J. theor. Biol. (1985) **116**, 479–493

Density-independent Mortality, Non-linear Competitive Interactions, and Species Coexistence

ROBERT D. HOLT

Department of Systematics and Ecology, Museum of Natural History, University of Kansas, Lawrence, Kansas 66045, *U.S.A.*

(*Received* 1 *August* 1984)

The influence of density-independent mortality on the coexistence of competing species is discussed. It is shown that equivalent mortality—an increase in mortality which reduces the intrinsic growth rate of all species by an equal proportional amount—does not affect the conditions for coexistence in the *n*-species Lotka–Volterra model. In this model the per capita growth rate of each species declines linearly with the population size of each competitor. In more general models, which incorporate non-linearities in competitive interactions, it is shown that equivalent mortality may substantially change the conditions for competitive coexistence. A graphical model of the conditions for invasion shows that equivalent mortality can either reduce or increase the likelihood of coexistence for competing species, depending upon the kind of non-linearity built into the competition model. Both outcomes are illustrated by the Ayala–Gilpin–Ehrenfeld competition model, which incorporates a non-linear term for intraspecific competition.

1. Introduction

The interplay of predation and competition in determining community composition and structure has long intrigued ecologists (e.g. Slobodkin, 1961; Paine, 1966; Cramer & May, 1971; Connell, 1975; Yodzis, 1978). In recent years, it has become clear that there is no single, simple rule that can predict the influence of predation upon the coexistence of competitors in all circumstances. Previous theoretical work has shown that such predictions require an understanding of, (among other factors) the degree to which predators are limited by the availability of their prey (Holt, 1977; Noy-Meir, 1981); the predator's preference for, and functional response to, each prey type (Lubchenco, 1978; Comins & Hassell, 1976); and the dynamics of the resource base over which competition is occurring (Abrams, 1977; Tilman, 1982). Most theoretical studies of the combined effects of predation and competition have been based upon the traditional Lotka–Volterra competition model. This model assumes that the per capita growth rate of each

0022-5193/85/200479+15 $03.00/0

species declines linearly with the density of both itself and its competitors. A growing body of evidence and theoretical arguments, however, support the idea that competitive effects are often non-linear functions of population density (Schoener, 1974; Smith-Gill & Gill, 1978; Pomerantz, Thomas & Gilpin 1980; Abrams, 1983). In this paper, I demonstrate that non-linearities in competitive interactions may substantially alter the way in which the outcome of competition is affected by the level of mortality.

We will assume that the direct effect of predation upon a set of competing species can be approximated by an increase in density-independent mortality. We will pay particular attention to "equivalent mortality", which is defined to be a uniform increase in density-independent mortality on competing species with equal intrinsic growth rates or, more broadly, as mortality that reduces the intrinsic growth rate of each species by an equal proportional amount (Van Valen, 1974). Several authors have claimed that equivalent mortality does not affect the conditions for competitive coexistence (Van Valen, 1974; Harper, 1977, p. 744; May, 1977; Levin, 1981). It is shown below that this claim is true for n species competing in accord with the Lotka–Volterra model, but it is not true in more general competition models with non-linear intraspecific or interspecific interactions. We demonstrate this first for a simple, graphical model, and then for a specific non-linear competition model proposed by Ayala, Gilpin & Ehrenfeld (1973).

2. Density-independent Mortality in the Lotka–Volterra Model

With an additional term for density-independent mortality, the familiar n-species Lotka–Volterra competition model is

$$\frac{dN_i}{dt} = r_i N_i\left(1 - \sum_{j=1}^{n} \frac{\alpha_{ij} N_j}{K_i}\right) - m_i N_i, \qquad i = 1, \cdots, n. \tag{1}$$

The parameters are r_i, the intrinsic growth rate of species i; K_i, its carrying capacity; m_i, a rate of density-independent mortality experienced by species i stemming from a particular mortality agent; and α_{ij}, the competition coefficient. The competition coefficient measures the fractional reduction in the per capita growth rate of species i produced by a small increase in the density of species j, scaled by the corresponding effect of species i upon itself. (By convention, $\alpha_{ii} = 1$.) A number of authors have discussed the two-species version of this model (Slobodkin, 1961; Abrams, 1977; Fenchel & Christiansen, 1976; Kapur, 1980; Levin, 1981; Williams & Banyikwa, 1981), and their conclusions are in broad agreement. Following Williams & Banyikwa (1981), let us define two transformed parameters as follows:

the *effective intrinsic rate of increase*, $e_i' \equiv r_i - m_i$, and the *effective carrying capacity*, $K_i' \equiv K_i(1 - m_i/r_i)$. Substituting these into equation (1) leads to

$$\frac{dN_i}{dt} = e_i' N_i \left(1 - \sum_{j=1}^{n} \frac{\alpha_{ij} N_j}{K_i'}\right), \qquad i = 1, \cdots, n,$$

the usual form of the n-species Lotka-Volterra model (Pianka, 1981). Species i when alone grows at the per capita rate e_i' and equilibrates at K_i'.

