypes at a locus; i.e. haploid models, or models with complete dominance. In section 5 we extend the analysis to diploid models with intermediate dominance. If the fitness of the heterozygote is exactly intermediate between the two homozygotes, there is little gain in robustness. However, Gillespie (1976) has proposed a model, with some empirical support, in which heterozygotes are intermediate in fitness but closer to the fitter homozygote in each niche. We show that Gillespie's conditions for the stability of his model can be derived from Levene's (1953) conditions. This model, perhaps because it produces an overall heterozygote advantage, preserves polymorphism over a much wider range of environmental conditions.

We have not attempted to analyse the full range of models of a heterogeneous environment. We do not consider any models with habitat selection or migration between niches. We consider only models which are coarse-grained (Levins, 1962), in the sense that selection acts on an individual only in a single niche, although in the model of Stewart & Levin (1973) conditions in the niche change in a regular way during the life of the individual. Finally, we take little account of temporal variation of the environment, although we do show in section 2 that this contributes nothing to the robustness of Levene's model.

## 2. A GENERAL MODEL LEADING TO DEMPSTER'S AND LEVENE'S MODELS

The essential features of the models of Levene (1953) and Dempster (1955) are:

- (i) the adults form a single random mating population,
- (ii) after mating, females lay their eggs in one of a number of 'niches', in which the selective values of the genotypes differ.

The models differ in the way in which the population is supposed to be regulated. We therefore start with a general model of the way in which the numbers and kinds of individuals emerging from a niche depend on the numbers and kinds of eggs laid in it. Consider first eggs of a single genotype, laid in a single niche. Let  $x$  be the number of eggs laid and  $y$  be the number of adults emerging. A reasonable functional form is (see Fig. 1)

$$y = \frac{vx}{1+ax}. \quad (1)$$

Then  $k = v/a$  is the carrying capacity of the niche,  $v$  is the non-density-dependent fitness, and is related to  $r$  in the logistic equation by  $r = vE$ , where  $E$  is the number of (female) eggs laid per adult female.  $v$  will be called the 'intrinsic fitness'.

The models analysed in this section and in sections 3 and 4 consider only two genotypes,  $g$  and  $G$ . For each genotype on its own:

$$\begin{aligned} \text{genotype } g, \quad y &= \frac{vx}{1+ax}; \quad k = \frac{v}{a}, \\ \text{genotype } G, \quad Y &= \frac{VX}{1+AX}; \quad K = \frac{V}{A}. \end{aligned}$$

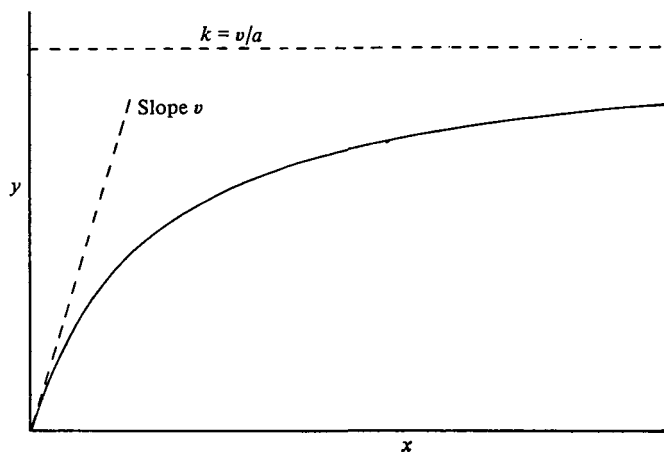


Fig. 1. Number of adults produced in a niche,  $y$ , as a function of the number of eggs laid,  $x$ .

When both genotypes are present simultaneously, an obvious functional form, which has the necessary property that, when  $ax + AX \ll 1$ , each genotype has a fitness unaffected by the other, is

$$y = \frac{vx}{1 + ax + A'X}; \quad Y = \frac{VX}{1 + a'x + AX}. \tag{2}$$

Now consider an adult population distributing eggs of two genotypes into two niches, indicated by subscripts.

