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Predation risk, unequal competitors and the ideal free distribution

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ABSTRACT

Ideal free distribution (IFD) theory has frequently been used to investigate habitat selection when fitness payoffs are frequency-dependent. To date, however, researchers have not considered the possibility that individuals may simultaneously differ in their ability to compete for resources and their susceptibility to predation. Such differences might be expected to occur as a consequence of differences in body size, morphology or behaviour. Here, we develop a model to investigate the effects of differences in competitive ability and mortality risk on the equilibrium distribution of competitors across habitats. For simplicity, we consider the case of two competitor types competing for resources in an environment containing two habitats: a productive, but risky habitat and a less productive, safer habitat. In general, the model predicts that when individual mortality risk is independent of the density of competitors within a habitat, competitor types will tend to be assorted by competitive ability, with the competitor type experiencing the higher ratio of mortality risk across the habitats occurring predominantly in the safer, but less productive habitat. In contrast, when individual mortality risk within a habitat is diluted by competitor number, the model predicts that both competitor types will tend to aggregate in the same habitat, with the chosen habitat depending on which competitor type experiences the higher ratio of mortality risk across the habitats. When good competitors experience a higher ratio of mortality risk than poor competitors, both competitor types will tend to aggregate in the risky, but productive habitat. However, when poor competitors experience the higher ratio of mortality risk, both competitor types will tend to aggregate in the safer, but less productive habitat. Because our model can be applied to both intra- and interspecific resource competition, its results suggest a new potential mechanism for the co-existence of competitor types within a habitat.

Keywords: habitat selection, ideal free distribution, predation risk, unequal competitors.

INTRODUCTION

The process of habitat selection often requires individuals to choose among habitats that differ in growth potential and mortality risk due to predation. When the habitat providing the highest rate of energetic gain is also the most dangerous, habitat selection should reflect

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a compromise between the conflicting demands of growth and survival. Indeed, many studies have demonstrated that animals are sensitive to both energetic gains and mortality risk during habitat selection, and are capable of responding to such trade-offs in an adaptive manner (for recent reviews, see Lima and Dill, 1990; Lima, 1998). In some cases, however, the fitness consequences of choosing a particular habitat depend not only on the characteristics of the habitat itself, but also on the number of other individuals present (i.e. fitness consequences are density-dependent).

Ideal free distribution (IFD) theory (Fretwell and Lucas, 1970; Fretwell, 1972) has often been used to study the effects of density-dependent resource competition on habitat selection (see Tregenza, 1995, for a recent review). Assuming that all individuals are of equal competitive ability, that each has perfect or 'ideal' information about the distributions of both competitors and resources, and is 'free' to move to the habitat where its resource payoff will be greatest, the model predicts that, at equilibrium, the distribution of competitors across habitats will 'match' the distribution of resources (i.e. 'input-matching'; Parker, 1974). Implicit in this approach is the assumption that individual resource payoffs decline as the number of competitors in a habitat increases. In some situations, however, individual survival may increase with increasing local population density (Pulliam and Caraco, 1984). Group members may experience reduced risk of mortality as a consequence of shared vigilance (Elgar, 1989), predator confusion (Milinski and Heller, 1978), or simple numerical dilution (Foster and Treherne, 1981; Morgan and Godin, 1985), particularly when predators are limited in their ability to capture more than a single prey item per attack.

Although several researchers have considered the effects of density-dependent growth and mortality on habitat selection within the framework of IFD theory (e.g. McNamara and Houston, 1990; Hugie and Dill, 1994; Moody *et al.*, 1996), none have allowed for the possibility that competitors might differ both in their ability to compete for resources and in their susceptibility to predation. There are many reasons why such differences might exist. For example, body size may influence an individual's ability to detect and acquire resources (Grand, 1997) and its probability of being captured by a predator (Werner and Gilliam, 1984). Similarly, individuals may possess morphological features that enhance competitive ability (Price, 1978) and/or reduce vulnerability to predators (e.g. Abrahams, 1995). Thus, differences in body size and morphology among competitors may affect each individual's best resolution to the conflicting demands of growth and survival and, consequently, the equilibrium distribution of competitors across habitats.

Individual differences in competitive ability have already been incorporated into IFD theory by Sutherland and Parker (1985) and Parker and Sutherland (1986), who assumed that an individual's resource payoff is related to its competitive ability or 'competitive weight' (i.e. the proportion of a resource it obtains when competing with all other members of a group in a single habitat). When the relative competitive weights of individuals are unaffected by local resource or competitor densities, and thus remain the same across habitats, their model predicts that animals should distribute themselves such that the proportion of competitive weights in each habitat 'matches' the proportion of resources available there (i.e. input-matching of competitive weights; see Grand, 1997).

Here, we model the effect of mortality risk on the unequal competitors IFD and ask how differences in both competitive ability and susceptibility to predation might influence an individual's choice of habitat, and hence the equilibrium distribution of competitors across habitats. As with other models of this sort, we assume that competitors have 'ideal'

information about all habitat parameters and are 'free' to move to the habitat where their fitness payoff is greatest. We begin by considering situations where individual mortality risk is unaffected by competitor density, and then consider the effect of dilution of mortality risk on habitat selection. Finally, we compare the predictions of our model to the patterns of habitat selection exhibited by a well-studied assemblage of desert rodents, illustrating how the insights provided by IFD theory may prove useful for understanding patterns of species co-existence and community structure.

THE MODEL

We model the distribution of a large number of competitors of two types: 'poor' competitors (type 1) and 'good' competitors (type 2). The total number of type 1 and 2 competitors is given by N_1 and N_2, respectively. We define K as the competitive ability of good competitors relative to poor competitors (i.e. $K > 1$), and assume that K remains constant across habitats. We consider an environment containing two habitats: a 'good' habitat (A) and a 'poor' habitat (B), with resource availability in each given by R_A and R_B (energy $\cdot$ time^{-1}), respectively. We assume that resources are continually renewing, and therefore non-depleting, and that the rate of energy gain per unit of competitive ability is inversely proportional to the number of competitive units in a habitat ('continuous input' scenario of Tregenza, 1995). For a summary of all constants and variables used in the model, see Table 1.

Table 1. Summary and definitions of all constants and variables used in the model

Symbol	Definition	Units
$i = 1, 2$	competitor type	—
N_i	total number of type i competitors	—
K	competitive ability of type 2 competitors relative to type 1 competitors	—
j = A, B	habitat	—
R_j	prey availability in habitat j	energy $\cdot$ time^{-1}
μ_{ij}	mortality risk for type i competitors in habitat j	probability of death $\cdot$ time^{1}
p_i	proportion of competitor type i in habitat A	—
$1 - p_i$	proportion of competitor type i in habitat B	—
$l(i, j)$	lifespan of competitor type i in habitat j	time
$e(i, j)$	net energy intake of competitor type i in habitat j	energy $\cdot$ time^{-1}
F	proportion of energy available for growth	—
M_i	metabolic requirement of competitor type i	energy $\cdot$ time^{-1}
O	energetic cost per offspring	energy $\cdot$ offspring^{-1}
$w(i, j)$	fitness of competitor type i in habitat j	offspring
$\hat{p}_i$	equilibrium proportion of type i competitors in habitat A	—
c_j	sum of competitive abilities in habitat j	—
n_j	total number of competitors in habitat j	—
$\mu_{ij}(n_j)$	mortality risk for type i competitors in habitat j as a function of the number of competitors there	probability of death $\cdot$ time^{-1}
d	dilution exponent	—

In addition to differing in resource availability, habitats also differ in their associated mortality risk, such that the risk of death due to predation for type *i* competitors in habitat *j* is given by μ_{ij} (probability$\cdot$time^{-1}). We assume that competitor types are encountered at random by the predator, who exhibits no diet selectivity. Predation risk might be expected to differ between habitats as a consequence of differences in structural complexity, light level or the availability of refuge sites. The risk of mortality experienced by the two competitor types might be expected to differ as a consequence of differences in their morphology, body size and predator avoidance behaviour, including flight initiation distance and flight speed (Lima and Dill, 1990). Some competitor types may also be more easily detected by predators than others, particularly when competitive ability is correlated with body size. Initially, we assume μ_{ij} to be independent of the number of competing individuals in a habitat. In keeping with our interest in foraging–predation risk trade-offs, we consider only scenarios where $\mu_{i\mathrm{A}} \geq \mu_{i\mathrm{B}}$ (i.e. the more productive habitat is at least as dangerous as the less productive habitat), for both competitor types.