Thus, any analysis of the usual Lotka-Volterra model (e.g. the telegraphic treatment in Strobeck (1973)) has implicitly already incorporated the effects of density-independent mortality; to predict the outcome of a change in density-independent mortality rates, one merely replaces the parameters r_i and K_i with their respective effective parameters, e_i' and K_i'. This transformation may often simplify analyses of how mortality alters coexistence. For instance, the stable coexistence of two competing species requires that

$$\alpha_{21}^{-1} > K_1'/K_2' > \alpha_{12}. \tag{2}$$

If $\alpha_{12}\alpha_{21} > 1$, it is clearly impossible for *any* pattern of density-independent mortality to change exclusion into coexistence.

Define *equivalent mortality* to be mortality that reduces the intrinsic growth rate of each community member by the same proportional amount: $m_i = cr_i$ for $0 \leq c < 1$. This implies that $e_i' = (1 - c)r_i$. For two competitors, as noted by Abrams (1977) and Williams & Banyikwa (1981), if $r_1/m_1 = r_2/m_2$ (i.e. mortality is equivalent), then $K_1'/K_2' = K_1/K_2$, and the condition for coexistence is independent of the level of equivalent mortality.

This conclusion also holds for n species in the general Lotka-Volterra model,

$$\frac{dN_i}{dt} = N_i\left(r_i + \sum_j a_{ij} N_j\right) - m_i N_i, \qquad i = 1, \ldots, n. \tag{3}$$

Assume that without the additional mortality the community exists at a feasible (Roberts, 1974), stable point equilibrium. This equilibrium is determined from the matrix equality $\hat{\mathbf{N}} = -A^{-1}\mathbf{r}$, where $\hat{\mathbf{N}}$ is a column vector of equilibrium densities, $\mathbf{r}$ is the corresponding vector of intrinsic growth rates, and A is a matrix of interaction coefficients. With the added mortality, the new vector of equilibrial densities is $\mathbf{N}^* = -A^{-1}(\mathbf{r} - \mathbf{m})$ ($\mathbf{r} - \mathbf{m}$ is a column vector in which $r_i - m_i$ is the ith element.) With equivalent mortality, $r_i - m_i = r_i(1 - c)$, hence $\mathbf{N}^* = -A^{-1}\mathbf{r}(1 - c) = \hat{\mathbf{N}}(1 - c)$; the equilibrial densities are all reduced by a common proportional amount. (We assume

$c<1$.) With the transformation of variables $t' \equiv t(1-c)$ and $X_i \equiv N_i/\hat{N}_i(1-c)$, model (3) becomes

$$\frac{dX_i}{dt'} = X_i\left(r_i + \sum_j a_{ij}\hat{N}_j X_i\right), \qquad i = 1, \ldots, n.$$

In this representation of the model, the parameter c no longer explicitly appears. This re-scaling of variables shows that varying the level of equivalent mortality cannot alter qualitative properties of the model, and in particular cannot affect the existence and stability of its equilibrial states. Equivalent mortality thus does not change the deterministic conditions for the coexistence of n competing species, given that the Lotka–Volterra model describes their interactions.

High levels of equivalent mortality, however, may make the community more likely to lose species due to chance events. An increase in c reduces all species' equilibrial densities uniformly, heightening the probability of extinctions from demographic stochasticity (Leigh, 1981). The community also requires more time to recover from disturbances. Let λ be the dominant eigenvalue of the Jacobian matrix of the original community. With the added mortality, perturbations away from the equilibrium dampen at a rate scaled by the dominant eigenvalue, which is $\lambda(1-c)$. The characteristic time required for the community to return to equilibrium following a disturbance thus increases by a factor $(1-c)^{-1}$. This has the effect of increasing the probability of extinction for those populations perturbed to low densities. Despite these caveats, in the Lotka–Volterra model equivalent mortality does not affect the conditions for coexistence in any systematic way.

3. Density-Independent Mortality and Robust Coexistence: A Graphical Model

Many ecologists have criticized the Lotka–Volterra model for its assumption of linear per capita competitive interactions (e.g. Wilbur, 1972; Ayala *et al.*, 1973; Neill, 1974; Schoener, 1974; Smith-Gill & Gill, 1978; Abrams, 1980). In thinking through the consequences of predation and other mortality factors for species coexistence, it is clearly desirable to examine models of competition other than the Lotka–Volterra. In this section we develop a simple graphical technique for analyzing the effect of uniform mortality on competitive coexistence. The models we examine all have the following form

$$\frac{dN_1}{dt} = N_1 f_1(N_1, N_2) - m_1 N_1, \quad \text{and} \quad \frac{dN_2}{dt} = N_2 f_2(N_1, N_2) - m_2 N_2. \quad (4)$$

The per capita growth functions $f_i(N_1, N_2)$ encapsulate the consequences of within- and between-species competition. For simplicity, we assume that density-dependence is negative at all densities, i.e. $\partial f_i/\partial N_j < 0$ $(i, j = 1, 2)$. The intrinsic rate of growth of species i is defined to be $r_i \equiv f_i(0, 0)$.

