

The population-dynamic functions of seed dispersal

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Keywords: Allocation costs, Bet-hedging, Breeding systems, Density dependence, Dispersal, Metapopulations, Patch structure, Sib competition

Abstract

We summarize some of the population-dynamic consequences of the mosaic structure of plant populations for the evolution of seed dispersal. A fairly elaborated set of theoretical ideas exist regarding the evolution of dispersal and we have synthesized some of them in an attempt to make them more accessible to field ecologists. We consider the relationship of these general theoretical ideas to our understanding of fruit and seed dispersal.

We develop three related models to describe the similarities and differences in how dispersal functions for risk reduction (bet hedging), escaping the negative consequences of crowding, and escaping high concentrations of relatives. We also briefly discuss directed dispersal as a fourth population-dynamic aspect of dispersal. Dispersal can have a risk-reducing function only when there is global (metapopulation) temporal variance in success. Dispersal to escape the negative consequences of crowding requires only spatial and local temporal environmental variation. Dispersal for escaping high concentrations of relatives requires no environmental variation, but does require genetic population structure. Directed dispersal, defined as non-random into particular patch types contingent on the expectation of local success, is always valuable when possible and represents an advantage independent the others which can occur with random dispersal.

In an effort to accommodate for the differences between simple mathematical models and the behavior of complex natural fruit and seed dispersal systems we have discussed the following issues: actual patterns of patch structure and dispersal distance; the implications of plant cosexuality, perennality, and allocation costs of dispersal structures; and the impact of the detailed nature of density dependence, breeding systems, and genetic structure. We briefly compare the population-dynamic functions of dispersal presented here with the widely cited functions of colonization, escape, and directed dispersal. Finally, we suggest how the theoretical models can be used with field data to estimate the fitness consequences of dispersal.

Introduction

As pointed out by John Harper (1977), an advantage of studying the population biology of plants is that they sit in one place and 'wait to be

counted'. Yet this same fixity of position is a double-edged sword. Rather than experiencing the average habitat quality of some potentially communal home range, plants experience the abiotic conditions of the local safe site or patch where

they germinated. Likewise, rather than interacting with a variety of individuals and experiencing the average density of their population, plants interact with specific individuals within a canopy or root crown diameter (Silander & Pacala 1990). Even different spatial distributions with the same overall density can have dramatically different consequences for population dynamics (Harper 1961; Bergelson 1990). Thus, the 'other edge' of fixity of position is that an understanding of plant population dynamics requires a understanding of the patchy spatial distributions of plants. Central to this is the phenomenon of seed dispersal.

To appreciate the status of research on the population-dynamic functions of seed dispersal, it is instructive to compare progress in this field to that in other branches of plant reproductive ecology. Plant reproductive ecology encompasses the areas of breeding systems, pollination, seed dispersal, dormancy and establishment, and possibly vegetative reproduction (though many prefer to consider the latter as simply a form of growth). Breeding system and pollination ecology have grown rapidly in the last 15 years due to increased interest in experimental plant population biology and an application of insights from ecological and evolutionary theory to the topic of plant reproduction (Lovett Doust & Lovett Doust 1988). The theoretical developments include a careful use of 'selection thinking', often phrased in terms of the ESS concept, and of ideas derived from the cost-of-sex concept, sex-ratio theory, and Bateman's principle (Charnov 1982; Lloyd 1983). The evolutionary ecology of seed and fruit dispersal has remained mainly at the natural history level with considerable interest generated by plant-animal interactions and patterns seemingly explained by coadaptation (Murray 1986; Estrada & Fleming 1986). The integration of theoretical and experimental approaches aimed at understanding fitness implications of evolutionary strategies has been less prominent in the seed dispersal literature than in the pollination and breeding system literature (but see Alvarez-Buylla & García-Barrios 1991; Olivieri & Gouyon 1985; and Horvitz & Schemske 1986 for some work in this direction). The reasons for the differences in

approach and rates of progress in these two branches of plant evolutionary ecology are no doubt varied. The fitness consequences of seed dispersal is an inherently complicated issue involving spatio-temporal patterns of microhabitat suitability (Cohen & Levin 1991) with no simple 'target' of the dispersal event (c.f. the 'target' of pollination – to land on a compatible stigma [Wheelwright & Orians 1982]). Also there are fewer simple rules of the kind that have been so useful in breeding system theory, such as the equalization of male and female fitness at the population level (Lloyd 1983).

We have at present a fairly well-elaborated, if somewhat scattered and diffuse, theoretical literature on how patterns of spatial and temporal heterogeneity favor the evolution of greater or lesser dispersibility (Johnson & Gaines 1990). Here we tie some of these diverse threads together into a more unified story and explore how this theory can be used to understand and investigate seed and fruit dispersal. A closer interaction of theoretical and experimental population-dynamic approaches may spur a period of more rapid advance in our understanding of seed dispersal similar to that seen for pollination and breeding systems ecology.

A few years ago when writing a review of the evolutionary ecology of seed banks, Venable (1989) was able to summarize the results of research on dormancy strategies by referring in a general way to a few intuitive formulae. We intended to take a similar approach in the present chapter, but immediately ran into trouble. First of all, the population-dynamic consequences of dispersal are more complicated than those of dormancy. While dormancy can be explored in models of spatially homogeneous environments experiencing temporal variation, the very nature of dispersal requires a consideration of population subdivision and the details of spatio-temporal variation. Thus as recently as 15 years ago John Harper (1977) was able to accurately state that, 'Attempts to determine the fittest dispersal strategies for specified environmental regimes have so far proved too complex to be handled other than through numerical experiments on digital com-

puters.' Today this is no longer true and many insights can be derived from a few simple analytical formulae. However, because of the inherent complications of dispersal models, the origin and logic behind them are less intuitive at first glance than for the models of dormancy. Finally, the three basic models to which we refer are probably less accessible elsewhere to the empirical ecologist interested in fruit and seed dispersal. As a result we have decided to provide a little more substance to the theoretical synthesis presented here.

We will begin by presenting three related models which explore three basic population-dynamic functions of dispersal for organisms in spatially structured populations, and then discussing a fourth function. We will use simple analytical models in an attempt to shed some light on the general nature and function of dispersal. For the sake of simplicity and hopefully clarity we will model each function in isolation to clearly distinguish its effects from those of other functions. Later in the chapter we will return and ask what are the consequences of relaxing some of the more important assumptions of the models and hopefully, in this manner, begin to get a feel for the importance of these processes in fruit dispersal systems. The four functions of dispersal that we will explore or discuss are risk reduction or bet hedging, escaping the negative consequences of crowding, escaping the negative consequences of high concentrations of relatives, and directed dispersal. These functions contrast somewhat with the widely cited functions of dispersal: colonization, escape from the vicinity of the parent, and directed dispersal (Howe & Smallwood 1982). We will briefly discuss these contrasts later in the chapter.

Models

In each model we concern ourselves with the aggregate behavior of a 'metapopulation' (*sensu* Levins 1970) structured into a set of separate 'cells' or 'patches'.

A risk-reduction model

In this model we only consider density-independent processes, thus we exclude the possibility of selective effects due to escape from crowding or interactions with relatives. We consider n patches, each of equal area. Conditions in each patch vary over time and we will assume, at least initially, that the variability in each patch is independent of variation in the others and can be described by the same random variable. Most of these assumptions can be easily relaxed but they are useful for examining the main points.