We seek the equilibrium distribution of competitor types across the habitats, assuming that all individuals seek to maximize their fitness. We describe the distribution of the *i*th competitor type (where $i = 1, 2$) by the proportion of those competitors in habitat A, p_i; their proportion in habitat B is given by $1 - p_i$. To incorporate both energetic gains and mortality risk in a single currency, we calculate fitness in terms of expected lifetime production of offspring. We assume that population size is held constant due to density-dependent factors (i.e. parasitism or disease) and impose no maximum lifespan (as in Hugie and Dill, 1994).

Since we begin by assuming that mortality risk is independent of competitor density, the expected lifespan of competitor type *i* in habitat *j* ($l(i, j)$) is simply:

$$l(i, j) = \frac{1}{\mu_{ij}} \tag{1}$$

The expected net energy intake rate of competitor type *i* in habitat *j* ($e(i, j)$), however, depends on the distribution of both type 1 (p_1) and type 2 (p_2) competitors. As a consequence of differences in competitive ability, energy intake rates differ for good and poor competitors. For good competitors, expected net energy intake rates in habitats A and B are equal to:

$$e(2, \mathrm{A}) = K\left(\frac{R_{\mathrm{A}}}{p_1 N_1 + p_2 N_2 K} \right) F - M_2 \tag{2}$$

and

$$e(2, \mathrm{B}) = K\left(\frac{R_{\mathrm{B}}}{(1 - p_1) N_1 + (1 - p_2) N_2 K} \right) F - M_2 \tag{3}$$

respectively, where F is the proportion of acquired energy that is available for reproduction after accounting for all costs which are proportional to gross energy intake (such as searching and handling), and M_i is the metabolic requirement (energy$\cdot$time^{-1}) of competitor type *i*. The corresponding expected net energy intake rates of poor competitors are equal to:

$$e(1, \mathrm{A}) = \left(\frac{R_\mathrm{A}}{p_1 N_1 + p_2 N_2 K}\right) F - M_1 \tag{4}$$

and

$$e(1, \mathrm{B}) = \left(\frac{R_\mathrm{B}}{(1-p_1) N_1 + (1-p_2) N_2 K}\right) F - M_1 \tag{5}$$

For simplicity, we assume that F is the same for both competitor types, that metabolic requirements are negligible (i.e. $M_i \approx 0$), and that F and M_i are independent of habitat. Thus, fitness of the ith competitor type in the jth habitat ($w(i, j)$) equals:

$$w(i, j) = \frac{l(i, j) e(i, j)}{O} \tag{6}$$

where O is the energy required to produce a single offspring. We assume that all individuals in the population are capable of reproduction and can therefore translate energy directly into offspring.

The distribution of competitor type i will be at equilibrium when its fitness payoffs in the two habitats are equal:

$$w(i, \mathrm{A}) = w(i, \mathrm{B}) \tag{7}$$

Substituting in the appropriate expressions for $l(i, j)$ and $e(i, j)$, and solving equation (7) for the equilibrium distribution of each competitor type as a function of the other, produces two straight lines, each having a negative slope and a positive intercept:

$$\hat{p}_2 = -\left(\frac{N_1}{N_2 K}\right) p_1 + \left(\frac{R_\mathrm{A} \mu_{2\mathrm{B}}}{R_\mathrm{A} \mu_{2\mathrm{B}} + R_\mathrm{B} \mu_{2\mathrm{A}}}\right)\left(\frac{N_1 + N_2 K}{N_2 K}\right) \tag{8}$$

$$\hat{p}_1 = -\left(\frac{N_2 K}{N_1}\right) p_2 + \left(\frac{R_\mathrm{A} \mu_{1\mathrm{B}}}{R_\mathrm{A} \mu_{1\mathrm{B}} + R_\mathrm{B} \mu_{1\mathrm{A}}}\right)\left(\frac{N_1 + N_2 K}{N_1}\right) \tag{9}$$

where $\hat{p}_1$ and $\hat{p}_2$ are the equilibrium proportions of type 1 and type 2 competitors, respectively, in the good habitat (A). Equations (8) and (9) represent the 'best response curves' or 'rational reaction sets' (Vincent and Grantham, 1981) for good and poor competitors, respectively. Points on competitor type i's best response curve denote the distribution of competitor type i for which fitness payoffs are the same in both habitats for any particular distribution of the other competitor type. To compare their slopes and intercepts directly, we plot these two best response curves on a common set of axes (i.e. p_2 versus p_1) by rearranging equation (9) and solving for p_2. Thus, the best response curve for type 1 competitors becomes:

$$p_2 = -\left(\frac{N_1}{N_2 K}\right) \hat{p}_1 + \left(\frac{R_\mathrm{A} \mu_{1\mathrm{B}}}{R_\mathrm{A} \mu_{1\mathrm{B}} + R_\mathrm{B} \mu_{1\mathrm{A}}}\right)\left(\frac{N_1 + N_2 K}{N_2 K}\right) \tag{10}$$

Note that the best response curves for type 1 and 2 competitors have the same, negative slope and differ only with respect to intercept. As a consequence, these response curves will never intersect and the usual method of solving for the simultaneous equilibrium of the two competitor types (or more generally, two alternative strategies) cannot be used (see Hugie

and Grand, 1998). Instead, we use the graphical methods of Rosenzweig and MacArthur (1963) to determine what the combined equilibrium distribution of type 1 and 2 competitors will look like under a variety of conditions. We confirm these equilibria and their stability by computer simulation, using the evolutionary difference equations described in the Appendix.