Assume no 'habitat selection', i.e.

$$\frac{x_1}{X_1} = \frac{x_2}{X_2} = u. \tag{3}$$

It will be convenient to put  $x_1/x_2 = X_1/X_2 = r$ , where  $r$  is a measure of the relative sizes of the niches.

The basic equations then are:

$$\left. \begin{array}{l} \text{Niche 1} \qquad \qquad \qquad \text{Niche 2} \\ \text{genotype } g, \quad y_1 = \frac{v_1 x_1}{1 + a_1 x_1 + A'_1 X_1}, \quad y_2 = \frac{v_2 x_2}{1 + a_2 x_2 + A'_2 X_2}, \\ \text{genotype } G, \quad Y_1 = \frac{V_1 X_1}{1 + a'_1 x_1 + A_1 X_1}, \quad Y_2 = \frac{V_2 X_2}{1 + a'_2 x_2 + A_2 X_2}. \end{array} \right\} \tag{4}$$

By varying the parameters in these equations it is possible to obtain a range of models, including Dempster's (1955) and Levene's (1953). From (4) equations for the gene frequencies of  $g$  and  $G$  can be derived; by linearizing these in the neighbourhood of the trivial gene frequency equilibria 0 and 1, conditions for protected polymorphism can be obtained.

*Case 1. Low density in the niches*

Assume  $ax + AX \ll 1$  in both niches; i.e. the density in the niche is low. The absolute fitnesses are independent both of density and of frequency.

The conditions for a protected polymorphism are

$$\frac{rv_1+v_2}{rV_1+V_2} > 1 < \frac{rV_1+V_2}{rv_1+v_2}, \tag{5}$$

which cannot be satisfied. This is Dempster’s model, in which polymorphism is impossible.

*Case 2. High density: genotypes identical as density-limiting factors*

Assume  $ax + AX \gg 1$  in both niches; i.e. the niches are at their carrying capacities. The conditions for a protected polymorphism are

and 
$$\left. \begin{aligned} \frac{v_1}{A'_1} + \frac{v_2}{A'_2} &> \frac{V_1}{A_1} + \frac{V_2}{A_2} \\ \frac{V_1}{a'_1} + \frac{V_2}{a'_2} &> \frac{v_1}{a_1} + \frac{v_2}{a_2}. \end{aligned} \right\} \tag{6}$$

We now assume that  $A'_1 = a_1$ ,  $A'_2 = a_2$ ,  $a'_1 = A_1$  and  $a'_2 = A_2$ . Thus individuals of the two genotypes have identical effects as density-limiting factors, although they have different intrinsic fitnesses ( $v$ ) and carrying capacities ( $v/a$ ). With these conditions, (6) cannot be satisfied, and polymorphism is impossible.

*Case 3. High density: each genotype has the same limiting effect on itself as on its competitor*

Assume  $ax + AX \gg 1$  in both niches and

$$a'_1 = a_1, \quad A'_1 = A_1, \quad a'_2 = a_2, \quad A'_2 = A_2.$$

It is convenient to consider three cases, illustrated in Fig. 2.

*Case 3(a). Carrying capacity proportional to intrinsic fitness*

Assume  $a_1 = A_1$  and  $a_2 = A_2$ , so that

$$\frac{k_1}{K_1} = \frac{v_1}{V_1} \quad \text{and} \quad \frac{k_2}{K_2} = \frac{v_2}{V_2} \quad (\text{see Fig. 2a}).$$

With these constraints on the  $a$ 's, conditions (6) cannot be satisfied, and no polymorphism is possible.