A recursive formula gives the number of individuals in a single patch, j , at time $t + 1$ as a function of the number of seeds in patch j and in all of the other patches at time t ,

$$N_{j(t+1)} = (1 - D)N_{j(t)}S_{ij} + \frac{Da}{n} \sum_{k=1}^n N_{k(t)}S_{ik}. \quad (1)$$

In (1), D is the 'dispersal fraction' i.e. the proportion of the seeds produced by an adult plant that disperse out of the home patch and into a 'dispersal pool'. For simplicity, we assume an annual life history so that S_{ij} describes the 'success' of seeds at reproducing (i.e. the product of their survival and fecundity) in patch j experiencing conditions i . Thus $i = 1, \dots, m$ indexes a random variable responsible for the independent variation in each of the patches. The seeds in a patch come from 2 sources: *in situ* reproduction, and dispersal into the patch. Seeds leaving the home patch enter a dispersal pool from which seeds fall evenly among all patches (i.e. an equal proportion of dispersing seeds ($1/n$) goes to each patch). It is assumed that dispersing seeds experience some cost of dispersal indexed by $a \in [0, 1]$ (i.e. dispersal is cost-free if $a = 1$). This formulation provides a caricature of the dispersal process that captures its essence though in a somewhat exaggerated fashion (i.e. the idea that dispersal involves sampling and averaging over a variety of sites).

We will briefly sketch 2 simple cases that illustrate most of the important points: no dispersal and 100% dispersal. With no dispersal, (1)

reduces to

$$N_{j(t+1)} = N_{j(t)} S_{ij}. \quad (2)$$

The per capita growth rate in patch j will equal

$$\lambda_{ij} = \frac{N_{j(t+1)}}{N_{j(t)}} = S_{ij}. \quad (3)$$

The per capita growth rate for the metapopulation will equal the sum over all patches of seeds at time $t+1$ divided by the sum over all patches of seeds produced at time t ,

$$\begin{aligned} \lambda_{\text{meta}} &= \frac{\sum_{j=1}^n N_{j(t)} S_{ij}}{\sum_{j=1}^n N_{j(t)}} = \sum_{j=1}^n \left(\frac{N_{j(t)} S_{ij}}{\sum_{j=1}^n N_{j(t)}} \right) \\ &= \sum_{j=1}^n \rho_j S_{ij} = \sum_{j=1}^n \rho_j \lambda_{ij} \end{aligned} \quad (4)$$

where ρ_j equals $N_{j(t)}/\sum_j N_{j(t)}$, the proportion of the global seed population that is found in patch j .

Equation (4) is the formula for the per capita growth rate of the whole metapopulation for a single year. But since the conditions in each patch vary from year to year, this growth rate will vary from year to year, and thus we must consider its average over time. Since population growth is multiplicative over time, we must use a multiplicative average, i.e. the geometric mean. The geometric mean (also population growth generally) is variance sensitive, as is illustrated in the simple example in Table 1. In fact, the geometric mean can be approximated as a variance-discounted arithmetic mean (Real 1980).

$$GEO(\lambda) \approx E(\lambda) - k\sigma^2(\lambda) \quad (5)$$

where $GEO(\cdot)$ stands for ‘the geometric mean of’ and $E(\cdot)$ stands for the ‘the expected value of’ or ‘arithmetic average of’ and k is a constant. This expression states that the geometric mean (temporal average) of the population growth rate is

Table 1. An example demonstrating that fitness is sensitive to temporal variance.

	Demographic success		
	Year		
	1	2	3
Phenotype A*	2	5	8
Phenotype B	4	4.5	5
	per capita growth after 3 years		
	= 80		
	= 90		

* In this example, the phenotype with higher mean and variance of success (A) actually had lower growth than the one with lower mean and variance (B).

smaller than the arithmetic mean growth rate by an amount proportional to the magnitude of the year-to-year variance in growth rate. ‘Bet hedging’ refers to a sacrifice of $E(\lambda)$ to increase fitness by reducing $\sigma^2(\lambda)$. Since this term has been used quite loosely in the literature to refer to a number of situations which do not involve a tradeoff between mean and variance (Seeger & Brockmann 1987), we will use the term ‘risk reduction’ to refer to bet hedging in this more technical sense.

We can understand the risk-reducing consequences of dispersal by understanding the effect of dispersal on the tradeoff between mean and variance of λ_{meta} . In what follows we rely on some of the simple rules of probability for expected values and variances which the reader may need to review to follow this section in detail (see e.g. Ostle & Mensing 1975). The expected value of λ_{meta} is

$$\begin{aligned} E(\lambda_{\text{meta}}) &= E\left(\sum_{j=1}^n \rho_j S_{ij}\right) = \sum_{j=1}^n \rho_j E(S_{ij}) \\ &= E(S_i) \sum_{j=1}^n \rho_j = E(S_i). \end{aligned} \quad (6)$$

Note that the expected value of the per capita growth of the metapopulation is the same as the expected value of the per capita growth rate of a particular patch

$$E(\lambda_{ij}) = E(S_{ij}) = E(S_i). \quad (7)$$

While the variance of a single patch is $\sigma^2(S_i)$, the variance of the metapopulation is

$$\begin{aligned}\sigma^2(\lambda_{\text{meta}}) &= \sigma^2\left(\sum_{j=1}^n \rho_j S_{ij}\right) \\ &= \sum_{j=1}^n \sum_{k=1}^n \rho_j \rho_k \sigma(S_{ij} S_{ik}).\end{aligned}\quad (8)$$

where $\sigma(x, y)$ refers to the covariance of x and y . Since patches are assumed to be independent and S is identically distributed across patches, this reduces to

$$\sigma^2(\lambda_{\text{meta}}) = \sum_{j=1}^n \rho_j^2 \sigma^2(S_{ij}) = \sigma^2(S_i) \sum_{j=1}^n \rho_j^2. \quad (9)$$

The term $\Sigma \rho_j^2$ is smaller the more patches there are and the more similar the ρ_j 's are (remember ρ_j 's are the proportion of the global seed population found in each patch). The intuition behind this can be understood by recognizing that $\Sigma \rho_j^2$ is an information statistic, biologically analogous (and mathematically equal to) the reciprocal of Simpson's diversity index. Remember that a diversity index is higher the more species there are and the more evenly distributed their abundances are. Similarly, $\Sigma \rho_j^2$ is lower the more patches there are and the more evenly distributed individuals are among patches.

Thus the conclusion for the density-independent risk-reduction model with zero dispersal are: 1) the expected value of λ is the same for one patch or many, but; 2) the variance of λ_{meta} (the global temporal variance) is a declining function of patch number and of the evenness of the spread of individuals among patches; 3) since the geometric-mean population growth can be expressed as a variance-discounted arithmetic mean, and the arithmetic mean is the same for 1 patch or many, the long-term-average population growth will be higher for metapopulations with more patches and with seeds more evenly spread among patches.

Now consider what happens with 100% dispersal. Since the model assumes uniform dispersal and equal patch size, an equal number of

dispersing seeds lands in each patch. With 100% dispersal, all patches will have the same growth rate in a given year (which comes entirely from the global dispersal pool). Thus the metapopulation growth rate equals the local patch growth rate.

$$\begin{aligned}\lambda_{ij} &= \frac{a}{n} \sum_{j=1}^n S_{ij}; \lambda_{\text{meta}} = \frac{1}{n} \sum_{j=1}^n \left(\frac{a}{n} \sum_{j=1}^n S_{ij}\right) \\ &= \left(\frac{a}{n} \sum_{j=1}^n S_{ij}\right) \sum_{j=1}^n \frac{1}{n} \\ &= \frac{a}{n} \sum_{j=1}^n S_{ij} = \lambda_{ij}.\end{aligned}\quad (10)$$

The mean and the variance of the growth rate are

$$\begin{aligned}E(\lambda_{\text{meta}}) &= E(\lambda_j) = E\left(\frac{a}{n} \sum_{j=1}^n S_{ij}\right) \\ &= a \sum_{j=1}^n \frac{E(S_i)}{n} = aE(S_i) \sum_{j=1}^n \frac{1}{n} \\ &= aE(S_i)\end{aligned}\quad (11)$$

$$\begin{aligned}\sigma^2(\lambda_{\text{meta}}) &= \sigma^2(\lambda_j) = \sigma^2\left(\sum_{j=1}^n \frac{a}{n} S_{ij}\right) \\ &= \sum_{j=1}^n \left(\frac{a}{n}\right)^2 \sigma^2(S_{ij}) = \frac{a^2 \sigma^2(S_i)}{n}.\end{aligned}\quad (12)$$

Compared to the case with no dispersal, with 100% dispersal the expected value of λ is lower by an amount determined only by the cost of dispersal (if dispersal is cost-free, the mean is the same for 0 and 100% dispersal).