A general analysis of model (4), although in principle straightforward, in practice may be complex because of the existence of multiple interior equilibria at which both N_1^* and N_2^* are positive. (The asterisks denote equilibrial values.) Here, we tackle the more modest task of analyzing the influence of density-independent mortality on mutual invasibility. We will say that a community is characterized by "robust coexistence" if each species can in turn increase when it is rare and its competitor is at carrying capacity. The equilibrial community should surely contain both species if each can invade when rare. An invasibility criterion thus should be sufficient for predicting long-term species coexistence.

Species 1, when alone, equilibrates at a density determined from solving $f_1(N_1^*, 0) = m_1$. The quantity N_1^* is the effective carrying capacity, K_1', of the environment for species 1; K_1' is a decreasing function of m_1. If species 2 is introduced at sufficiently low density, as a first-order approximation we can assume that N_1^* is unchanged and N_2 nearly zero. We then determine whether the per capita growth rate of species 2, $f_2(K_1', 0) - m_2$, is positive. If so, species 2 can invade at this particular level of mortality. Our concern will be with how this simple criterion for invasion changes with changing patterns of mortality.

A given mortality agent may have both direct and indirect effects on the rate of increase of species 2. The direct effect, measured by $-m_2$, clearly inhibits invasion, whereas the indirect effect, stemming from reduced numbers of species 1 resulting from the same mortality agent, is to ease invasion by reducing interspecific competition experienced by species 2. To analyze the relation between a given mortality level and robust coexistence, we must also examine the reciprocal case in which species 1 invades with species 2 at its equilibrial density (determined from $f_2(0, N_2^*) = m_2$). We emphasize the special case of uniform mortality ($m_1 = m_2 \equiv m$) upon competing species with equal intrinsic growth rates—i.e. equivalent mortality.

To introduce the graphical model, we use as a simple example the Lotka–Volterra competition model. In Fig. 1(a) the per capita growth rate of species 1, $f_1(N_1, 0)$ is plotted as a function of N_1, together with the growth rate of species 2 against N_1 when species 2 is rare (i.e. $N_2 \equiv 0$, so $r_2' \equiv f_2(N_1, 0)$). The effect of a uniform mortality agent upon both species is represented by the horizontal line marked m_0. The difference between f_1 and m_0 is the realized or effective growth rate of species 1; the density at which these lines cross is the effective carrying capacity of the environment

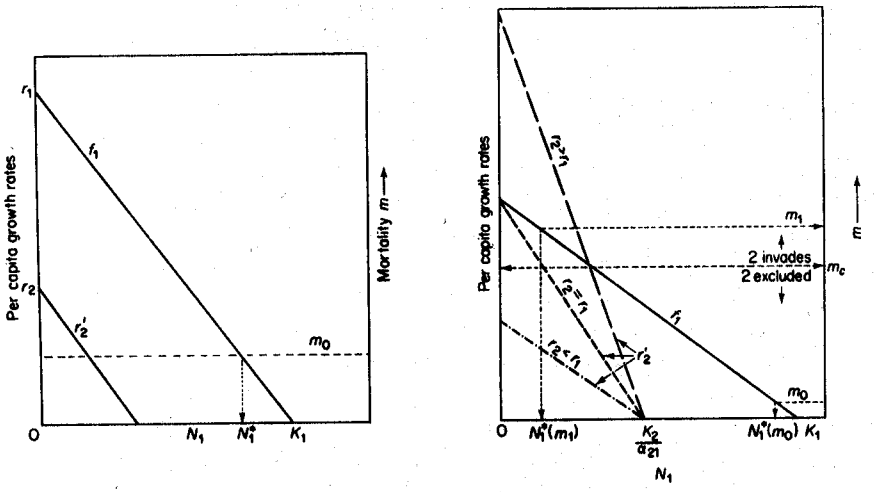

FIG. 1. (a) Growth rates of two competitors, as a function of the density of competitor 1. Competitor 2 is assumed to be rare enough that its competitive effects, both upon itself and competitor 1, can be ignored. $f_1 \equiv f_1(N_1, 0)$, $r_2' \equiv f_2(N_1, 0)$. The linear per capita growth rates are those of the Lotka-Volterra model. (b) Per capita growth rates of two competitors. Three different values for r_2 are depicted. Species 2 is excluded when mortality equals m_0, but can invade when the rate of mortality equals m if $r_2 > r_1$.

for species 1, at the given mortality rate. In like manner, the difference between f_2 and m_0 is the realized growth rate of species 2 when it is invading.