*Case 3(b). Carrying capacity constant and independent of genotypes*

Assume

$$k_1 = K_1 \quad \text{so} \quad \frac{v_1}{a_1} = \frac{V_1}{A_1} \quad \text{and} \quad k_2 = K_2 \quad \text{so} \quad \frac{v_2}{a_2} = \frac{V_2}{A_2} \quad (\text{see Fig. 2b}).$$

This case corresponds to Levene’s model. Strictly, Levene assumes only that the fraction of the total population produced in each niche remains constant and independent of genotype. However, it is hard to see how this could be so

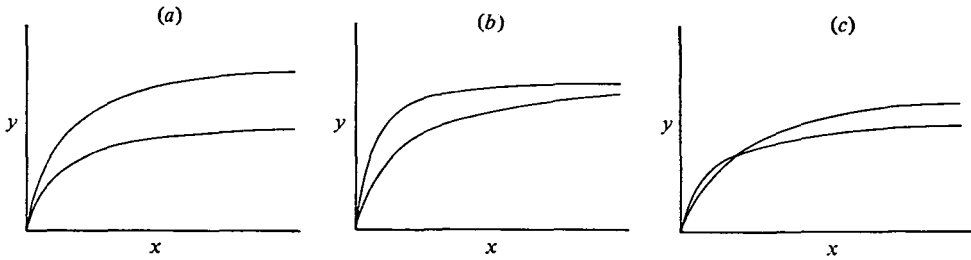


Fig. 2. Three types of relationship between adults produced,  $y$ , and eggs laid,  $x$ .

unless the absolute numbers produced were constant, and that is what is now being assumed.

Conditions (6) become:

$$\left. \begin{aligned} \frac{v_1}{V_1} k_1 + \frac{v_2}{V_2} k_2 &> k_1 + k_2, \\ \frac{V_1}{v_1} k_1 + \frac{V_2}{v_2} k_2 &> k_1 + k_2. \end{aligned} \right\} \quad (7)$$

In Levene's notation,  $v_1 = v_2 = 1$ ,  $V_1 = w_1$  and  $V_2 = w_2$ , and  $c_i = k_i/(k_1 + k_2)$ . Hence condition (7) becomes

$$\sum c_i w_i > 1, \quad \sum \frac{c_i}{w_i} > 1, \quad (8)$$

which are the conditions for a stable polymorphism for Levene's model with two genotypes (dominance, or haploid).

Case 3(c). Carrying capacity inversely related to intrinsic fitness

Assume

$$\begin{aligned} k_1 &= \frac{b_1}{v_1^\alpha}, & k_2 &= \frac{b_2}{v_2^\alpha}, \\ K_1 &= \frac{b_1}{V_1^\alpha}, & K_2 &= \frac{b_2}{V_2^\alpha}, \end{aligned}$$

where  $b_1$  and  $b_2$  are constants. Then, if  $\alpha > 0$ , the higher the intrinsic fitness of a genotype the lower is its carrying capacity (see Fig. 2c). (The special cases  $\alpha = -1$  and  $\alpha = 0$  yield cases 3(a) and 3(b) respectively.) Conditions (6) become

$$\frac{v_1^{1+\alpha}}{v_2^{1+\alpha}} < \frac{b_1(v_1 - V_1)}{b_2(V_2 - v_2)} < \frac{V_1^{1+\alpha}}{V_2^{1+\alpha}}. \quad (9)$$

In Levene's notation,  $v_1 = v_2 = 1$ ,  $V_1 = w_1$ ,  $V_2 = w_2$  and  $b_i/(b_1 + b_2) = c_i$ . Then (9) becomes

$$\sum c_i w_i > 1; \quad \frac{c_1(1 - w_1)}{w_1^{1+\alpha}} > \frac{c_2(w_2 - 1)}{w_2^{1+\alpha}}. \quad (10)$$

The first condition is identical to that for Levene's model with fixed carrying capacity, and the second is less restrictive (for  $\alpha$  positive). Fig. 3 compares the range of niche sizes compatible with polymorphism for Levene's model, and for the