Thus in this density-independent model, the only way dispersal can be of benefit is via reduction in the global temporal variance and the positive effect this has on geometric-mean fitness. The variance terms for 0 and 100% dispersal are similar and, aside from the cost of dispersal term, differ only in the term describing the distribution of seeds among patches. With 100% dispersal, seeds are distributed uniformly such that $\Sigma_j \rho_j^2$

takes on its lowest possible value for a given number of patches, $1/n$ (analogously, a Simpson's index takes on its highest possible value for a given number of species [maximum 'information'] when all species have equal abundance).

In summary, in this risk-reduction model, dispersal decreases arithmetic mean fitness. Thus, dispersal can only evolve for risk reduction if there is global (metapopulation) temporal variance and if the reduction in global temporal variance brought about by dispersal more than compensates for the decrease in arithmetic-mean fitness. Global temporal variance declines with the number of patches and with a more even distribution of seeds among patches. The risk-reducing function of dispersal is to reduce global temporal variance at the expense of lower arithmetic-mean fitness by spreading seeds more evenly among patches.

Escaping the negative consequences of crowding

Escaping the effects of crowding is a separate factor favoring dispersal that is functionally independent of risk reduction, and arises from different causes. To illustrate this we will construct a model with crowding effects but no risk reduction and use it to explore the evolution of dispersal. We will make a number of simplifying assumptions in order to get a general analytical model which will reveal some general properties of dispersal as a mechanism to escape from crowding. Let there be n patches, each of equal area. Let density dependence be described by a 'constant-' or 'reciprocal-yield law'. Density dependence acts locally within patches and 'total yield' can vary from site to site and from year to year. Thus this model takes into account the local nature of density interactions championed by Harper (1977) and Silander & Pacala (1990). The model is similar to one given by Levin *et al.* (1984).

In density-dependent models, individual fitness depends not only on an individual's own dispersibility, but also on the dispersibility and the resulting population dynamics of the plants with which it interacts. To deal with this context- and frequency-dependent selection, we must either use

explicitly genetic models or an ESS (evolutionarily stable strategy) approach. Since, as ecologists, we seldom know the genetic details underlying the phenomena we study, and since population-genetic models are usually more complicated (Taper and Case, 1992), we will use the ESS approach. This permits the incorporation of greater ecological sophistication, maintains mathematical tractability, and in most instances captures the essence of individual-level natural selection (Vincent & Brown 1988).

One simple way to construct a model to explore the influence of dispersal on avoiding crowding is to consider competition for a fixed number of 'safe sites'. We can define a function (known as a fitness-generating function, *sensu* Vincent & Brown 1988) which gives the number of post-competition safe sites occupied by plants with an arbitrary dispersal type, D , (sometimes called the 'mutant' strategy) within a metapopulation dominated by individuals using strategy \hat{D} (the 'resident' strategy). For example, we might assume that seeds compete for safe sites at germination. Alternatively, safe sites could represent canopy sites for adults or neighborhood areas for reproduction. For this model it is critical that the density-dependent effect be local (within patches) but it is not critical which life stage is represented by N (though this does become critical in the next model).

To avoid the evolution of dispersal as a mechanism for risk-reduction, in this model we assume that the number of patches, n , is very large; i.e. $n \rightarrow \infty$. As the number of patches gets large, the variance over time of the per capita rate of increase of the metapopulation approaches zero and the geometric-mean fitness approaches the arithmetic mean. That is, the long-term-average per capita growth rate of the metapopulation is the same as the spatial average across patches in any one year (cf. flip a fair coin once for each of an infinite number of patches and you will get 50% heads each year and a temporal average of 50%).

To avoid the evolution of dispersal as a mechanism for escaping sib interactions, assume that the number of safe sites per patch, N , is suffi-

ciently large that the contribution of any one plant to the seed production of the patch is negligible (an analogous assumption is often made in sex allocation problems to eliminate local mate competition, a related phenomenon, cf. Lloyd 1984). Relaxation of this assumption forms the topic of the next section. Under the assumptions of many patches and many safe sites within a patch, we can now formulate the fitness-generating function.

The expected per capita growth rate of an individual using strategy D can be described by the number of safe sites occupied in the following year for each safe site occupied during the present year. To simplify our analysis and without loss of generality, we assume that all patches have a same number of safe sites. The per capita increase in the number of safe sites can be separated into a contribution from *in situ* reproduction and a contribution from seeds dispersing out of the patch. We will assume 'fair-lottery density dependence' (i.e. that safe sites are won in proportion to propagule availability). Thus the probability of an individual 'winning' a particular safe site within its natal patch is given by the proportion of all seed landing in the patch that are produced by the individual. This is given by dividing the number of the individual's non-dispersing seeds, $(1 - D)S_i$, by the sum of all non-dispersing seeds produced within the patch, $N(1 - \hat{D})S_i$, and all seeds that disperse into the patch from elsewhere, $an\hat{D}\bar{S}/n$. Note that the seeds dispersing into the patch come from sampling the spatial average of the environment and so the average seed production of a dispersing seed's parent is \bar{S} . The total number of safe sites won by the mutant individual within its natal patch, Y_{home} , is the product of this 'proportion won' and the number of safe sites:

$$\begin{aligned} Y_{\text{home}} &= N \cdot \frac{(1 - D) S_i}{N(1 - \hat{D}) S_i + aN\hat{D}\bar{S}} \\ &= \frac{(1 - D) S_i}{(1 - \hat{D}) S_i + a\hat{D}\bar{S}}. \end{aligned} \quad (13)$$

Assuming fair-lottery density dependence, the probability of an individual 'winning' a particular

safe site within a non-natal patch is given by the ratio of the number of its dispersing seeds landing in the patch divided by the sum of all non-dispersing seeds produced within the patch, $N(1 - \hat{D})S_j$, and all seeds that disperse into the patch from elsewhere, $an\hat{D}\bar{S}/n$. Assuming uniform dispersal, the total number of dispersing seeds landing in all of the patches experiencing conditions j will be $p_j aDS_i$. Since there are N safe sites to be 'won' in each patch, the dispersing seeds will capture a total of $Np_j aDS_i / (N(1 - \hat{D})S_j + aN\hat{D}\bar{S})$ safe sites summed over all the patches experiencing conditions j . Summed over all patches types, the total number of safe sites won by the individual in non-natal patches, Y_{away} , is

$$\begin{aligned} Y_{\text{away}} &= \sum_{j=1}^m p_j \cdot \frac{NaDS_i}{N(1 - \hat{D})S_j + aN\hat{D}\bar{S}} \\ &= \sum_{j=1}^m \frac{p_j aDS_i}{(1 - \hat{D})S_j + a\hat{D}\bar{S}}. \end{aligned} \quad (14)$$