The best response curves of the two competitor types will overlap completely when their intercepts are identical:

$$\left(\frac{R_A\mu_{2B}}{R_A\mu_{2B}+R_B\mu_{2A}}\right)\left(\frac{N_1+N_2K}{N_2K}\right)=\left(\frac{R_A\mu_{1B}}{R_A\mu_{1B}+R_B\mu_{1A}}\right)\left(\frac{N_1+N_2K}{N_2K}\right) \tag{11}$$

or, more simply, when the ratio of mortality risk across the habitats (hereafter referred to as the 'risk ratio') is the same for both competitor types:

$$\frac{\mu_{1A}}{\mu_{1B}}=\frac{\mu_{2A}}{\mu_{2B}} \tag{12}$$

In this case, the simultaneous equilibrium of type 1 and 2 competitors can occur anywhere along the shared best response curve, its exact location depending only upon the initial distribution of competitor types, $(p_1, p_2)_{t=0}$ (Fig. 1). When $(p_1, p_2)_{t=0}$ lies below the shared best response curve, both competitor types experience higher fitness payoffs in habitat A. As a consequence, both will increase their proportion in A until payoffs in the two habitats are equal. Similarly, when $(p_1, p_2)_{t=0}$ lies above the response curve, both competitor types experience higher fitness payoffs in habitat B and will decrease their proportion in A until fitness payoffs in the two habitats are equal. All points along the shared best response curve represent neutrally stable distributions of competitor types 1 and 2 (see the Appendix). For all such $(\hat{p}_1, \hat{p}_2)$:

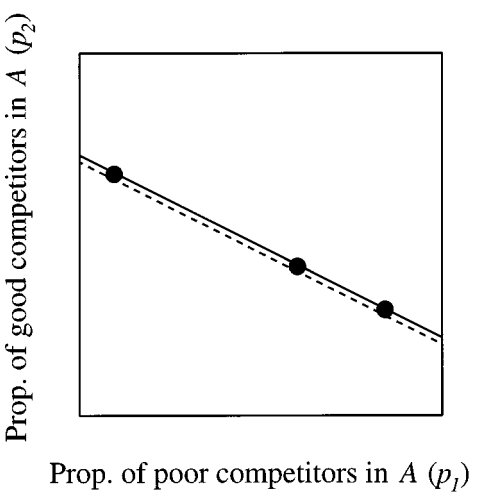

Fig. 1. Best response curves for type 1 (- - -) and type 2 (——) competitors when both experience the same ratio of mortality risk across the two habitats. The combined equilibrium (●) can occur anywhere along the shared response curve, depending on the initial distribution of competitor types.

$$\frac{c_A}{c_B} = \frac{\hat{p}_1 N_1 + \hat{p}_2 N_2 K}{(1-\hat{p}_1)N_1 + (1-\hat{p}_2)N_2 K} = \left(\frac{R_A}{R_B}\right)\left(\frac{\mu_{1B}\mu_{2B}}{\mu_{1A}\mu_{2A}}\right)\left(\frac{\mu_{2A}-\mu_{1A}}{\mu_{2B}-\mu_{1B}}\right) \quad (13)$$

where c_A and c_B are the sums of competitive weights in habitats A and B, respectively. Hence, when competitor types experience the same ratio of mortality risk across the habitats (i.e. when expression (12) is true), regardless of the absolute mortality risk in each, the ratio of the sum of competitive weights across the two habitats will be proportional to (1) the ratio of resource availabilities, (2) the inverse of each competitor type's risk ratio, and (3) the ratio of the within-habitat differences in mortality risk between competitor types. All equilibria that satisfy equation (13) are characterized by under-matching of competitive weights (i.e. there are fewer competitive weights in the good habitat than predicted by the distribution of resources alone), given that both competitor types experience a higher risk of mortality in habitat A than in habitat B. Note that when habitats have the same mortality risk (i.e. $\mu_{1A} = \mu_{1B}$ and $\mu_{2A} = \mu_{2B}$), the distribution of competitive weights matches the distribution of resources, as originally predicted by Parker and Sutherland (1986).

When competitor types experience different ratios of mortality risk across the habitats (i.e. when expression (12) is false), their best response curves no longer share a common intercept. The best response curve of the competitor type with the higher risk ratio is lower in elevation, corresponding to a decrease in the proportion of that competitor type in habitat A for any given distribution of the other competitor type. Intuitively, this makes sense, since the competitor type whose risk of mortality is most greatly reduced by using the poor habitat should be more likely to be found there.

The location of the combined equilibrium ($\hat{p}_1$, $\hat{p}_2$) now depends primarily on which competitor type experiences the higher ratio of mortality risk across the habitats. When poor (type 1) competitors have a higher risk ratio than good (type 2) competitors, their response curve is lower in elevation than that of good competitors. The combined equilibrium usually occurs at the intersection of the type 2 competitors' best response curve and the y-axis, regardless of the initial distribution of competitor types (Fig. 2a). However, depending on the steepness and elevation of this curve (see below), its intersection with the y-axis may occur at $p_2 > 1$, in which case the equilibrium occurs at the intersection of the type 1 competitors' best response curve and the line $p_2 = 1$ (Fig. 2b). In both cases, the combined equilibrium is characterized by at least one competitor type occurring exclusively in a single habitat. Either good competitors occur exclusively in habitat A, accompanied by only a small proportion of poor competitors (Fig. 2b), or poor competitors occur exclusively in habitat B, accompanied by only a small proportion of good competitors (Fig. 2a). Note that at this equilibrium, only the competitor type that occurs in both habitats experiences the same fitness payoff in each habitat (i.e. only for this competitor type will equation (7) be satisfied).

When good competitors have a higher ratio of mortality risk across the habitats than poor competitors, the best response curve of type 2 competitors is lower in elevation than that of type 1 competitors, and the combined equilibrium usually occurs where the type 2 competitors' response curve intersects the line $p_1 = 1$, regardless of the initial distribution of competitor types (Fig. 2d). However, depending on the steepness and elevation of the response curve (see below), this intersection may occur below the x-axis (i.e. at $p_2 < 0$), in which case the equilibrium occurs at the intersection of the type 1 competitors' best response curve and the x-axis (Fig. 2e). Again, the combined equilibrium is characterized by