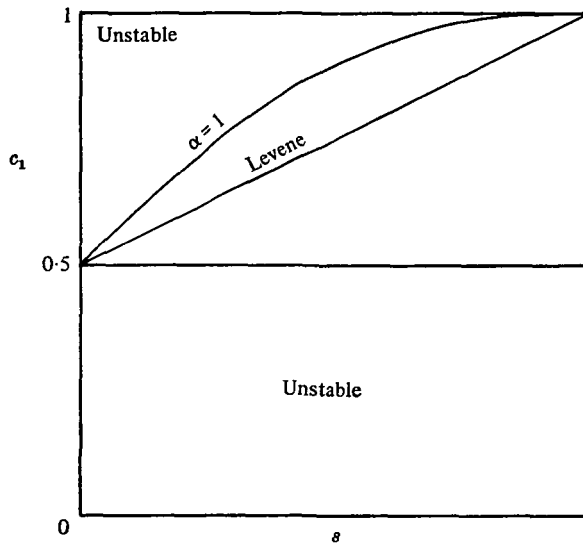


Fig. 3. Range of niche sizes,  $c$ , for which polymorphism is stable, as a function of the selective advantage,  $s$ . The values are for a 2-niche, two-phenotypes case, for Levene's model, and for a model with the carrying capacity inversely proportional to intrinsic fitness ( $\alpha = 1$ ).

present model with  $\alpha = 1$ . It is assumed that  $w_1 = 1 + s$ ,  $w_2 = 1 - s$ . For small  $s$ , the range of niche sizes is approximately twice as broad for the present model.

*Case 4. High density: intrinsic fitnesses of genotypes identical*

Assume  $ax + AX \gg 1$  in both niches and  $v_1 = V_1$ ,  $v_2 = v_2$ .

Conditions (6) become

$$\begin{aligned} \frac{V_1}{A_1} + \frac{V_2}{A_2} &> \frac{V_1}{A_1} + \frac{V_2}{A_2}, \\ \frac{V_1}{a_1} + \frac{V_2}{a_2} &> \frac{V_1}{a_1} + \frac{V_2}{a_2}. \end{aligned} \quad (11)$$

These inequalities can be met if the terms  $a_1$ ,  $a_2$ ,  $A_1$ ,  $A_2$  measuring the self-limiting effects of each genotype are greater than  $a'_1$ ,  $a'_2$ ,  $A'_1$ ,  $A'_2$  measuring the effects of each genotype on its competitor (it is not necessary for all four comparisons to go this way, but the overall effect must exist). If differences of this kind exist, it is possible to have stable polymorphism in a single niche. The biological significance of this is discussed in the next section.

Before leaving this class of model, it is worth asking whether a temporal fluctuation in relative niche sizes is likely to make the polymorphism more robust. We answer this question only for the two-niche Levene model shown in Fig. 3, but it can be shown that a similar conclusion holds for the general diploid Levene model. Thus we assume the fitness constant over time, at  $1 + s : 1$  in one niche and  $1 - s : 1$  in the other. We assume that in alternate generations the relative niche

sizes are  $C_1:1-C_1$  and  $C_2:1-C_2$  (note that  $C_1$  and  $C_2$  are now the sizes of the same niche in alternate generations). The conditions for a protected polymorphism (Hoekstra, 1978) are, for the dominant case,

$$\left(\frac{C_1}{1+s} + \frac{1-C_1}{1-s}\right) \left(\frac{C_2}{1+s} + \frac{1-C_2}{1-s}\right) > 1$$

and 
$$C_1(1+s) + (1-C_1)(1-s) + C_2(1+s) + (1-C_2)(1-s) > 2. \tag{12}$$

For the haploid case, the first condition remains the same, and the second becomes

$$[C_1(1+s) + (1-C_1)(1-s)] [C_2(1+s) + (1-C_2)(1-s)] > 1. \tag{13}$$

Conditions (12) for the dominant are slightly less restrictive. It can be shown that they are somewhat more stringent than those for the Levene model. Thus for (12) to hold, it is necessary but not sufficient that

$$\frac{1}{2} < \frac{C_1 + C_2}{2} < \frac{1+s}{2},$$

whereas for Levene’s model it is sufficient that

$$\frac{1}{2} < c < \frac{1+s}{2}.$$

### 3. CLARKE’S MODEL OF DENSITY-DEPENDENT SELECTION

Clarke (1972) considers competition between two genotypes, say  $g$  and  $G$ . If the numbers of zygotes present are, respectively,  $x$  and  $X$ , he assumes fitnesses:

$$W(g) = \frac{k_1 w_1}{k_1 + w_1 x + \alpha w_2 X}; \quad W(G) = \frac{k_2 w_2}{k_2 + \beta w_1 x + w_2 X}. \tag{15}$$