The increase of an individual using strategy D in a particular year will be given by the sum of Y_{home} and Y_{away} , which will vary depending on the conditions, i , experienced in the home patch. Since an arbitrary individual using strategy D might occur in any patch type, $i = 1, \dots, m$, its *expected* per capita increase in a metapopulation composed of individuals using \hat{D} is given by $\sum p_i (Y_{\text{home}} + Y_{\text{away}})$. Thus the fitness-generating function for this model is

$$\begin{aligned} G(D, \hat{D}) &= \sum_{i=1}^m p_i \left[\frac{(1 - D)S_i}{(1 - \hat{D})S_i + a\hat{D}\bar{S}} \right. \\ &\quad \left. + \sum_{j=1}^m \frac{p_j aDS_i}{(1 - \hat{D})S_j + a\hat{D}\bar{S}} \right] \\ &= \sum_{i=1}^m \frac{p_i (1 - D)S_i}{(1 - \hat{D})S_i + a\hat{D}\bar{S}} \\ &\quad + \sum_{j=1}^m \sum_{i=1}^m \frac{p_j p_i aDS_i}{(1 - \hat{D})S_j + a\hat{D}\bar{S}} \end{aligned}$$

$$\begin{aligned}
&= \sum_{i=1}^m \frac{p_i(1-D)S_i}{(1-\hat{D})S_i + a\hat{D}\bar{S}} \\
&\quad + \sum_{i=1}^m \frac{p_j a D \bar{S}}{(1-\hat{D})S_j + a\hat{D}\bar{S}} \\
&= \sum_{i=1}^m p_i \left(\frac{(1-D)S_i + aD\bar{S}}{(1-\hat{D})S_i + a\hat{D}\bar{S}} \right). \quad (15)
\end{aligned}$$

This fitness-generating function is simple and useful in its final form, though it may not be very intuitive at first glance. It is the formula for the expected per capita rate of increase in the number of safe sites occupied by an individual using an arbitrary dispersal strategy D in a metapopulation with strategy \hat{D} where all plants experience fair-lottery density dependence in local patches. The derivation tells us that this proportional increase in safe sites can be calculated simply as an average of the ratio of the reproductive success that a mutant and resident can expect to have in each patch type. The numerator of this ratio can be thought of as the non-dispersing seeds produced locally by the mutant strategy plus the dispersing seeds of the mutant sampled from all patch types. The denominator is the same for the resident strategy. Thus the fairly complicated density-dependent rate of increase we desire reduces to a fairly intuitive average of the ratio of per capita mutant and resident reproductive success. This suggests a simple way of calculating expected fitness for different dispersal strategies from easily-collected field data, which we will discuss in a subsequent section of this paper.

When the average of this ratio over all patch types is greater than 1, the dispersal type D will increase in frequency. In the bet-hedging model of the previous section, dispersal reduced variance at the expense of the cost of dispersal. In the present model, spatial and local temporal variances are being reduced rather than global temporal variance (which we have explicitly assumed away by making a very large number of patches). The selective force here is to escape from the negative effects of density which are created by spatial variation in success. With no dispersal,

there will be much variance in density between patches prior to the action of density dependence. Even the uniform (non-directed) dispersal used in these models will tend to move seeds from areas of high density to areas of low density. This is because in any given year, most mutant seeds are produced in areas that will have high seed density in the following year since resident plants do well at the same times and places as mutants. Thus, if a mutant disperses more seeds than the resident, it can have high per capita increases when it arrives in sites with low *in situ* success. Dispersal is a way of averaging across variable patches, which allows plants to escape the negative effects of spatial variation, but plants must pay the cost of dispersal. Increased dispersal evolves when the benefits of landing in patches where residents did poorly (because of spatial and local temporal environmental variation) outweigh the cost of dispersal. Some simple numerical examples to illustrate this are given in Table 2.

The examples in Table 2 also illustrate that the fitness of a given dispersal type, D , depends the dispersal strategy of others, \hat{D} (compare cases 2 and 3). In such situations we can use the ESS concept to find the fitness-maximizing dispersibility. An ESS is a strategy which, when common, cannot be invaded by rare alternative strategies (Maynard-Smith & Price 1973). The ESS problem can be conceived of informally as follows (Fig. 1). The dispersal strategy D represents the strategy of an individual in a metapopulation of individuals using \hat{D} . Natural selection should favor the strategy D that maximizes fitness in this context. As long as there is genetic variance for dispersibility, such selection will, in turn, change the dispersal strategy of the population, \hat{D} . This process of shifting \hat{D} towards the dispersal strategy that maximizes individual fitness (which may also be changing as \hat{D} changes) should continue until the two coincide (Fig. 2). At this point (the ESS) there will be no further selection for change because the dispersal strategy that maximizes the fitness of the individual is the same as the population's dispersal strategy. Any other rare alternative dispersibility will have lower fitness (at the

Table 2. Some examples with the model for escaping the negative effects of crowding showing how dispersal can increase fitness by reducing spatial variance^a.

Patch types					
Case	1	2	3	Mean ^b	
1	8/8	5/5	3/3	1	Mutant and resident are identical
2	5/8	5/5	5/3	1.1	Greater dispersal of the mutant provides cost-free variance reduction (dispersal invades).
3	5/9	5/5	5/1	2.1	Greater variance in the success of the less dispersive resident (dispersal invades).
4	4/8	4/5	4/3	0.88	Cost of dispersal is too high for the variance reduction achieved.
5	4.56/8	4.56/5	4.56/3	1	Break-even cost of dispersal.

^a The formula for this model calculates the average of the ratio for each patch type of mutant over resident demographic success $\sum p_i(M/R)$ where mutant success is given by $M = (1 - D)S_i + aD\bar{S}$ and resident success is given by $R = (1 - \hat{D})S_i + a\hat{D}\bar{S}$. (see equation (15) and its explanation in the text). For these examples assume three equiprobable patch types, $p_1 = p_2 = p_3 = 1/3$ and that the resident strategy is non-dispersal, i.e. $\hat{D} = 0$. For case 1 the mutant strategy is non-dispersal ($D = 0$) but for the other cases the mutant individual disperses all of its seeds ($D = 1$). Note that complete dispersal of the seeds of the mutant individual eliminates variance in the numerator. In case 2 and 3, this is done at no cost, but in cases 4 and 5, the cost of dispersal reduces the mean success of the mutant individual.

^b The mean is the mutant fitness defined as the *per capita* increase in number of ‘safe sites’ occupied by the mutant.

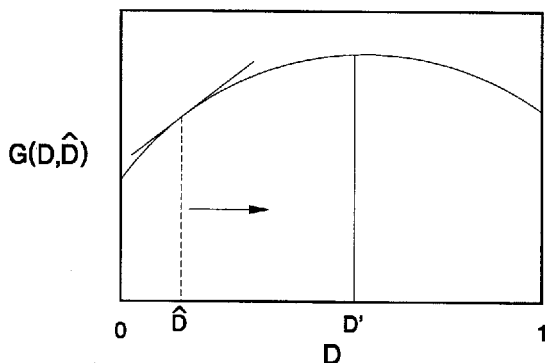


Fig. 1. $G(D, \hat{D})$, the fitness of a ‘mutant’ individual with dispersal D is plotted against D for a given dispersal fraction \hat{D} in the rest of the metapopulation. In the example illustrated, individuals with dispersibility slightly higher than \hat{D} have higher fitness than the rest of the metapopulation (as indicated by the positive fitness slope at \hat{D} and the position of the fitness maximizing mutant dispersibility, D' to the right of \hat{D}). The arrow indicates the direction of selection on \hat{D} . The shape of the mutant fitness curve may change as \hat{D} evolves.

ESS there is no selection for a unilateral change in strategy). Computationally, the ESS can be found by applying an ESS maximum principle (Vincent & Brown 1988) and setting the derivative of the fitness-generating function with respect to the individual’s dispersibility, D , equal to zero (to determine the dispersibility that maximizes an individual’s fitness). The solution to the resulting

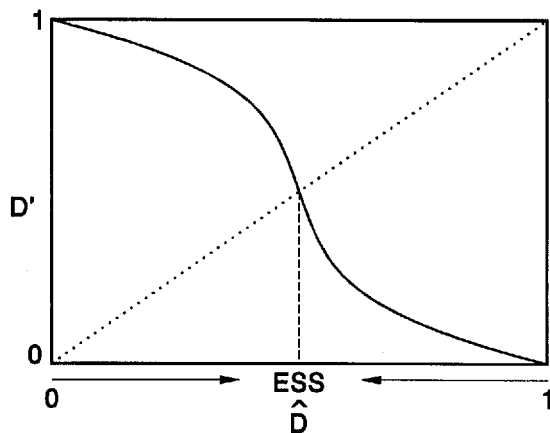


Fig. 2. A hypothetical plot of dispersibility that maximizes individual fitness (D') for the full range of possible dispersibilities in the metapopulation (\hat{D}). For any dispersibility to the left of the ESS, selection will favor a shift to the right (indicated by arrow) because the dispersibility that maximizes individual fitness is greater than the resident dispersibility. Likewise, for any resident dispersibility to the right of the ESS, selection will favor a shift to the left because the individual-fitness-maximizing dispersibility is lower. The ESS occurs where the 2 coincide.

expression must then be found for the situation in which $D = \hat{D} = D^*$, the ESS dispersibility.