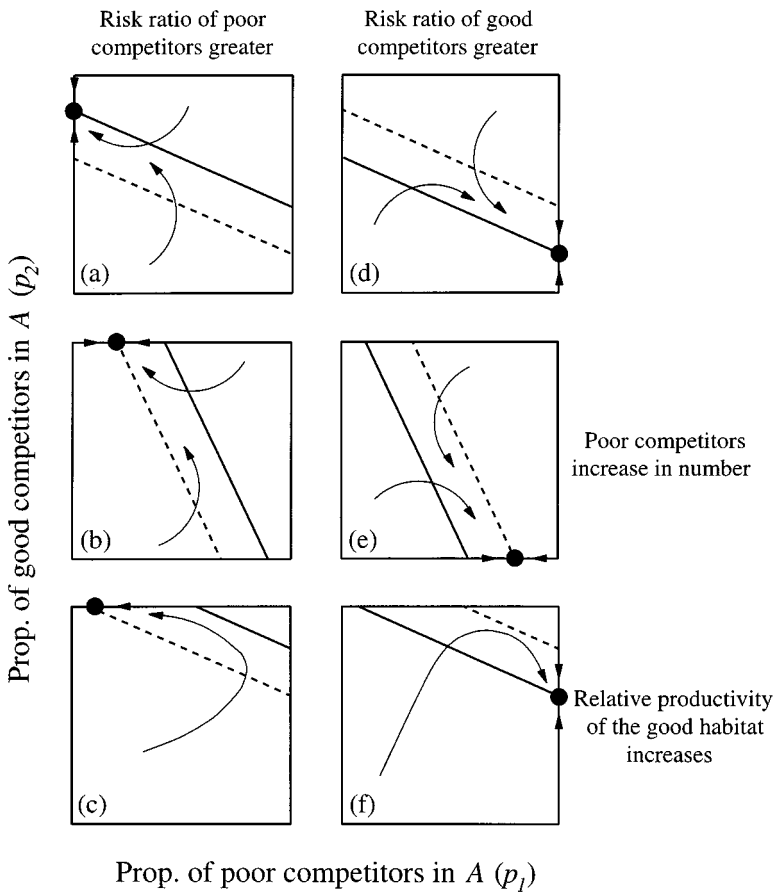

Fig. 2. The effects of changing relative competitor density ($N_1 : N_2$) and relative habitat productivity ($R_A : R_B$) on the best response curves of type 1 (- - -) and type 2 (——) competitors, when (a, b, c) poor competitors experience a higher ratio of mortality risk across the habitats, or (d, e, f) good competitors experience a higher ratio of mortality risk across the habitats. The locations of the combined equilibrium and sample trajectories of the change in the proportion of each competitor type in habitat A for all $(p_1, p_2) \neq (\hat{p}_1, \hat{p}_2)$ are indicated by ● and →, respectively. In all cases, $\mu_{1A} = \mu_{2A} = 0.5$, $K = 2$ and $N_2 = 1000$. The remaining parameter values are: (a) $R_A = 1.2$, $R_B = 0.8$, $\mu_{2B} = 0.5$, $\mu_{1B} = 0.3$, $N_1 = 1000$; (b) $R_A = 1.2$, $R_B = 0.8$, $\mu_{2B} = 0.5$, $\mu_{1B} = 0.3$, $N_1 = 3000$; (c) $R_A = 1.6$, $R_B = 0.4$, $\mu_{2B} = 0.5$, $\mu_{1B} = 0.3$, $N_1 = 1000$; (d) $R_A = 1.2$, $R_B = 0.8$, $\mu_{2B} = 0.3$, $\mu_{1B} = 0.5$, $N_1 = 1000$; (e) $R_A = 1.2$, $R_B = 0.8$, $\mu_{2B} = 0.3$, $\mu_{1B} = 0.5$, $N_1 = 3000$; (f) $R_A = 1.6$, $R_B = 0.4$, $\mu_{2B} = 0.3$, $\mu_{1B} = 0.5$, $N_1 = 1000$.

at least one competitor type occurring exclusively in a single habitat. Either poor competitors occur exclusively in habitat A, accompanied by only a small proportion of good competitors (Fig. 2d), or good competitors occur exclusively in habitat B, accompanied by only a small proportion of poor competitors (Fig. 2e).

Thus, when competitor types experience different ratios of mortality risk across the habitats, equilibria tend to be characterized by segregation of competitor types (i.e. animals tend to be assorted by competitive ability). The competitor type with the higher risk ratio tends to avoid the risky habitat, regardless of which competitor type is at absolutely greater

risk there. Again, distributions of competitive weights are always under-matched relative to the distribution of resources, assuming that both competitor types experience a higher risk of mortality in habitat A than in habitat B.

The slopes and elevations of the two competitor types' best response curves, and therefore the location of the combined equilibrium, are influenced by the values of N_1, N_2 and K, and R_A, R_B and μ_{ij}, respectively. As the abilities of the competitor types become more similar (i.e. $K \to 1$), or the number of poor competitors increases relative to the number of good competitors, the slopes of both response curves increase (Fig. 2a,b and Fig. 2d,e) and become bounded by the line $p_2 = 1$. This bounding also occurs as the productivity of the good patch increases relative to that of the poor patch and the response curves increase in elevation (Fig. 2a,c and Fig. 2d,f). As a consequence of increases in the slope of the best response curves, both competitor types increase their proportion in habitat A, as long as poor competitors experience a higher ratio of mortality risk across the habitats than do good competitors (e.g. Fig. 2a,b). This occurs because the 'resource space' required by good competitors decreases with their competitive advantage, leaving vacancies to be filled in habitat A (in the case of decreasing K) and because increasing numbers of poor competitors in both habitats reduce the benefits associated with the safer habitat, particularly for good competitors (in the case of increasing $N_1 : N_2$). In contrast, when good competitors have a higher risk ratio than poor competitors, increases in best response curve slope result in both competitor types decreasing their proportion in habitat A (e.g. Fig. 2d,e). This is because the energetic benefits received by good competitors no longer outweigh the mortality cost associated with the riskier habitat.

An increase in the elevation of best response curves results in both competitor types increasing their proportion in habitat A, regardless of which competitor type experiences the higher ratio of mortality risk across the habitats (compare Fig. 2a and 2e or 2d and 2f), solely as a consequence of increased resource availability. Finally, the magnitude of the difference in elevation between the best response curves depends on the difference in the risk ratios of the two competitor types: as the difference between risk ratios increases, the difference in elevation between the response curves also increases.

Regardless of the parameter values chosen, when competitor types experience different ratios of mortality risk across the habitats and risk is undiluted by competitor number, individuals will tend to be assorted by competitive ability, with the competitor type experiencing the higher risk ratio occurring predominantly in the less productive (but safer) habitat.

Incorporating dilution of mortality risk

Thus far, we have assumed that the mortality risk experienced by each individual is independent of the number of individuals in the habitat. However, as with foraging payoffs, mortality risk may also be density-dependent if, for example, predators are constrained in their ability to pursue, capture and handle more than one prey item at a time. We now consider the effect of dilution of mortality risk on the equilibrium distribution of competitor types. Per-capita mortality risk experienced by the ith competitor type in the jth habitat, μ_{ij} (where μ_{ij} is defined as the risk experienced by a single competitor of the ith type in the jth habitat), is now a function of the total number of competitors in that habitat, $\mu_{ij}(n_j)$, independent of their respective competitive abilities. For example, in habitat A, the mortality risk experienced by type 2 competitors is equal to:

$$\mu_{2A}(n_A) = \frac{\mu_{2A}}{(p_1 N_1 + p_2 N_2)^d} \tag{14}$$

where d scales the relationship between competitor number and risk of mortality ($0 \le d \le 1$). When $d = 0$, there is no dilution of mortality risk and the risk experienced by each individual in the habitat is as described earlier. When $d = 1$, mortality risk is fully diluted, and all individuals in the habitat experience a reduction in risk that is directly proportional to the number of individuals there. Again, we assume that competitor types are encountered at random and that there is no diet selectivity on the part of the predator.

With the addition of the dilution exponent, the equilibrium distribution of competitor type i can no longer be expressed as a simple function of the distribution of the other competitor type (i.e. in the terms of equations (8) and (9)). We can, however, approximate the best response curves of the two competitor types by computer simulation. In doing so, we ask what distribution of type i competitors is required to satisfy expression (7), given a variety of distributions of the other competitor type. As before, we use these best response curves to determine what the combined equilibrium distribution of type 1 and 2 competitors will look like under a variety of conditions and confirm these equilibria and their stability via computer simulation (see the Appendix).

As shown previously, when $d = 0$, the best response curve of each competitor type is a straight line with negative slope and positive intercept. As d increases, both response curves rotate counter-clockwise, their slopes first decreasing to 0 then increasing positively, in some cases, decelerating or accelerating as $d \to 1$ (Fig. 3a,b, respectively).