When $f_2 - m < 0$ at N_1^*, species 2 is excluded. But N_1^* is found from $f_1 - m = 0$. The criterion for invasion at a particular m is simply that $f_2 - m > 0 = f_1 - m$, or $f_2 > f_1$ at N_1^*. In Fig. 1(a), in the absence of the additional mortality, species 1 equilibrates at K_1, and species 2 is excluded; the same is true for $m > 0$. In Fig. 1(b), the lines correspond to the cases $r_2 < r_1$, $r_2 = r_1$, and $r_2 > r_1$. For the first two cases, no level of added mortality will allow invasion by species 2. For the third case, invasion is possible if $m > m_c$. The geometric condition for this to occur is for the two growth curves to cross at some positive density. When this happens, there is a range of densities of the resident species for which it has the higher per capita rate of growth, and there is another range of densities in which the invader has the higher growth rate. With the linear per capita growth curves assumed in the Lotka-Volterra model, this crossing of growth curves requires $r_2 > r_1$.

To complete the analysis of robust coexistence, we must in turn examine the relation between mortality and invasion by species 1, given that species 2 is at equilibrium. An example is shown in Fig. 2(a). Along a gradient of increasing mortality there is a zone with species 1 alone, abutting a zone with both species coexisting, followed by a zone with just species 2, and

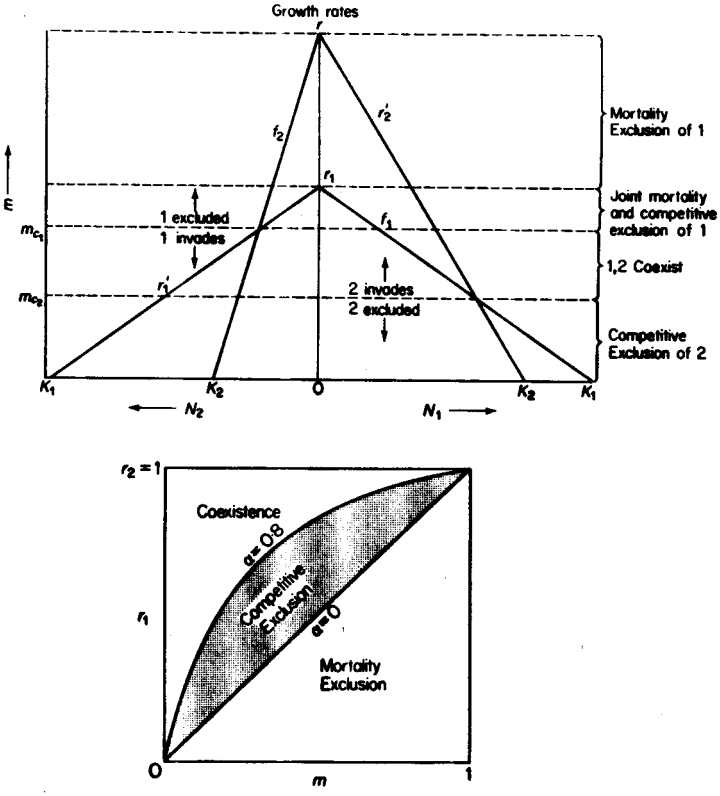

FIG. 2. (a) Changes in invasibility for two competing species along a gradient in density-independent mortality (see text). (b) Conditions for coexistence and exclusion. The two competitors have equal K's, and the competitive interaction is symmetrical, but species 2 has the higher intrinsic growth rate. Exclusions may result from this difference in r.

finally a zone with neither species. The zone occupied by species 2 alone can be further sub-divided into a zone where both the mortality factor and competition from species 2 are required to exclude species 1, and a zone where mortality alone suffices. This points out one potential pitfall in interpreting certain kinds of field experiments. The removal of a given species, followed by the invasion of a species that was previously absent, does provide evidence for the existence of interspecific competition. However, a full explanation for the factors leading to exclusion of the second species requires consideration of the mortality factors affecting both species, too. For instance, in model (1) let $K_1 = K_2$ and $\alpha_{12} = \alpha_{21} \equiv \alpha$, but $r_1 < r_2$. Without loss of generality we can let $r_2 = 1$. Because $K_2' > K_1'\alpha$,

species 2 should be present for all $m > 1$. Species 1 will also be present if

$$r_1 > \frac{m}{1-\alpha+\alpha m}.$$

Figure 2(b) plots this invasibility condition for $\alpha = 0$ and $\alpha = 0{\cdot}8$. When $r_1 < m$, the mortality agent alone can exclude species 1. For $\alpha = 0{\cdot}8$, in the hatched region species 2 competitively excludes species 1: if we remove species 2, species 1 invades. But the competitive superiority of species 2 rests entirely upon its superior capacity to withstand the mortality factor. Were the agent of this mortality removed, and species 2 not, species 1 could invade and both species stably coexist. Field studies of competition or other interspecific interactions should always be interpreted in the context of the full constellation of environmental factors that can influence birth or death rates in the interacting species.