These are identical to our equation (2), with  $w_1 = v$ ,  $w_2 = V$ ,  $k_1 = k$ ,  $k_2 = K$ ,  $\alpha = A'k/V$  and  $\beta = a'K/v$ . Thus Clarke is considering two genotypes competing in a single niche. Note that one does not obtain equations of this form if one calculates average fitnesses in two niches from (4). In our notation, Clarke’s necessary conditions for a stable equilibrium (his equations 16 and 17) become

$$\frac{A'}{A} < \frac{v-1}{V-1}; \quad \frac{a'}{a} < \frac{V-1}{v-1}. \tag{16}$$

For the special case,  $v = V$ , this reduces to  $A' < A$  and  $a' < a$ . This says that each species has a greater limiting effect on itself than on its competitor. How could this be so for competition in a single niche? The simplest interpretation is that the niche contains two resources, and that the two genotypes take these resources in different proportions.

This point seems to have been seen clearly by the founding fathers of mathematical ecology. Thus Clarke’s use of an expression of the form of (2) and (15) was, intentionally, related to the equations

$$\left. \begin{aligned} dx/dt &= vx(1-ax-A'X), \\ dX/dt &= VX(1-a'x-AX), \end{aligned} \right\} \tag{18}$$

but he modified these equations to prevent the occurrence of negative fitnesses. Volterra (1927) used equations (18) to describe competition between two species. However, he explicitly referred to competition for a single resource, and accordingly put  $a/A' = a'/A$ . With this restriction no equilibrium exists with both species present and, equivalently, Clarke's conditions (16) cannot be satisfied. Lotka (1932) pointed out that Volterra's restriction implies 'that the two species consume one and the same food materials, or, if they consume a mixed diet, that the proportion of each ingredient of the diet which they consume is the same for both species'. He showed that, if the restriction is relaxed, stable coexistence is possible.

#### 4. THE MODEL OF STEWART & LEVIN

Stewart & Levin (1973) considered a model in which two 'species' can coexist in a single type of niche on a single resource. The two species are supposed to have population growth rates which depend on resource concentration as shown in Fig. 4. If the rate of supply of substrate is constant (as, for example, in a chemostat) then coexistence is impossible. One or other species will win, depending on whether the equilibrium resource level is above or below the critical value  $R_c$ . Suppose, however, that the two species enter a 'patch' with a high resource concentration, and reproduce there without further addition of resource until it is exhausted. The species then colonize a new patch with high resource concentration, and the procedure is repeated indefinitely. Stewart & Levin show that there is a range of initial resource concentrations for which stable coexistence is possible. De Jong (1976) has reached the same conclusion for a similar model.

Clarke & Allendorf (1979) and Clarke (1979) have recently suggested that this process is an important one in maintaining enzyme polymorphism. They argue that, if two allozymes exist, their kinetics are likely to be such that the relative velocities of the reactions catalysed by them vary with substrate concentration. They do not in fact claim that the velocities would cross over, as in Fig. 4, but they do imply that the fitnesses would do so. How plausible is this argument?

There are obvious difficulties in applying Stewart & Levin's model directly to enzyme polymorphism. Thus in their model the reproductive rate of individuals depends on substrate concentration, and substrate concentration in turn depends on the abundance of the two kinds of individual. It is not clear how far their conclusions can be transferred, for example, to competition between *Drosophila* larvae. However, our purpose here is to ask how far various models escape from the objection that polymorphism can be maintained only for a narrow range of environmental conditions. We will argue that Stewart & Levin's model does not escape this objection, and hence that Clarke & Allendorf's suggestion does not do so either.