Once again, when the mortality risk ratios of the two competitor types are identical, their best response curves overlap completely. The combined equilibrium can occur

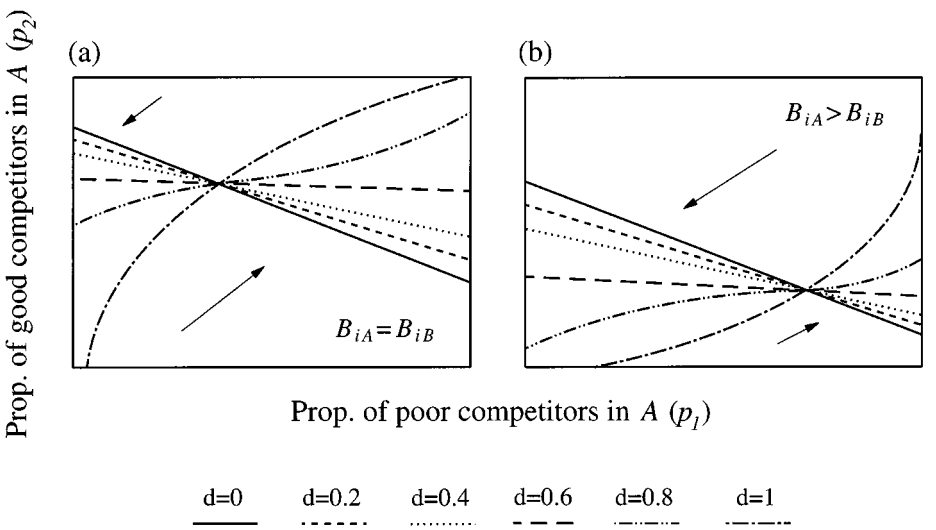

Fig. 3. The effect of increasing the strength of dilution on the shared best response curve of type 1 and 2 competitors when (a) inherent mortality risk in the two habitats is equal and (b) habitat A is inherently riskier than habitat B. Arrows indicate sample trajectories of the change in the proportion of each competitor type in habitat A for all $(p_1, p_2) \neq (\hat{p}_1, \hat{p}_2)$. In both (a) and (b), $R_A = 1.2$, $R_B = 0.8$, $N_1 = N_2 = 1000$, $K = 2$ and $\mu_{1A} = \mu_{2A} = 0.5$. In (a), $\mu_{1B} = \mu_{2B} = 0.5$; in (b), $\mu_{1B} = \mu_{2B} = 0.3$.

anywhere along the shared response curve, its exact location depending on both the initial distribution of competitor types, $(p_1, p_2)_{t=0}$, and the degree of dilution. When $(p_1, p_2)_{t=0}$ lies below the shared best response curve, both competitor types experience higher fitness payoffs in habitat A than in habitat B. As a consequence, both competitor types will alter their proportion in A until payoffs in the two habitats are equal (see sample trajectories in Fig. 3). Similarly, when $(p_1, p_2)_{t=0}$ lies above the best response curve, both competitor types experience higher fitness payoffs in B than in A, and will alter their distribution until fitness payoffs in the two habitats are equal. All points along the shared best response curve represent neutrally stable distributions of competitor types 1 and 2. Regardless of the initial distribution of good and poor competitors, for all such $(\hat{p}_1, \hat{p}_2)$ it can be shown that:

$$\frac{c_A}{c_B} = \left(\frac{R_A}{R_B} \right) \left(\frac{\mu_{1B}\mu_{2B}}{\mu_{1A}\mu_{2A}} \right) \left(\frac{\mu_{2A} - \mu_{1A}}{\mu_{2B} - \mu_{1B}} \right) \left(\frac{\hat{p}_1 N_1 + \hat{p}_2 N_2}{(1 - \hat{p}_1)N_1 + (1 - \hat{p}_2)N_2} \right)^d \tag{15}$$

Hence, when competitor types experience the same ratio of mortality risk across the habitats, the ratio of the sum of competitive weights across the habitats will be proportional to: (1) the ratio of resource availabilities; (2) the inverse of each competitor type's risk ratio; (3) the ratio of the within-habitat differences in mortality risk between competitor types; (4) the ratio of competitor numbers across the habitats; and (5) the strength of dilution. Equilibria that satisfy equation (15) may be characterized by input-, under- or over-matching of competitive weights, depending on the relative risk of mortality in the two habitats and the degree of dilution. In general, when habitats differ greatly in mortality risk and the strength of dilution is weak, under-matching of competitive weights is usually observed.

When the risk ratios of competitor types differ, their best response curves are no longer identical. As was the case without risk dilution, the best response curve of the competitor type with the higher risk ratio is lower in elevation, corresponding to a decrease in the proportion of that competitor type in habitat A for any given distribution of the other competitor type. Again, this makes intuitive sense, since the competitor type whose probability of survival is most greatly increased by using the poor habitat should be more likely to be found there.

As before, the location of the combined equilibrium $(\hat{p}_1, \hat{p}_2)$ depends primarily on which competitor type experiences the higher ratio of mortality risk across the habitats. When poor competitors have a higher risk ratio than good competitors, their best response curve is lower in elevation than that of good competitors, and the combined equilibrium usually occurs at the intersection of the type 2 competitors' best response curve and the y-axis, regardless of the initial distribution of competitor types (Fig. 4a). Depending on the steepness and elevation of this curve, particularly when dilution is weak, the intersection may occur beyond (0, 1), in which case the equilibrium occurs where the type 1 competitors' best response curve crosses the line $p_2 = 1$ (see Fig. 2b). In either case, poor competitors tend to occur almost exclusively in habitat B, with the proportion of good competitors occurring there increasing as the dilution exponent increases. Competitive weights are always under-matched relative to the distribution of resources, given that habitat A is riskier than habitat B for both competitor types.

Conversely, when good competitors have a higher risk ratio than poor competitors, their best response curve is lower in elevation than that of poor competitors, and the combined equilibrium occurs at the intersection of the type 2 competitors' response curve and the line

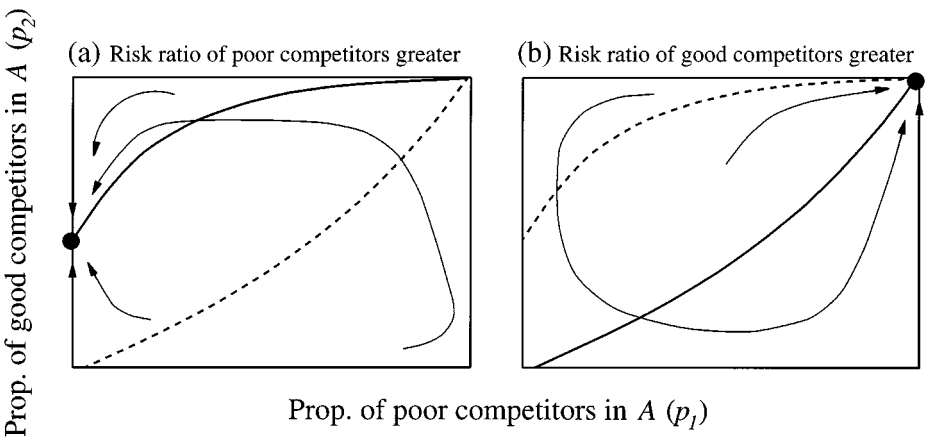

Fig. 4. Best response curves for type 1 (- - -) and type 2 (——) competitors under full dilution of mortality risk ($d = 1$), when (a) poor competitors experience a higher ratio of mortality risk across the habitats and (b) good competitors experience a higher ratio of mortality risk across the habitats. The locations of the combined equilibrium and sample trajectories of the change in the proportion of each competitor type in habitat A for all $(p_1, p_2) \neq (\hat{p}_1, \hat{p}_2)$ are indicated by ● and →, respectively. In both (a) and (b), $R_A = 1.2$, $R_B = 0.8$, $\mu_{1A} = \mu_{2A} = 0.5$, $K = 2$ and $N_1 = N_2 = 1000$. In (a), $\mu_{2B} = 0.5$ and $\mu_{1B} = 0.3$; in (b), $\mu_{2B} = 0.3$ and $\mu_{1B} = 0.5$.