We now return to non-linear competition models. Non-linearities may occur either in the intraspecific competitive interaction, or in interspecific competition, or in both. In Fig. 3 we show two examples of competitors with linear interspecific interactions and equal intrinsic growth rates but with non-linear intraspecific interactions. As before, mortality affecting both species equally is represented by a horizontal line. These graphical examples show that an increase in mortality, applied uniformly to competitors with equal r's, may sometimes permit invasion that otherwise is prevented (Fig. 3(a)), or preclude invasion that otherwise would have taken place (Fig. 3(b)). What these figures have in common with the case $r_2 > r_1$ in Fig. 1(b) is that the growth curves intersect at some $N_1 > 0$. In the Lotka–Volterra model, such an intersection necessarily implies that $r_2 \neq r_1$, but this is not required in more general competition models. In other words, equivalent mortality *can* influence competitive coexistence if the effect of competition is a non-linear function of density.

If competition is symmetrical between the two species, we could re-label these curves so as to display invasion by species 1. For the competitive model depicted in Fig. 3(a), increasing uniform mortality allows the coexistence of competitors that otherwise would have shown contingent dominance (i.e. a priority effect). By contrast, in the competitive situation depicted in Fig. 3(b), there is coexistence at low levels of mortality, and contingent competition at high levels of mortality.

With non-linear competition, we could observe complex patterns of relationship between the level of mortality and coexistence. Let us assume that competition is highly asymmetrical, so that species 2 has a negligible competitive impact on the dynamics of species 1, but is itself strongly affected (i.e. amensalism; for examples, see Lawton & Hassell, 1981). The

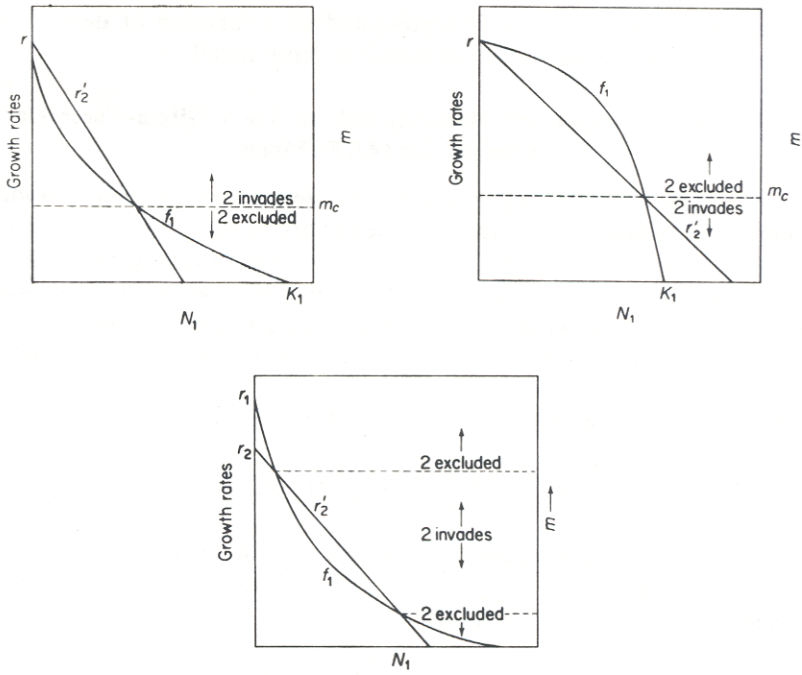

FIG. 3. (a) Equal intrinsic growth rates, non-linear per capita growth functions. A uniform increase in density-independent mortality allows species 2 to invade. (b) Equal intrinsic growth rates, non-linear per capita growth functions. A uniform increase in density-independent mortality leads to the competitive exclusion of species 2. (c) Non-linear per capita growth functions. Coexistence requires an intermediate level of density-independent mortality.

coexistence conditions then reduce to conditions for the invasion of species 2. In the example shown in Fig. 3(c), for instance, coexistence rests upon an intermediate level of density-independent mortality; species 2 is excluded by species 1 if mortality is either too high or too low.

Although the specific examples depicted in the figures assume non-linear intraspecific effects and linear interspecific effects, we could just as easily have constructed examples in which the non-linearity resides in the interspecific interaction, or in which both intra- and interspecific competition are non-linear functions of density. The basic conclusion is that in competition models other than the Lotka–Volterra model, equivalent mortality *can* affect species coexistence. However, the character of the effect depends upon the specific details of the model. Using the graphical approach outlined here, one can examine how uniform, density-independent mortality changes the conditions of mutual invasibility for any competition model in which

the competitive interaction is represented as a function of density. It is instructive to consider a specific model in more detail.