Before solving for the ESS we will ask a related question: what is required for dispersal to be of any value at all in this escape-from-density sys-

tem, i.e., under what circumstances will dispersal be favored in a metapopulation without dispersal? This is the same as asking, when is $\partial G/\partial D|_{D=\hat{D}=0} > 0$ (c.f. Fig. 1)? Calculating the derivative gives:

$$\frac{\partial G}{\partial D} = \sum_{i=1}^m p_i \left(\frac{-S_i + a\bar{S}}{S_i(1-D_r) + aD_r\bar{S}} \right)$$

$$\left. \frac{\partial G}{\partial D} \right|_{D=\hat{D}=0} = a \sum_{i=1}^m p_i \frac{\bar{S}}{S_i} - 1. \quad (16)$$

Thus $\partial G/\partial D|_{D=\hat{D}=0} > 0$ requires that

$$a \sum_{i=1}^m p_i \frac{\bar{S}}{S_i} > 1 \Rightarrow a\bar{S} \sum_{i=1}^m p_i \frac{1}{S_i} > 1 \Rightarrow \frac{a\bar{S}}{H(S_i)} > 1 \quad (17)$$

where $H(S_i)$ is the harmonic mean of S (the harmonic mean is the reciprocal of the average of reciprocals, i.e. $1/E(1/S_i)$). Thus dispersal will be favored in a metapopulation without dispersal whenever the harmonic mean of success at reproduction is less than the dispersal-cost-discounted average success. As long as there is any variation, the harmonic mean is lower than the geometric mean which is lower than the arithmetic mean. These differences among means disappear in the absence of variance and increase with increasing variance. Thus in the absence of variation in S_i (spatial and local temporal variance) dispersal cannot evolve in this model. The larger the variance, the easier it is for (17) to be true and consequently the easier it is for a dispersing morph to invade. Also, the greater the cost of dispersal (lower a), the harder it is for dispersal to be favored. As long as there is any variance in success, cost-free dispersal would be favored in a resident population with no dispersal. The greater the variance, the greater the cost of dispersal that can be tolerated to obtain improved dispersibility. Thus (17) proves the intuition given above and in Table 2.

The other 'boundary' question is, when, if ever, can dispersal go to fixation, i.e. when will $D^* = 1$ be the ESS? Solving for $\partial G/\partial D|_{D=\hat{D}=1} \geq 0$ gives

$a \geq 1$. Thus D^* can only be 1 if dispersal is cost-free (i.e. if $a = 1$).

If, for a particular system, we determine that the ESS dispersibility is greater than zero and less than 1, we can calculate it by setting $\partial G/\partial D = 0$ and then substituting $D = \hat{D} = D^*$. This gives the ESS dispersibility implicitly as,

$$\sum_{i=1}^m p_i \frac{a\bar{S}}{S_i(1-D^*) + aD^*\bar{S}}$$

$$= \sum_{i=1}^m p_i \frac{S_i}{S_i(1-D^*) + aD^*\bar{S}}. \quad (18)$$

The formula can be used to calculate D^* for different parameter values and to explore how changing parameter values changes D^* . A simpler expression for the ESS can be obtained by assuming 2 patch types, $i = 1, 2$:

$$\frac{p_1}{a\bar{S}} + \frac{p_2}{a\bar{S}}$$

$$D^* = 1 - \frac{p_1}{S_2} \frac{1}{1 - \frac{p_2}{S_1}}. \quad (19)$$

Since $\partial D^*/\partial a$ is always positive for this expression, the smaller the cost of dispersal (i.e. the larger the values of a), the higher the ESS dispersibility, all else being equal. The relationship of D^* to the magnitude of environmental variation (i.e. the difference between S_1 and S_2) is less straight forward, though if variance is sufficiently large, D^* will increase with variance. This is not necessarily true when variance is small (cf. Levin *et al.* 1984). The driving force for the evolution of dispersal in this model is escaping spatio-temporal variance. Thus it makes sense that for parameter values at which variance is not very important, other negative factors associated with dispersal may outweigh the variance escaping benefits of dispersal. Such factors include a high cost of dispersal and a high probability that dispersing seed will land in an unfavorable patch.

If there is complete reproductive failure in one of the patch types so that $S_1 = 0$, then (19) reduces to

$$D^* = \frac{p_1}{1 - ap_2}. \quad (20)$$

From this equation we see that the greater the probability of complete reproductive failure in patches, the greater the ESS dispersibility (as long as dispersal is not completely cost-free). Also, the greater the cost of dispersal the lower the ESS dispersibility.

We can obtain an even simpler expression by adding the assumption that $p_1 = p_2 = 0.5$: $D^* = 1/(2 - a)$. This means that if the probability of encountering a patch with complete reproductive failure is 0.5, the ESS dispersal is to disperse at least half of the seeds, even if it is almost suicidal to do so (i.e. even if a is very low). The same ESS formula will show up again in the next model, but the two results should not be confused. Here the driving force for the evolution of high dispersal rates is the high probability of total failure for non-dispersing seeds and there are no sib interactions. In the next model, the high dispersal rates evolve to reduce sib interactions in a constant environment.

In summary, because the problem of crowding is inherently frequency-dependent, it should be thought of in ESS terms (i.e. by analyzing the fitness of an individual given a particular average or resident strategy) or else in explicitly genetic terms. In a density-dependent model where crowding is a problem, dispersal can evolve in the absence of the bet-hedging effect which we showed in the previous model to be due to global temporal variance. While, global temporal variance is no longer required, there must be spatial and local temporal environmental variation to make it possible for dispersal to alleviate crowding. Dispersal will evolve when the benefits to a mutant of landing in the uncrowded patches where residents did poorly outweigh the costs of dispersal.

Escaping the negative consequences of high concentrations of siblings

In the previous model bet hedging was eliminated by having no global temporal variance in fitness and sib-interaction effects were eliminated by allowing so many individuals per patch that an in-

dividual's seed production represented a negligible part of the patch total. Now we will relax this latter assumption to explore the effects of sib-density interactions (c.f. Hamilton & May 1977; Schoen & Lloyd 1983).

We will start with most assumptions of the previous model such as constant-yield density effects and patches of equal area. To avoid bet hedging we assume many patches. However, we will ensure that the selective factors involved in the previous model do not operate by assuming temporal constancy, i.e. $i = 1$, $S_i = S$.