$p_1 = 1$ (Fig. 4b). Again, depending on the steepness and elevation of this curve, and the magnitude of d, the intersection may occur at $p_2 < 0$, in which case the equilibrium occurs at the intersection of the type 1 competitors' best response curve and the x-axis (e.g. Fig. 2e). Poor competitors tend to occur almost exclusively in habitat A, with the proportion of good competitors occurring there increasing as the dilution exponent increases. Depending on the difference in competitor type risk ratios and the degree of dilution, $(\hat{p}_1, \hat{p}_2)$ may be characterized by input-, under- or over-matching of competitive weights. In general, the greater the difference in the risk ratios of competitor types and the weaker the effect of dilution, the more frequently under-matching is expected to occur.

The slopes and elevations of the two best response curves and, consequently, the location of the combined equilibrium, are influenced by the values of N_1, N_2 and K, and R_A, R_B and μ_{ij}, respectively, in the same manner as previously described. Regardless of the parameter values chosen, however, when competitor types experience different ratios of mortality risk across the habitats and mortality risk is diluted by competitor number, competitors tend to aggregate in a single habitat. Furthermore, as the strength of dilution increases, the tendency to aggregate also increases. The habitat chosen depends on which competitor type experiences the higher ratio of mortality risk across the habitats. When good competitors experience the higher risk ratio, both competitor types tend to aggregate in the good habitat (i.e. $(\hat{p}_1, \hat{p}_2) \rightarrow (1, 1)$; compare Fig. 2d to 4b). When the risk ratio of poor competitors is higher than that of good competitors, both competitor types tend to aggregate in the poor habitat (i.e. $(\hat{p}_1, \hat{p}_2) \rightarrow (0, 0)$; compare Fig. 2a to 4a). According to our simulations (see Appendix), there is only one equilibrium, regardless of starting conditions.

Equal competitors – unequal risk: A comparison with Moody *et al.* (1996)

Recently, Moody *et al.* (1996) investigated the effects of mortality risk and risk dilution on Fretwell and Lucas' (1970) original equal competitors IFD model. Assuming that individuals are equally susceptible to predation and that current conditions do not alter future fitness expectations, their model predicts that individuals will tend to aggregate in the more productive of two habitats when risk is fully diluted by competitor number and the fitness value of food is relatively high. In contrast, our model predicts that competitor types will sometimes aggregate in the less productive of those habitats under full dilution of mortality risk. In an attempt to understand why such similar models make different predictions, we evaluate our model under the conditions assumed by Moody *et al.* (1996). Again, we generate best response curves by computer simulation and use them to determine what the equilibrium distribution will look like under a variety of conditions.

Although we generally expect animals to differ in their ability to compete for resources, in some cases individuals differing in phenotype may be more or less equal in competitive ability (i.e. $K \approx 1$). As was the case for $K > 1$, when $d = 0$, the best response curve of each *equal* competitor type is a straight line with negative slope and positive intercept. Now, however, as d increases, best response curves no longer change in slope. Instead, response curves increase in elevation, corresponding to an increase in the proportion of both competitor types in habitat A with an increase in the strength of dilution (see Fig. 5).

When competitor types experience the same risk of mortality within a habitat (i.e. $\mu_{1A} = \mu_{2A}$, $\mu_{1B} = \mu_{2B}$), as assumed by Moody *et al.* (1996), their best response curves overlap completely. The combined equilibrium can occur anywhere along the shared best response curve, its exact location depending on the initial distribution of competitor types, $(p_1, p_2)_{t=0}$, and the magnitude of the dilution exponent (Fig. 5). When $(p_1, p_2)_{t=0}$ lies below the shared response curve, both competitor types experience higher fitness payoffs in habitat A and,

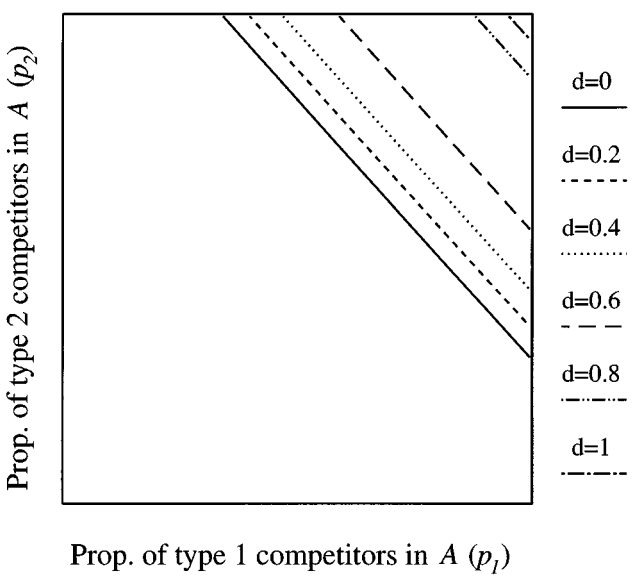

Fig. 5. The effect of increasing the strength of dilution on the shared best response curve of *equal* type 1 and 2 competitors. $R_A = 1.2$, $R_B = 0.8$, $N_1 = N_2 = 1000$, $K = 1$, $\mu_{1A} = \mu_{2A} = \mu_{1B} = \mu_{2B} = 0.5$.

consequently, increase their proportion in A until payoffs in the two habitats are equal. Similarly, when $(p_1, p_2)_{t=0}$ lies above the shared best response curve, both competitor types experience higher fitness payoffs in habitat B and decrease their proportion in A until fitness payoffs in the two habitats are equal. The stronger the effect of risk dilution, the greater the equilibrium proportion of both competitor types in the riskier, more productive habitat.

In many cases, however, 'equal' competitors may experience different mortality risk in the same habitat, perhaps as a consequence of differences in morphology or anti-predator behaviour. When such differences in habitat-specific mortality risk lead to competitor types having identical ratios of mortality risk across the habitats (i.e. when expression (12) is true), the above conclusions are unchanged. However, if competitor types experience different risk ratios, the best response curve of the competitor type with the higher risk ratio is lower in elevation (e.g. Fig. 6), and the location of the combined equilibrium will depend on the relative risk ratios of the two competitor types.

When the risk ratio of type 1 competitors is higher than that of type 2 competitors, $(\hat{p}_1, \hat{p}_2)$ tends to occur at the intersection of the type 1 competitors' best response curve and the line $p_2 = 1$ (Fig. 6a). Similarly, when type 2 competitors experience the higher risk ratio, $(\hat{p}_1, \hat{p}_2)$ tends to occur at the intersection of the type 2 competitors' best response curve and the line $p_1 = 1$ (Fig. 6b). In both cases, the combined equilibrium is characterized by relatively large proportions of both competitor types in habitat A, proportions that increase as the strength of dilution increases. Thus, when competitors are equal in their ability to compete for resources, both competitor types tend to aggregate in the riskier, but more productive habitat, regardless of which competitor type experiences the higher risk ratio.