4. Density-independent Mortality and the Ayala–Gilpin–Ehrenfeld Competition (AGE) Model

Ayala *et al.* (1973) have proposed a flexible, phenomenological model of competition which can exhibit both behaviors illustrated in Fig. 3. In this model, the effect of interspecific competition on per capita growth is linear, but the effect of intraspecific competition is nonlinear, with the character of the non-linearity being set by a single parameter, θ. This model is often cited as a useful generalization of the traditional Lotka–Volterra model (e.g. Hutchinson, 1978).

With an added mortality term, the AGE model is

$$\frac{1}{N_i}\frac{dN_i}{dt} = r_i\left(1-\left(\frac{N_i}{K_i}\right)^{\theta_i}-\frac{\alpha_{ij}N_j}{K_i}\right)-m_i, \qquad i=1,2.$$

If $m_1 = m_2 = 0$, the conditions for mutual invasibility are the same as in the Lotka–Volterra model: $\alpha_{21}^{-1} > K_1/K_2 > \alpha_{12}$. With the added mortality, species i when alone equilibrates at

$$N_i^* = K_i\left(1-\frac{m_i}{r_i}\right)^{1/\theta_i} \equiv K_i',$$

its effective carrying capacity. Both species are able to increase when rare provided

$$\frac{(1-m_2/r_2)}{\alpha_{21}(1-m_1/r_1)^{1/\theta_1}} > \frac{K_1}{K_2} > \frac{\alpha_{12}(1-m_2/r_2)^{1/\theta_2}}{(1-m_1/r_1)},$$

With uniform mortality on competitors with equal r's ($m_1 = m_2 \equiv m$, $r_1 = r_2 \equiv r$), and $\theta_1 = \theta_2 \equiv \theta$, this condition takes the slightly simpler form

$$\left[\alpha_{21}\left(1-\frac{m}{r}\right)^{(1-\theta)/\theta}\right]^{-1} > \frac{K_1}{K_2} > \alpha_{12}\left(1-\frac{m}{r}\right)^{(1-\theta)/\theta}.$$

Compared to the Lotka–Volterra model, an increase in uniform mortality makes invasion by species 1 more difficult if

$$\left(1-\frac{m}{r}\right)^{(1-\theta)/\theta} > 1 \quad \text{or} \quad \theta > 1,$$

and less difficult if $\theta < 1$. There is a corresponding effect on invasion by species 2. In the AGE model, equivalent mortality may either relax, or tighten, constraints on competitive coexistence.

More generally, in model (4) species 2 invades if $(dN_2/dt)/N_2 = f_2(N_1^*, 0) - m > 0$. A uniform increase in mortality make invasion by species 2 more likely if $\partial/\partial m(f_2 - m) > 0$, or $|\partial N_1^*/\partial m| > |\partial f_2/\partial N_1|^{-1}$. At equilibrium, $f_1(N_1^*, 0) = m$, so

$$\left|\frac{\partial f_1}{\partial N_1}\right|^{-1} = \left|\frac{\partial N_1^*}{\partial m}\right|.$$

We thus see that invasion by species 2 is aided by a uniform increase in mortality if $|\partial f_2/\partial N_1| > |\partial f_1/\partial N_1|$. Similarly, invasion is made more difficult if $|\partial f_1/\partial N_1| > |\partial f_2/\partial N_1|$. If $|\partial f_1/\partial N_1|$ is large, density-independent mortality in species 1 acts in a compensatory fashion. A large change in m is not reflected in a large change in N_1^*, so an invading species experiences more interspecific competition at any given m than if $|\partial f_1/\partial N_1|$ were small.

This model illustrates the limited utility of the concept of the competition coefficient as a universal measure of the magnitude of interspecific competition. The most widely accepted definition of the competition coefficient appears to be as follows. Let

$$a_{ii} = \frac{\partial}{\partial N_i}\left(\frac{1}{N_i}\frac{dN_i}{dt}\right)$$

measure intraspecific density-dependence, and

$$a_{ij} = \frac{\partial}{\partial N_j}\left(\frac{1}{N_i}\frac{dN_i}{dt}\right),$$

interspecific density-dependence, where the derivatives are evaluated at fixed densities (N_i, N_j). Define the competition coefficient to be $\tilde{\alpha}_{ij} = a_{ij}/a_{ii}$. The quantity $\tilde{\alpha}_{ij}$ measures the relative strength of intraspecific to interspecific density-dependence at given densities. In the Lotka–Volterra model either with or without density-independent mortality, $\tilde{\alpha}_{ij} = \alpha_{ij}$ at all densities. In the AGE model,

$$\tilde{\alpha}_{ij} = \alpha_{ij}(K_i^{\theta_i - 1}/\theta_i N_i^{\theta_i - 1}).$$

The competition coefficient varies with the density of species i. This implies that the competition coefficient measured at equilibrium varies indirectly with the intensity of density-independent mortality.