The per capita growth rate of an individual using strategy D can be described in a fashion similar to that in the previous model focused on crowding. We consider the growth rate of an arbitrary individual with dispersibility, D in a metapopulation in which others have dispersibility, \hat{D} . Per capita growth rate in the sum of sites gained in the natal patch, Y_{home} , and sites gained in non-natal patches through dispersal, Y_{away} . In determining Y_{home} , we now include the effect of the individual plant with dispersibility D on the sum of all non-dispersing seeds produced within the patch. The sum of all non-dispersing seeds is now given by: $(1 - D)S + (N - 1)(1 - \hat{D})S$. Assuming fair-lottery density dependence, the probability of an individual 'winning' a particular safe site within its natal patch is given by the ratio of its non-dispersing seed, $(1 - D)S$, divided by the sum of all non-dispersing seeds produced within the patch and all seeds that disperse into the patch from elsewhere, $aN\hat{D}S/n$. The total number of safe sites won by an individual within its natal patch, Y_{home} , is the product of this ratio and the number of safe sites:

$$Y_{\text{home}} = N \cdot \frac{(1 - D)S}{(1 - D)S + (N - 1)(1 - \hat{D})S + aN\hat{D}S}$$

$$= \frac{N(1 - D)}{(1 - D) + (N - 1)(1 - \hat{D}) + aN\hat{D}} \quad (21)$$

Assuming fair-lottery density dependence, the probability of an individual winning a particular safe site within a non-natal patch is given by the ratio of the number of its dispersing seeds land-

ing in the patch divided by the sum of all non-dispersing seeds produced within the patch, $N(1 - \hat{D})S$, and all seeds that disperse into the patch from elsewhere $aN\hat{D}S/n$. Even though the total number of dispersing seeds produced by one mutant individual, aDS , may be large, we assume that the number landing in any particular non-natal patch is small. Thus it need not be considered in the denominator of the proportion of safe sites captured by the mutant. The total number of safe sites won by this individual within non-natal patches, Y_{away} , is the product of this proportion and the number of safe sites, summed over all patches:

$$Y_{\text{away}} = \frac{aDNS}{N(1 - \hat{D})S + aN\hat{D}S} = \frac{aD}{(1 - \hat{D}) + a\hat{D}}. \quad (22)$$

The per capita growth rate of mutant individual using an arbitrary strategy D in a population composed of individuals using \hat{D} is given by the expected value of the sum of Y_{home} and Y_{away} . This is the fitness-generating function for the sib interaction model:

$$G(D, \hat{D}) = \frac{(1 - D)N}{(N - 1)(1 - \hat{D}) + Na\hat{D} + (1 - D)} + \frac{aD}{1 - \hat{D} + a\hat{D}}. \quad (23)$$

Remember that, in the ESS is zero dispersal for the environment specified in this model (no local or global temporal or spatial environmental variation). Let us now ask, in this model with sib interactions, under what conditions (if any) will a non-dispersing strategy be invadable by a dispersal strategy with D greater than zero? These conditions are found by solving for $\partial G / \partial D |_{D = \hat{D} = 0} > 0$:

$$\frac{\partial G}{\partial D} = \frac{-N[(N - 1)(1 - \hat{D}) + Na\hat{D} + 1 - D] + (1 - D)N}{[(N - 1)(1 - \hat{D}) + Na\hat{D} + 1 - D]^2} + \frac{a}{1 - \hat{D} + a\hat{D}}$$

$$\left. \frac{\partial G}{\partial D} \right|_{D = \hat{D} = 0} = \frac{-N(N - D) + (1 - D)N}{(N - D)^2} + a$$

$$\left. \frac{\partial G}{\partial D} \right|_{D = \hat{D} = 0} > 0 \Rightarrow \frac{N - N^2 + a(N - D)^2}{(N - D)^2} > 0$$

which implies,

$$aN^2 > N^2 - N \Rightarrow N(1 - a) < 1. \quad (24)$$

Thus, a non-zero dispersal type can invade a resident non-dispersal type whenever $N(1 - a) < 1$; otherwise, $D^* = 0$ is the ESS. This means that, for dispersal to evolve via sib-density effects, N , the number of safe sites per patch, must be small or the cost of dispersal must be small.

Before solving for the ESS we will explore what happens at the other boundary, $D = 1$, by determining under what conditions (if any) dispersal will go to fixation (i.e. when will $D^* = 1$ be the ESS?). This is the same as solving $\partial G / \partial D |_{D = \hat{D} = 1} \geq 0$:

$$\left. \frac{\partial G}{\partial D} \right|_{D = \hat{D} = 1} = \frac{-N(Na + 1 - D) + (1 - D)N}{(Na + 1 - D)^2} + 1 \geq 0. \quad (25)$$

With some algebraic manipulation this reduces to $a \geq 1$ which implies that $D = 1$ can only be an ESS (and a neutrally stable one at that) if there is no cost to dispersal. Otherwise, the ESS dispersal will be less than 1 (this is the same condition for $D^* = 1$ as for the previous model).

The ESS dispersal fraction can be calculated using the ESS maximum principle. Set $\partial G / \partial D = 0$ for $D = \hat{D} = D^*$ and solve for D^* :

$$\frac{(N - 1 - ND^* + D^* + NaD^*)}{N(1 - D^* + aD^*)^2} = \frac{a}{1 - D^* + aD^*}$$

$$D^* = \frac{1 - N(1 - a)}{1 - N(1 - a)^2}. \quad (26)$$

which can take on any value between 0 and 1 depending on parameter values (assuming that $N(1 - a) < 1$; otherwise, the ESS is $D^* = 0$).

Thus, under sib competition, dispersal can be favored even in the absence of spatial and local temporal environmental variance. What favors

dispersal is variance in how many relatives occur in a particular patch. With small N , the non-dispersing offspring of a single individual may form a large fraction of the individuals interacting within the natal patch. There are diminishing returns that can be achieved in the natal patch by an individual having lower and lower dispersal since the individual would only be gaining a higher and higher probability of winning a limited 'prize' (much as in the case of local mate competition in models of the evolution of sex allocation, see Lloyd 1984). As modelled, there are no diminishing returns to an individual from dispersal. Dispersing seeds land in low densities in patches where the mutant individual did not reproduce. While avoiding diminishing returns in the natal patch, these seeds must pay the cost of dispersal which is an inherent constant disadvantage of dispersal. The ESS dispersal fraction occurs where the returns to an individual from non-dispersal diminish to the point where they equal the constant cost-discounted returns on dispersal. If N is large, returns on non-dispersal diminished less rapidly and complete non-dispersal may be favored unless dispersal is relatively cost-free. If the cost of dispersal is very small (a close to 1) the ESS dispersal fraction declines almost linearly with increasing N (within the permitted range of N given by $N(1-a) < 1$).

If $N = 1$, $D^* = 1/(2-a)$ which is the result obtained by Hamilton and May (1977). In this case the ESS dispersal fraction varies from 1/2 to 1 even if the cost of dispersal is very high (i.e. even if a is very low; this same ESS condition was found in the previous model, but under a very different set of assumptions). However, if N is the slightest bit greater than 1, there will be a maximum cost (minimum a) beyond which $D^* = 0$. Also, low dispersal fractions may be the ESS if N is larger than 1. Because of the critical importance of the magnitude of N , the life stage at which density limitation occurs becomes critical in these sib models (since there are likely to be many more seedlings per patch than adults).

In summary, in this sib interaction model, dispersal can evolve in the absence of spatial and local temporal variation, but only if the number of

individuals per patch is small or the cost of dispersal is small. If $N = 1$, a high dispersal fraction is favored even in the face of virtually suicidal costs involved in dispersal. Yet as long as $N > 1$ by even a tiny amount, there is a maximum cost beyond which dispersal will not be favored, and low dispersal fractions may be ESS's. The life stage at which density limitation occurs is critical in these models.

Directed dispersal

We will not say much about directed dispersal other than to relate it to the general modelling scheme presented so far. In the previous models, dispersal was considered to be random with respect to the occurrence of favorable conditions in patches. Directed dispersal refers to the fact that, by attracting the appropriate vector, or having the appropriate morphology, a plant may be able to vary its dispersal into a patch or habitat depending on the likelihood of its success in that patch. There are two aspects to the definition of directed dispersal. First, seeds must land disproportionately in a subset of patch types. Secondly, in order to have 'adaptive' directed dispersal, these patch types must be a favorable subset. The possibility has been discussed extensively in the empirical seed dispersal literature (Howe & Smallwood 1982; Hanzawa *et al.* 1988) and this kind of behavior is regularly considered in models of animal movement (Swingland & Greenwood 1983).