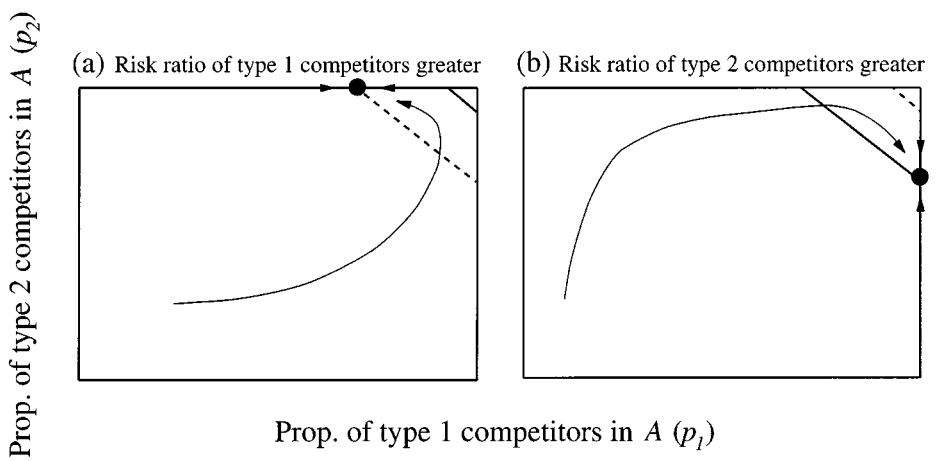

Fig. 6. Best response curves for *equal* type 1 (- - -) and type 2 (——) competitors under strong dilution of mortality risk ($d = 0.9$), when (a) type 1 competitors experience a higher ratio of mortality risk across the habitats and (b) type 2 competitors experience a higher ratio of mortality risk across the habitats. The locations of the combined equilibrium and sample trajectories of the change in the proportion of each competitor type in habitat A for all $(p_1, p_2) \neq (\hat{p}_1, \hat{p}_2)$ are indicated by ● and →, respectively. In both (a) and (b), $R_A = 1.2$, $R_B = 0.8$, $\mu_{1A} = \mu_{2A} = 0.5$, $K = 1$ and $N_1 = N_2 = 1000$. In (a), $\mu_{2B} = 0.5$ and $\mu_{1B} = 0.4$; in (b), $\mu_{2B} = 0.4$ and $\mu_{1B} = 0.5$.

Although our analysis of the equal competitors case confirms the results obtained by Moody *et al.* (1996), our earlier consideration of competitive inequalities demonstrates the lack of generality of this conclusion. Our model predicts that both unequal competitor types tend to reside in the same habitat when the effects of dilution are strong. However, the chosen habitat need not always be the one with the higher input rate, as predicted by Moody *et al.* (1996). When poor competitors experience a higher ratio of mortality risk across the habitats than do good competitors, the combined equilibrium is characterized by both competitor types occurring almost exclusively in the poor habitat (see Fig. 4a). Hence, aggregation in either the good or poor habitat can occur, depending on the relative risk ratios experienced by the competitor types, the strength of risk dilution and the relative abilities of competitor types to compete for resources.

DISCUSSION

We have considered the effect of differences in habitat-specific mortality risk on the equilibrium distribution of unequal competitors. We have shown that such distributions are characterized by either segregation of competitor types across habitats or aggregation of both competitor types within a single habitat, depending on the strength of risk dilution and the ratio of each competitor type's mortality risk across the habitats. Distributions of competitive weights no longer match the distribution of resources, as predicted by the original unequal competitors IFD model (Sutherland and Parker, 1985; Parker and Sutherland, 1986), but are usually under-matched (i.e. there will be too few competitive weights in the good habitat), as expected if individuals are willing to accept a reduction in foraging gains to decrease their risk of predation (Grand and Dill, 1997).

In the absence of risk dilution, our model predicts that competitor types tend to be assorted by competitive ability. The competitor type which experiences the higher ratio of mortality risk across the habitats occurs predominantly in the safer, less productive habitat, regardless of the absolute risk of mortality experienced by either competitor type. As the strength of dilution increases, the reduction in foraging gains associated with choosing a habitat where competitor density is high is increasingly compensated by a reduction in mortality risk, resulting in both competitor types aggregating in the same habitat. Which habitat is preferred depends primarily on which competitor type experiences the higher ratio of mortality risk across the habitats. When the risk ratio of good competitors is greater than that of poor competitors, both competitor types tend to aggregate in the risky, more productive habitat. The safer, less productive habitat is preferred, however, when poor competitors experience the higher risk ratio. In both cases, good competitors are 'drawn' towards the habitat chosen by poor competitors. This is because good competitors, by virtue of their great competitive ability, experience a smaller absolute reduction in foraging payoffs as competitor density increases than do poor competitors; a reduction that is balanced by a decrease in mortality risk for them, but not for poor competitors.

When competitor types experience the same ratio of mortality risk across the habitats, regardless of the strength of dilution or the absolute risk of mortality experienced by either competitor type, a number of neutrally stable equilibrium distributions are possible. Almost all such equilibria are characterized by both competitor types occurring in both habitats, and thus receiving equal fitness payoffs in each. However, as with Parker and Sutherland's (1986) original IFD for unequal competitors, which of these equilibria is actually observed depends on the initial distribution of competitor types.

In nature, individuals frequently exhibit differences in morphology, body size and behaviour that may influence their susceptibility to predation (see Lima and Dill, 1990). Furthermore, morphological and behavioural differences may interact with the physical features of the habitat to modify an individual's risk of predation, such that the relative risks of mortality experienced by competitor types differ across habitats. For example, the relative vulnerability of competitor types may depend on the degree of structural complexity within a habitat (Savino and Stein, 1982, 1989; Schramm and Zale, 1985; Christensen and Persson, 1993), such that one competitor type gains a greater reduction in mortality risk by choosing a particular habitat than do other competitor types, perhaps as a consequence of small body size (e.g. Werner and Gilliam, 1984; Power, 1987) or the absence of protective armour (e.g. McLean and Godin, 1989; Abrahams, 1995). In general, we expect that competitor types will experience different ratios of mortality risk across habitats, and thus that a single, stable distribution of competitor types will usually exist. This equilibrium will tend to be characterized by either segregation of individuals by competitive ability (in the absence of risk dilution) or aggregation of competitors in a single habitat (when risk is fully diluted by competitor number).

There is much evidence to suggest that, given a choice, individuals prefer to forage with competitors of similar body size (Theodorakis, 1989; Krause, 1994; Peuhkuri *et al.*, 1997) and phenotype (Wolf, 1985; Allan and Pitcher, 1986). Often, researchers attribute such assortment to the 'oddity effect' (Landeau and Terborgh, 1986), assuming that individuals who least resemble the group are more conspicuous to predators, and thus more likely to be targeted during a predatory attack. However, if differences in phenotype or body size are correlated with differences in competitive ability (e.g. Godin and Keenleyside, 1984; Grand and Grant, 1994; Grand, 1997), it is not necessary to invoke frequency-dependent predation risk to explain assortment by competitor phenotype. Segregation of competitor types is also frequently predicted to occur as a consequence of differences between competitor types in their habitat-specific resource utilization efficiency. Many habitat selection models, particularly those developed for multi-species systems, assume that each competitor type is most efficient at exploiting resources in a different habitat (e.g. MacArthur and Levins, 1967; Lawlor and Maynard Smith, 1976; Vincent *et al.*, 1996). In our model, good competitors are better at competing for resources than poor competitors in both habitats, and relative resource utilization efficiencies are assumed to remain constant across habitats. Thus, segregation of competitor types can occur in the absence of such 'distinct preferences' (Rosenzweig, 1991) and 'oddity effects', as long as competitor types experience different ratios of mortality risk across the habitats and risk dilution is weak.