In the general competition model given by equations (4), the competition coefficient $\tilde{\alpha}_{ij}$ is important for the local stability of a joint equilibrium (i.e. both N_1^* and $N_2^* > 0$). Let $F_i \equiv N_i f_i(N_1, N_2) - m_i N_i$, $i = 1, 2$. The local stability of an equilibrium is determined by evaluating the signs of the Jacobian

matrix (May, 1975)

$$\begin{pmatrix} \frac{\partial F_1}{\partial N_1} & \frac{\partial F_1}{\partial N_2} \\ \frac{\partial F_2}{\partial N_1} & \frac{\partial F_2}{\partial N_2} \end{pmatrix},$$

where the derivatives are evaluated at the equilibrium (N_1^*, N_2^*). The eigenvalues are the two roots of the characteristic equation

$$\lambda^2 + \lambda\left(-\frac{\partial F_1}{\partial N_1} - \frac{\partial F_2}{\partial N_2}\right) + \left(\frac{\partial F_1}{\partial N_1}\frac{\partial F_2}{\partial N_2} - \frac{\partial F_1}{\partial N_2}\frac{\partial F_2}{\partial N_1}\right) = 0.$$

Both roots are negative and the equilibrium stable provided the bracketed terms are positive. At equilibrium, $\partial F_i/\partial N_j = N_i\,\partial f_i/\partial N_j < 0$. The necessary and sufficient condition for local stability is thus

$$(\partial f_1/\partial N_1)(\partial f_2/\partial N_2) > (\partial f_1/\partial N_2)(\partial f_2/\partial N_1),$$

which may be re-written

$$1 > \left(\frac{\partial f_1/\partial N_2}{\partial f_1/\partial N_1}\right)\left(\frac{\partial f_2/\partial N_1}{\partial f_2/\partial N_2}\right) \quad \text{or} \quad 1 > \tilde{\alpha}_{12}\tilde{\alpha}_{21}.$$

This shows that competition coefficients determine the stability of a joint competitive equilibrium, given that one exists.

However, away from equilibrium the concept of a competition coefficient appears to lose its usefulness. Consider what happens when species i invades in the AGE model. If $\theta_i < 1$, then $\alpha_{ij} \cong 0$ at $N_i \cong 0$. This seems to say that species i when rare experiences no competition. This, of course, is false, since species i may be competitively excluded if the other species is present in sufficiently high density. In like manner, if $\theta_i > 1$, then $\tilde{\alpha}_{ij} \to \infty$ as $N_i \to 0$. This appears to say that species i will not be able to invade, because competition is so strong upon it, but once again this would be a fallacious conclusion. The notion of a competition coefficient, even when properly generalized to include the dependence of competition upon density, does not capture that aspect of the competitive interaction that is actually important for predicting invasion or exclusion. When a species is rare it mainly experiences density-dependence from the resident species. Its effect upon itself does not matter, for it is so rare that any self-effects are dwarfed by its interaction with the resident.

5. Discussion

In this paper I have shown two things. First, in the Lotka–Volterra model of n-species competition, equivalent mortality (defined as a proportional

reduction in intrinsic growth rates) does not alter the deterministic conditions for the existence and stability of point equilibria. If a mortality factor is to influence species coexistence, then it must be non-equivalent in its effects. Second, even equivalent mortality can change species coexistence if the per capita effects of competitive interactions are non-linear functions of densities. The graphical requirement for this to occur is for the growth curves to intersect in the fashion illustrated in Fig. 3. This can occur because of non-linearities in either intraspecific or interspecific competitive interactions. Abrams (1977) and Hanski (1981) have made similar observations about particular, non-linear models of competition.

Having made this point, it does not follow that equivalent mortality has a *typical* effect upon species coexistence. Whether in any given case an added mortality factor makes coexistence easier to achieve, or more difficult, depends upon the biological details that lead to non-linear competitive interactions. This complicates the task of constructing a general theory for the role of predation in communities (Holt, 1984).

There are good biological reasons to suspect that competitive interactions are often non-linear intra- as well as interspecifically. Schoener (1974) shows that very simple models of exploitative competition can be highly non-linear. Models such as (4) can be thought of as abstractions of more complex consumer-resource models in which resource populations equilibrate sufficiently faster than consumer populations that we can describe interactions between consumers solely in terms of consumer densities (Schaffer, 1981). However, the form of the resulting population growth models will reflect the underlying resource dynamics and consumer utilization strategies. Non-linearities in resource renewal or consumer functional responses typically lead to nonlinear density-dependent effects, both within- and between-species (Abrams, 1980, 1983). Glasser (1978) argues that when consumer populations are rare, and their required resources correspondingly abundant, they may be more discriminate in their resource use than when resources are rare. In other words, resource overlap increases with increasing population density. At low densities there will still be intraspecific competition, but little interspecific competition. This situation matches the density-dependent growth curves of Fig. 3(a). This implies, as noted by Glasser, that predation makes coexistence more likely (barring the effects of over-exploitation explored in Holt (1977, 1984)). However, this prediction may be reversed if species become more similar, rather than more dissimilar, during times of plenty. Schoener (1982) notes that in many empirical studies of temporal variation in resource use, the least overlap is observed during lean periods (seasons or years) and the highest overlap when resources are abundant. If resources are abundant because the population sizes of the

competing species are low, and the low population sizes result from predation, then we might observe an increase in the intensity of interspecific competition with an increase in predation. This scenario fits the growth curves of Fig. 3(b).