Stewart & Levin assume that growth rate is proportional to  $a_i r / (r + b_i)$ , where  $r$  is concentration and  $a_i$ ,  $b_i$  are constants for the  $i$ th species. They consider a particular numerical case of two species, with  $a_1 = 10$ ,  $a_2 = 1$ ,  $b_1 = 5$ ,  $b_2 = 1/20$ . Thus growth rates at low concentration,  $a/b$ , differ by a factor of 10, and at high



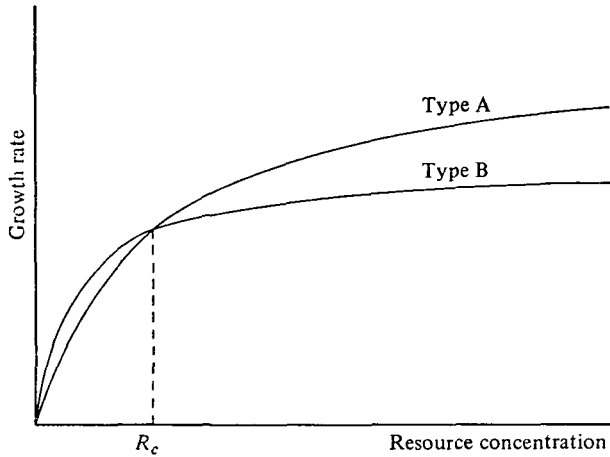


Fig. 4. Relation between population growth rate and resource concentration assumed by Stewart & Levin (1973). The curves are drawn such that they also represent reaction velocity against substrate concentration of two allozymes A and B with the following kinetic properties:  $Km_A = 3 \times 10^{-4}$  M;  $Km_B = 10^{-4}$  M;  $V_{maxA} = \frac{3}{2} V_{maxB}$ .

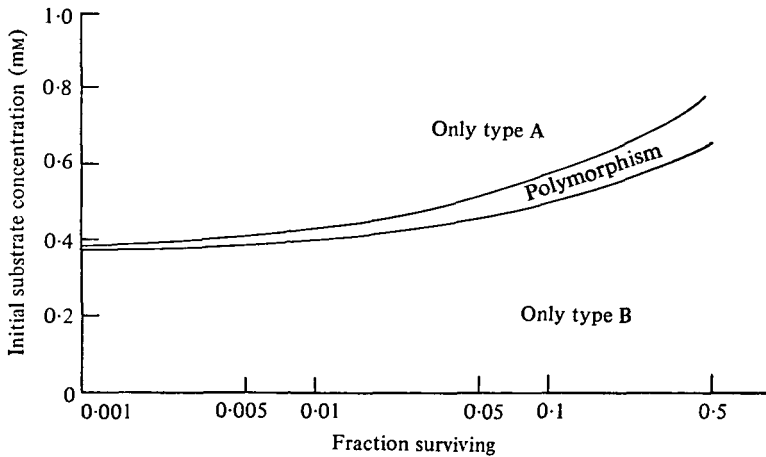


Fig. 5. Range of initial concentrations for which polymorphism is stable in Stewart & Levin's (1973) model, as a function of the fraction of individuals emerging from one patch which survive to enter the next. Population growth rates are as in Fig. 4.

concentrations by a factor of 10 in the opposite direction. Even with this very extreme difference, the range of initial resource concentrations for which co-existence is possible is relatively narrow, the upper and lower bounds having a ratio of from 1.2 to 1.6 depending on another system parameter. With more realistic values, the range is very narrow, as illustrated in Fig. 5.

### 5. THE MODEL OF GILLESPIE

Gillespie (1976, 1977) has proposed a model which differs from the others described in this paper in assuming the heterozygote at a locus to be pheno-