Traditionally, IFD theory has been used to investigate the effects of intraspecific competition on habitat selection (see Tregenza, 1995). However, the theory (and modifications of it) may also enhance our understanding of interspecific patterns of habitat use, particularly in communities where multiple species compete for access to a common resource pool. For example, segregation by habitat has frequently been observed within North American assemblages of granivorous desert rodents. In general, large, bipedal species (e.g. kangaroo rats, *Dipodomys*) tend to forage in open areas, where the risk of encountering predators is high (Kotler *et al.*, 1988, 1991), whereas small, quadrupedal species (e.g. deer mice, *Peromyscus*) restrict their foraging to bushes and other relatively safe habitats (Kotler, 1984, 1985). Two classes of mechanisms have been proposed to explain this pattern: (1) species differ in the habitat in which they are competitively superior, and (2) species differ in the habitat in which they are most vulnerable to predation. According to the predictions

of our model, this pattern of habitat selection could also result if: (1) both species are at greater risk in the open habitat, but quadrupedal species experience a higher ratio of mortality risk across the habitats than bipedal species; (2) the relative competitive abilities of bipedal and quadrupedal species are similar across habitats; (3) open habitats are at least as productive as bush habitats (i.e. $R_A \geq R_B$); and (4) dilution of mortality risk is weak.

Both bipedal and quadrupedal species are more likely to be captured by predators in open habitats than in bush habitats (Kotler, 1984, 1985; Kotler *et al.*, 1988, 1991). However, bipedal species are less likely to be captured than quadrupedal ones in open habitats (Kotler *et al.*, 1988), presumably as a consequence of the former's enlarged auditory bullae and bipedal locomotory habits, which enhance predator detection and avoidance abilities, respectively (Rosenzweig, 1973). Assuming that bipedal species are at least as vulnerable to predators in bush habitats as are quadrupedal species, quadrupeds will experience a higher ratio of mortality risk across the habitats than bipeds (see table 1 of Kotler *et al.*, 1988), as required by our model.

Although differences in morphology, body size and locomotory ability may influence the relative abilities of species to harvest resources in open and bush habitats (see Kotler, 1984), it is unclear how different the competitive abilities of bipedal and quadrupedal species actually are and whether they remain constant across habitats. However, large (bipedal) species are generally able to harvest (Price and Heinz, 1984) and husk (Rosenzweig and Sterner, 1970) seeds more rapidly than small (quadrupedal) species. Such skills are likely to reflect competitive ability and are unlikely to vary greatly with habitat type, although large species may have more difficulty searching for food in the bush habitats than smaller species (Brown *et al.*, 1988). Although relative habitat productivities have not been rigorously quantified, open areas are perceived to contain richer seed resources than bush habitats (Kotler, 1984), as required by our model.

To date, the effect of competitor density on per-capita predation rates has not been studied in this system, although Rosenzweig *et al.* (1997) have found evidence for a dilution effect in small populations of old-world desert gerbils. If, however, North American desert rodents do not gain a significant reduction in mortality risk by associating with conspecific or heterospecific competitors, our model predicts that quadrupedal and bipedal species should occur in different habitats, given the relationships between body form, competitive ability and habitat-specific mortality risk discussed above.

Unlike the two aforementioned explanations for habitat segregation in desert rodent communities, our explanation does not require competitor species to rank habitat profitabilities differently (e.g. Rosenzweig, 1973; Brown *et al.*, 1988) or to differ in the habitat in which they experience the highest mortality risk (e.g. Longland and Price, 1991). Furthermore, species that occur predominantly in open habitats need not experience an absolutely greater risk of mortality there than species which occur predominantly in bush habitats. Thus, in comparing the assumptions and predictions of our model to the patterns of habitat use exhibited by desert rodents, we have provided another potential explanation for the co-existence of species who exploit the same resources (for other explanations, see Kotler and Brown 1988; Brown 1989). Clearly, however, the ability of such a behavioural mechanism to promote and maintain species co-existence cannot be evaluated without some consideration of population dynamics. An investigation into the range of parameter values capable of producing stable co-existence of competitor types over ecological time is currently underway and will be reported elsewhere.

As is true of all models, ours makes a number of assumptions which may have influenced the predictions generated. For simplicity, we have assumed that relative competitive abilities remain constant across habitats, such that both competitor types rank habitat profitabilities identically. However, if competitor types disagree on which habitat is the most profitable, or relative competitive abilities change across habitats, segregation of competitor types is likely to be absolute, even in the absence of mortality risk (e.g. Lawlor and Maynard Smith, 1976; Parker and Sutherland, 1986). We have also assumed that the fitness value of food remains constant over time and is the same for all type *i* competitors. However, as demonstrated by Moody *et al.* (1996) and McNamara and Houston (1990), relaxation of these assumptions can lead to competitor distributions which reflect neither the distribution of resources nor the spatial distribution of mortality risk. We have assumed that mortality risk is spatially fixed, such that predators are unable to alter their distribution in response to the distribution of their prey. However, if predators are also free to move to the habitat where their fitness gains are highest, competitors may no longer benefit from the dilution effect (Hugie and Dill, 1994). Under such circumstances, it is unlikely that competitor types will aggregate in a single habitat. Finally, in contrast to other foraging–predation risk models that treat food and safety as complementary resources (e.g. Brown, 1988, 1992), we assume that food and safety are perfect substitutes. As a consequence, individuals will treat habitats as alternative ways to maximize fitness (i.e. living a short time and experiencing a high reproductive rate or living a long time and experiencing a low reproductive rate), rather than requiring a mix of both.

Because our model can be applied to both intra- and interspecific resource competition, its results may help to predict circumstances under which stable co-existence of competitor types is likely to occur, and when we should expect divergent habitat 'preferences' and the beginnings of niche specialization. Ideal free distribution theory has long been heralded as a potential method of linking individual decision-making to population and community-level phenomena (see Kacelnik *et al.*, 1992; Rosenzweig, 1995; Sutherland, 1996). By considering more than a single competitor type, and differences between competitor types in habitat-specific patterns of mortality risk, we believe that we have strengthened this link.

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APPENDIX

We simulated solutions to the model and examined the stability of all equilibria produced using the finite evolutionary game dynamics described by equation (A1), based on the evolutionary difference equation described by Maynard Smith (1982) and Hofbauer and Sigmund (1988). In doing so, we assume that fitness represents the multiplication 'rate' (R_0) defined over the generation time. The finite change in the proportion of the ith competitor type in habitat A, Δp_i, over one time unit will be equal to:

$$\Delta p_i = p_i \frac{w_{i\mathrm{A}} - \bar{w}_i}{\bar{w}_i} \tag{A1}$$

where $\bar{w}_i$ is the mean fitness of all type i competitors, given by:

$$\bar{w}_i = p_i w_{i\mathrm{A}} + (1 - p_i)\, w_{i\mathrm{B}} \tag{A2}$$

When $\Delta p_i = 0$, the distribution of type i competitors will be at equilibrium, such that no individual can increase its fitness payoff by switching habitats. When competitor type i occurs in both habitats, for any equilibrium, $w(i, \mathrm{A}) = w(i, \mathrm{B})$. When competitor type i occurs exclusively in a single habitat (for example, habitat A), for any equilibrium, $w(i, \mathrm{A}) > w(i, \mathrm{B})$ for all p_i.

To test for local stability of equilibria, we add a perturbation factor (ε) to our simulations:

$$\Delta p_i = p_i \frac{w_{i\mathrm{A}} - \bar{w}_i}{\bar{w}_i} \pm \varepsilon \tag{A3}$$

such that, at each time step, a small, random number of type i competitors are either added to or subtracted from habitat A. We might think of ε as representing individuals who occasionally move between habitats as a consequence of imperfect information (e.g. Abrahams, 1986), or to escape from predators, search for mates or avoid agonistic encounters (e.g. Hugie and Grand, 1998). If, despite these random perturbations, an equilibrium is repeatedly returned to once reached, it can be said to be locally stable. In all cases, simulations rapidly converged on a single, stable equilibrium distribution of type 1 and 2 competitors ($\hat{p}_1$, $\hat{p}_2$).