Noy-Meir (1981) recently reviewed models of two competing prey species interacting with a predator, and he concluded "Even the simplest model system which combines competitive with predatory interactions shows a rich repertoire of different dynamic behaviours, which all are possible . . ." In the models discussed by Noy-Meir, non-linearities were built into the predator's functional response (e.g. predator satiation or switching) but the competitive interaction was described by the Lotka-Volterra model. Here we have shown that even uniform mortality may produce a great variety of qualitatively different results, depending on the character of the non-linearities in intraspecific and interspecific competitive interactions. This reinforces Noy-Meir's conclusion that a general, predictive theory of multi-species communities is likely to be quite complex, since a wealth of natural history details must be specified before one can make firm predictions about the influence of predation upon species coexistence.

REFERENCES

ABRAMS, P. A. (1977). *Am. Nat.* **111,** 539.
ABRAMS, P. A. (1980). *Theor. Pop. Biol.* **17,** 80.
ABRAMS, P. A. (1983). *Am. Nat.* **121,** 887.
AYALA, F. M., GILPIN, M. E. & EHRENFELD, J. G. (1973). *Theor. Pop. Biol.* **4,** 331.
COMINS, H. N. & HASSELL, M. P. (1976). *J. Theor. Biol.* **62,** 93
CONNELL, J. H. (1975). In: *Ecology and Evolution of Communities.* (Cody, M. L. & Diamond, J. M. eds). pp. 460-490. Cambridge: Harvard University Press.
CRAMER, N. F. & MAY, R. M. (1971). *J. Theor. Biol.* **34,** 289.
FENCHEL, T. & CHRISTIANSEN, F. B. (1976). *Theories of Biological Communities.* New York: Springer Verlag.
GLASSER, J. W. (1978). *Ecology* **59,** 724.
HANSKI, I. (1981). *Oikos* **37,** 306.
HARPER, J. L. (1977). *Population Biology of Plants.* New York: Academic Press.
HOLT, R. D. (1977). *Theor. Pop. Biol.* **12,** 197.
HOLT, R. D. (1984). *Am. Nat.* **124,** 377.
HUTCHINSON, G. E. (1978). *An Introduction to Population Ecology.* New Haven: Yale University Press.
KAPUR, J. N. (1980). *Math. Biosci.* **51,** 175.
LAWTON, J. H. & HASSELL, M. P. (1981). *Nature* **289,** 793.
LEIGH, E. G. JR. (1981). *J. Theor. Biol.* **90,** 213.
LEVIN, S. (1981). In: *The Mathematical Theory of the Dynamics of Biological Populations* II. (Hiorns, R. W. & Cooke, D. eds). pp. 173-196. London: Academic Press.
LUBCHENCO, J. (1978). *Am. Nat.* **112,** 23.
MAY, R. M. (1975). *Stability and Complexity in Model Ecosystems.* (Second Edition.) Princeton: Princeton University Press.
MAY, R. M. (1977). *Nature* **269,** 103.

NEILL, W. E. (1974). *Am. Nat.* **108,** 399.
NOY-MEIR, I. (1981). *Oecologia* **50,** 277.
PAINE, R. T. (1966). *Am. Nat.* **100,** 65.
POMERANTZ, M. J., THOMAS, W. R. & GILPIN, M. E. (1980). *Oecologia* **47,** 311.
ROBERTS, A. (1974). *Nature* **251,** 607.
SCHAFFER, W. M. (1981). *Ecol. Monogr.* **57,** 383.
SCHOENER, T. W. (1974). *Theor. Pop. Biol.* **6,** 265.
SCHOENER, T. W. (1982). *Am. Sci.* **70,** 586.
SMITH-GILL, S. J. & GILL, D. E. (1978). *Am. Nat.* **117,** 557.
SLOBODKIN, L. B. (1961). *Growth and Regulation of Animal Populations.* New York: Holt, Rinehart & Winston.
STROBECK, C. (1973). *Ecology* **54,** 650.
TILMAN, D. (1982). *Resource Competition and Community Structure.* Princeton: Princeton University Press.
VAN VALEN, L. (1974). *J. theor. Biol.* **44,** 19.
WILBUR, H. M. (1972). *Ecology* **53,** 3.
WILLIAMS, B. G. & BANYIKWA, F. F. (1981). *Am. Nat.* **117,** 774.
YODZIS, P. (1978). *Competition for Space and the Structure of Ecological Communities.* Berlin: Springer Verlag.