Table 1. Assumptions of Gillespie's (1976) model

Genotype	$A_1 A_1$	$A_1 A_2$	$A_2 A_2$
Enzyme activity	$x_1$	$\frac{x_1 + x_2}{2}$	$x_2$
Fitness	$\phi(x_1)$	$\phi\left(\frac{x_1 + x_2}{2}\right)$	$\phi(x_2)$
Fitness in a specific niche	1	$1 - hs$	$1 - s$

$$\text{where } \phi(x) = \frac{(1 + \alpha)x}{\alpha + x}$$

Table 2. Fitnesses for Gillespie's (1976) model

Genotype ...	$A_1 A_1$	$A_1 A_2$	$A_2 A_2$
Enzyme activity	$m_1$	$\frac{m_1 + m_2}{2}$	$m_2$
Enzyme activity in $i$ th niche	$1 - \epsilon_i$	$\frac{2 - \epsilon_i + \delta_i}{2}$	$1 + \delta_i$
Fitness	$\frac{(1 - \epsilon_i)[2 - r(\epsilon_i - \delta_i)]}{(1 - r\epsilon_i)[2 - (\epsilon_i - \delta_i)]}$	1	$\frac{(1 + \delta_i)[2 - r(\epsilon_i - \delta_i)]}{(1 + r\delta_i)[2 - (\epsilon_i - \delta_i)]}$

$$\text{where } r = \frac{1}{1 + \alpha}$$

typically intermediate between the two homozygotes. His assumptions are shown in Table 1.

Thus he supposes enzyme activities to be exactly additive, but the function  $\phi$  connecting fitness and activity to be convex. For the particular function chosen,  $h = \alpha / (2\alpha + s)$ . Thus in every niche the heterozygote is fitter than the arithmetic mean of the two homozygotes. If in some niches one homozygote is fitter, and in other niches the other, this can give rise to a stable polymorphism.

Gillespie (1976) derives conditions for a stable polymorphism which are, (as will be shown below) identical to those given by Levene (1953), namely

$$\sum \frac{c_i}{V_i} > 1 < \sum \frac{c_i}{W_i}, \tag{19}$$

where  $V_i, 1, W_i$  are the fitnesses of  $A_1 A_1, A_1 A_2, A_2 A_2$  in the  $i$ th niche and  $c_i$  is the proportion of the population produced from the  $i$ th niche.

Table 2 gives the enzyme activities and fitnesses for Gillespie's model. Gillespie makes the following additional assumptions:

(i) Variation in activity levels is small; hence third and higher order terms in  $\epsilon$  and  $\delta$  can be ignored.

(ii) Mean activities  $\bar{m}_1$  and  $\bar{m}_2$  are close to unity; hence  $\text{var}(m_1) \simeq \sum c_i \epsilon_i^2$  and  $\text{var}(m_2) \simeq \sum c_i \delta_i^2$ .

(iii)  $\text{var}(m_1) = \text{var}(m_2)$ . Hence  $\sum c_i \epsilon_i^2 = \sum c_i \delta_i^2$ , and  $\text{var}(\epsilon_i - \delta_i) \simeq \sum c_i (\epsilon_i - \delta_i)^2$ ; also,  $\text{var}(\epsilon_i - \delta_i) = \text{var}(\epsilon_i) + \text{var}(\delta_i)$ .

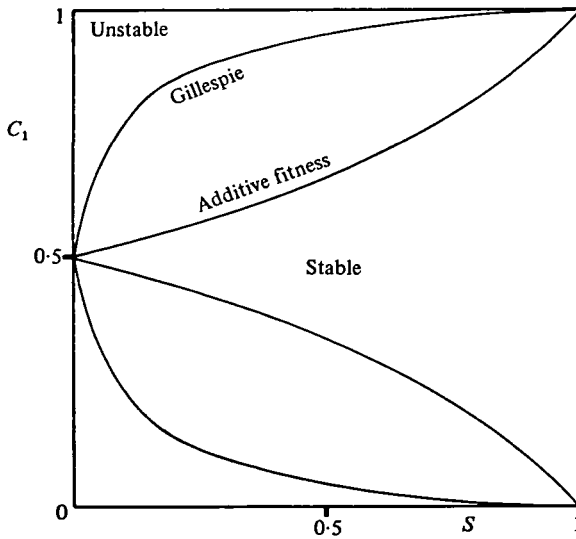


Fig. 6. Range of niche sizes,  $c$ , for which polymorphism is stable as a function of the selective advantage,  $s$ . The values are for a 2-niche version of Gillespie's (1976) model, and for a case in which the fitness of the heterozygote is equal to the arithmetic mean of the fitnesses of the homozygotes.