This situation is readily modelled in terms of conditional probabilities. If some cue is available upon which a plant can condition its dispersal into a patch, the plant should use it to the extent that it is a successful predictor of growth conditions. For example, ants should be used as vectors to the extent that ant middens are better-than-average sites for growth (Hanzawa *et al.* 1988), and 'gulpers' (*sensu* Moermond & Den-slow 1985) should be used as vectors to the extent that associated fecal matter functions as fertilizer. In the risk-reduction and escape-from-crowding models, directed dispersal implies that D can vary contingent upon some cue (e.g. pres-

ence of a gap or ant nest) which is predictive of either poor reproduction in the recent past (and thus indicative of low density in the present) or high reproduction in the near future (indicating high potential success). Theoretically, dispersal into a patch could be contingent on the density of relatives, thus extending the directed dispersal concept to sib-interaction models. Yet it is difficult to conceive of a realistic mechanism to achieve this. If dispersing seeds arrive nonrandomly at a favorable subset of patches, selection for dispersibility will be stronger than otherwise predicted in the previous models. Predictive dispersal is directly analogous to the phenomenon of predictive dormancy (Venable & Lawlor 1980; Venable 1989).

Real seed dispersal systems

In the previous section we presented three heuristic models in an attempt to explain four general population-dynamic functions of dispersal. With these caricatures of the dispersal process we tried to capture the essential population-dynamic properties of dispersal in models that are analytically tractable and simple enough that we can readily grasp the intuition behind the results. Yet an understanding of the population-dynamic consequences of seed and fruit dispersal requires an accommodation for the differences between the assumptions of the relatively simple mathematical models and the behavior of complex natural seed dispersal systems. In the rest of the paper we would like to discuss this accommodation.

Patch structure, dispersal distance, and implications for bet hedging

To understand the fitness consequences of seed dispersal, we must know the actual patterns of spatial and temporal variance in the environments experienced by plants as well as know how dispersal distance corresponds to the scale of spatial and temporal variance. These are big questions for which we only have partial answers at

present. Review articles have been written on each of these issues (White 1979; Pickett & White 1985; Willson, this volume; Levin & Kerster 1974).

Ecological patch structure is a species-specific property consisting of biotic and abiotic components. Thus it is most readily explored on a species-by-species basis, focussing on plant responses rather than measuring environmental parameters (the phytometer approach; Antonovics & Primack 1982; Clements & Goldsmith 1924).

Real patch structures are likely to be more or less continuous with spatial and temporal auto-correlations. Such correlations are expected to have strong effects on the fitness consequences of dispersal (c.f. Venable & Brown 1988 for an analysis of their effects on a risk-reduction model of dispersal). Real patch structures are likely to be hierarchical, with different processes occurring at different spatial and temporal scales. For example, solitary treefalls typically occur at small spatial and temporal scales, while hurricanes or epidemics may result in a superimposed patch structure occurring at a much larger spatial and temporal scale.

Mathematical tools are available for succinctly describing how the texture of environmental variation changes at different spatial and temporal scales (e.g. applied fractal geometry; Sugihara & May 1990). There are probably some systems, for example those involving dominant plant species in low diversity communities, for which a good idea of patch structure could be obtained from fractal geometric analyses of aerial photographs (c.f. Krummel *et al.* 1987). Such analyses can be performed on time series data as well, which might be useful for determining how much variation is occurring at different temporal scales in local patches (Sugihara & May 1990). However, since the property undergoing variation is a plant species-specific perception of environmental variation, the immediate problem for most plant demographic systems is the initial collection of the proper data, not its succinct mathematical description.

Real seed dispersal patterns tend to be leptokurtic, often described successfully by a negative exponential distribution, with most seeds

travelling on a scale of meters (Willson, this volume). Limited seed dispersal creates an interesting dilemma with respect to the risk-reducing function of dispersal. Remember that dispersal only evolves for risk-reduction in systems in which global temporal variance is important and that global temporal variance declines with patch number. It would seem that risk reduction may not be an important function of seed dispersal for most widespread plant species because of one of the following factors. First, a widespread plant species occurs in so many patches that global temporal variance may be low. How many patches are 'many'? Recall from the risk-reduction model above that, assuming independent patches and uniform plant density across patches, global temporal variance equals the local temporal variance divided by patch number. Under these assumptions, global temporal variance is dramatically reduced with 20–50 patches. Accounting for spatial and temporal autocorrelation of conditions and the uneven distribution of plants among patches, the number of patches necessary to dramatically reduce global temporal variance will increase by some generally unknown amount (though calculable in specific cases).

If global temporal variance is high in a widely distributed species, it is probably generated by processes occurring at a spatial scale not readily travelled by dispersing seeds. For example, weather phenomena, such as El Niño events, are likely to create global temporal variance for many plant species, but the spatial scale of variation is so great that few seeds can be expected to travel the necessary distances (c.f. Ellner & Shmida 1981). Under these circumstances, risk-reducing adaptations are likely to evolve, but the plant traits affected are more likely to be such things as seed dormancy or fire resistance rather than seed dispersibility (see Venable & Brown 1988 for a discussion of the fitness interactions of dispersal, dormancy, and other traits, such as seed size with respect to risk reduction).

These comments refer only to widespread plant species. Seed dispersal might have a very important risk-reducing function in narrowly distributed species, and the role of dispersal in risk re-

duction should not be ignored when considering conservation measures for such species (c.f. Simberloff 1989).

This discussion leads logically to another possible risk-reducing role of dispersal, i.e. increasing the number of patches by expanding the species range. While a logical possibility, we did not mention this previously because we are not aware of any evidence supporting it. This would be an interesting empirical question to pursue further: do related taxa which differ in dispersibility also differ systematically in the sizes of their range of distribution?

The implications of plant cosexuality for seed dispersal

To measure dispersal distances, most plant ecologists find some technique for measuring the distances each seed travels from its mother (Willson, this volume). The resulting distribution is considered to give the seed dispersal curve for that species. It is important to recognize that this approach only describes dispersal for approximately 1/2 of the offspring of an outcrossing cosexual plant, i.e. those produced through female function. The other 1/2 typically travel much further than we usually think. Seeds resulting from a plant's paternal function fall in the seed shadows of all of the mates of the plant in question and, by definition travel a greater distance and cover a much larger area (Fig. 3; c.f. Schoen & Lloyd 1983). These seeds carry just as many parental genes as the ones actually filled on the parent, and they should be considered when estimating seed dispersal distance as part of any attempt to understand the fitness consequences of seed dispersal.

Since most plant species are cosexual, most plants will produce (and disperse) seeds via both male and female function. This observation is a direct result of the recognition by breeding system ecologists that we have been ignoring male function in estimates of plant fitness (Bertin 1988; Stanton & Galloway 1991). For example, uniparental plants (selfers and asexuals) will only dis-

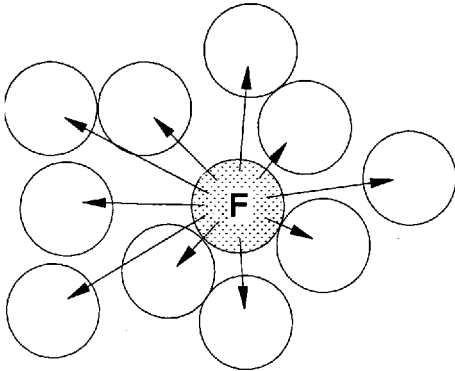


Fig. 3. Dispersal shadows for a plant in question (stippled) and its potential pollen recipients. Seeds produced through female function will land in the stippled seed shadow. Yet seed produced through male function will land in the open shadows and travel farther from the plant in question.

perse seeds to the stippled seed shadow in Fig. 3, whereas males of dioecious populations will only disperse seeds to the outer unstippled shadows. Monomorphic outbreeding cosexuals will disperse roughly 1/2 of their offspring to the stippled circle and 1/2 to the outer circles. Thus, counter to normal assumptions, seed dispersal distances for cosexual plants depend intimately on the nature of pollination and breeding systems.