Hence

$$\sum c_i \epsilon_i \delta_i \simeq 0.$$

With these approximations, and the fitnesses of Table 2, (17) reduces to

$$|\sum c_i \epsilon_i + \sum c_i \delta_i| < \sum c_i \epsilon_i^2, \tag{20}$$

which is identical to Gillespie's condition,  $|\Delta m| < \delta m^2$ , where  $\Delta m$  is the difference between the mean enzyme activities of the two homozygotes, and  $\delta m^2$  the variance of enzyme activity for a given homozygote between niches.

This model is much less sensitive to fluctuations in relative niche size than those already discussed. This is best seen considering a two-niche model with fitnesses:

	$A_1 A_1$	$A_1 A_2$	$A_2 A_2$
Fitness in niche 1	1	$1 - hs$	$1 - s$
Fitness in niche 2	$1 - s$	$1 - hs$	1

Using (19), we can calculate the range of niche sizes for which polymorphism is stable. The critical upper value of niche size is

$$c = \frac{\alpha + s}{2\alpha + s - \alpha s}, \tag{21}$$

the critical lower value being  $1 - c$ . Using data from Crow & Temin (1964) and Mukai *et al.* (1972), Gillespie (1977) estimates a reasonable value of  $\alpha$  to be 0.05, which gives the range of niche sizes shown in Fig. 6.

Even with a value of  $s$  as low as 0.01, polymorphism is stable for  $0.55 > c > 0.45$ .

Table 3. *Range of relative niche sizes for which a protected polymorphism exists, for various models, for a 2-niche case with symmetric fitnesses*

Selective advantage $s$ ...	0.01	0.1
Levene model with dominance	0.50–0.505	0.5–0.55
Carrying capacity inversely proportional to intrinsic fitness	0.50–0.51	0.50–0.60
Levene model with additive fitnesses	0.4975–0.5025	0.474–0.526
Gillespie's model ( $\alpha = 0.05$ )	0.45–0.55	0.23–0.77

However, it is important to remember that polymorphism does depend on the non-linear relation between gene dosage and fitness. If we assume  $h = \frac{1}{2}$  (additive fitnesses), then the permissible range of niche sizes (Fig. 6) is similar to the range with dominance (Fig. 3).

## 6. CONCLUSIONS

The model analysed in section 2 confirms the conclusion that niche differentiation on its own can maintain polymorphism only if selective advantages at a single locus are large. With moderate selective advantages (10% or less) the range of relative niche sizes over which polymorphism is stable is very narrow. The most favourable case is that in which the carrying capacities of two genotypes in a niche are inversely related to their intrinsic fitnesses when rare in the same niche. Some results are summarized in Table 3.

This type of model is more favourable for the maintenance of polymorphism if there is habitat selection, either because individuals actively seek out a particular niche, or because niche size is large relative to the dispersal distance of individuals.

The density-dependent model proposed by Clarke (1972) refers to competition within a single niche. It can maintain polymorphism if the niche contains two resources, and if the different genotypes consume those resources in different amounts, but apparently not otherwise.

The model of Stewart & Levin (1973) can maintain polymorphism only if the resource concentration fluctuates widely in time, and then only if the concentration is very precisely adjusted. The model seems even less robust than Levene's. The suggestion by Clarke & Allendorf (1979) that enzyme polymorphism may be maintained because of the kinetic properties of enzymes is a form of this model, and therefore seems unlikely to be of wide relevance.

The only model considered here which escapes the dilemma of requiring either intense selection or a nicely adjusted environment is that proposed by Gillespie (1976, 1977). The robustness of this model depends essentially on the assumption that the fitness of a genotype is a convex function of its enzyme activity, an assumption which can give rise to a net (arithmetic mean) heterozygous advantage.

We thank Dr Brian Charlesworth for pointing out to us that Gillespie's (1976) conditions for polymorphism can be derived from Levene's (1953) conditions.

While this work was carried out R. Hoekstra was in receipt of a stipend from the Netherlands Organisation for the Advancement of Pure Research (Z.W.O.).

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