The population-dynamic consequences of dispersing seeds through male function are different from those for seeds produced via female function in that these 'male' seeds cannot numerically swamp patches in which local seed production is low. This is because the seeds actually fill on the local plants which are experiencing low seed production. The swamping effect of dispersal occurs via paternity, i.e. while few seeds are produced locally, most of them will be fathered by plants from nearby patches undergoing conditions more favorable for reproduction.

The models presented above only considered female function. Technically, they are accurate for plants which have the same dispersal fractions for seeds produced via male and female functions. In practice this is likely to mean agamospermous or selfing species. ESS models could be created that explicitly treat the dispersal of seeds produced through male as well as female function.

The impact of perenniality

The models presented above were framed for simplicity in terms of an annual life cycle. Since most plant species are perennials, we might ask what the likely impacts of perenniality are for the fitness consequences of seed dispersal. We will take a step towards answering this problem by analyzing the simplest case, that of Type II survivorship or constant mortality (cf. Comins 1982). As long as adult survival is less variable than reproductive success, perenniality will buffer variance (cf. Chesson & Huntley 1988). This means that the risk-reducing function of dispersal is likely to be less important for perennial plants than for annuals. The interaction of perenniality and dispersal as adaptations for reducing risk has been analyzed in depth for one form of perenniality, the production of long-lived seed banks (Cohen & Levin 1985; Venable & Brown 1988).

For the models of general density effects and sib-interactions it can be shown that perenniality does not necessarily affect the ESS conditions nor the conditions for invasion. For the general density effects model, let q stand for the mortality of adults, so that $1 - q$ is adult survival. Note that the proportion of safe sites available for colonization or establishment will equal q . The equation (15) becomes

$$\begin{aligned} G(D, \hat{D}) &= \sum_{i=1}^m p_i \left((1 - q) + q \frac{S_i(1 - D) + aD\bar{S}}{S_i(1 - \hat{D}) + a\hat{D}\bar{S}} \right) \\ &= 1 - q + q \sum_{i=1}^m p_i \left(\frac{S_i(1 - D) + aD\bar{S}}{S_i(1 - \hat{D}) + a\hat{D}\bar{S}} \right) \end{aligned} \quad (27)$$

and (16) becomes

$$\frac{\partial G}{\partial D} = q \sum_{i=1}^m p_i \left(\frac{-S_i + a\bar{S}}{S_i(1 - \hat{D}) + a\hat{D}\bar{S}} \right). \quad (28)$$

Thus the slope of the adaptive landscape with respect to dispersal will be shallower, the greater adult survivorship is. Yet the conditions for invasion and the ESS conditions will not change since q will cancel out of these conditions (i.e. the

peaks and valleys of the adaptive landscape will be in the same place but the topography will be flatter).

The intuition behind the lower slopes is this: greater adult survival increases the importance of survival adaptations (competitive ability, etc.) and reduces the importance of reproduction and dispersal. ESS conditions do not change because sites occupied by adults will be unavailable for establishment, but within the fewer available safe sites for establishment, the same dynamics will be going on (e.g. the same proportions of mutant and resident seeds) and the need for dispersal will be the same.

Perenniality can be introduced into the sib-interaction model as follows:

$$G(D, \hat{D}) = 1 - q + \frac{q N(1 - D)}{(N - 1)(1 - \hat{D}) + Na\hat{D} + (1 - D)} + \frac{q aD}{1 - \hat{D} + a\hat{D}}. \quad (29)$$

If N is large, this model can be conceived of as indicating that in each patch, a proportion $1 - q$ of the adults survive to the next year. If N is small (e.g. if $N = 1$), the same model could indicate that the adults survive in a proportion $1 - q$ of the patches and die in a proportion q of the patches.

In this model, all terms containing D , the dispersibility of a mutant individual, are multiplied by q . Thus, once again, all terms in the expression for $\partial G/\partial D$ will contain q thus the slope of mutant fitness with respect to a change in mutant dispersibility will be shallower for a perennial than for an annual by an amount given by q . Once again, q cancels out of the ESS and invasion conditions.

If adults survive from one year to the next there will be fewer sites in a patch that are available for establishment. Since sib-density effects are more important when the number of sites per patch is small, it might at first seem counter-intuitive that perenniality does not favor increased dispersal in a sib-interaction model. Yet, in sib models, a low number of safe sites has its impact on the proportion that seeds of the mutant individual constitute of all seeds competing for safe sites in the

patch. This proportion will be determined by the total number of sites in which seeds are produced in a patch and not by the number of safe sites available for establishment.

We can conclude this analyse by stating that perenniality, by reducing variance, will tend to reduce the importance of the risk-reducing function of dispersal. Yet perenniality *per se* does not necessarily change the ESS dispersibility for escaping crowding or sib interactions. Perenniality does reduce the fitness impact of a change in seed dispersibility, simply because seed reproduction becomes a less important vehicle for persistence from one year to the next.

Several caveats should be mentioned. This analysis of the effects of perenniality was done on an 'all-else-being-equal' basis. Other complications can occur. For example, it might be reasonable in some cases to assume that perennials are larger than annuals, but that patch size is the same. This would result in fewer safe sites per patch (lower N) and thus stronger sib-density effects favoring greater dispersal.

Other complications are likely to arise with Type I or Type II survivorship curves which must be analyzed with age- or stage-specific matrix models. For example, the ESS dispersibility with a Type I survivorship curve would probably be to disperse most seeds produced at early ages but to manufacture a declining dispersal fraction as the probability of dying increases. Similarly, the ESS might be to increase dispersibility with age for a Type III curve. It would be interesting to examine empirical examples suggesting age-specific shifts in dispersal patterns in light of these ideas. There clearly are age-specific changes in dispersibility in some species due to changes in plant height (e.g. wind, adhesive, and ballast systems) and fruit crop size (e.g. frugivore systems). Yet the most obvious such changes are more likely to be proximate correlates of growth rather than adaptive shifts in dispersibility as a function of changing probabilities of mortality. Nevertheless, the possibility of adaptive shifts should not be discarded out of hand.

Another potential complication is that, by creating habitat texture (i.e. vacant and occupied

sites), perenniality creates a substrate for directed or contingent dispersal. If mechanisms are available to direct dispersing seeds to unoccupied microsites, then greater dispersibility could be favored for perennials. But this would be due to greater predictive dispersal in the perennial system not to perenniality *per se*. The above analysis assumes that dispersal is random with respect to site favorability.

Two kinds of dispersal cost

Most models of dispersal in the theoretical literature represent the cost of dispersal in a form that is not fully applicable to the seed and fruit dispersal problem. As usually modelled, only dispersing individuals pay the cost of dispersal (but see Cohen & Motro 1989). This is a reasonable representation of the mortality cost of dispersal (e.g. the production of seeds destroyed during passage through a vertebrate gut, or eaten by a scatterhoarder). But, the allocation costs of dispersal, the proportion of fruit pulp, adhesive barbs, wind-borne parachutes, etc. are typically paid by dispersing and non-dispersing fruits alike (an exception can be made for those dispersal polymorphisms in which only dispersing morphs have dispersal structures, e.g. Venable & Levin 1985 and Venable *et al.* 1987). It is easier to conceive of seed and fruit dispersal problems if both kinds of costs are included.

The two types of costs operate differently and have different effects on the evolution of dispersal. The mortality cost of dispersal is adequately represented by a which is a proportional reduction in the number of successfully dispersing seeds. Allocation costs ultimately involve fitness tradeoffs, so we will consider that, for a given amount of resources, a plant can either produce a larger seed crop with lower allocation to dispersal structure such as high quality pulp, or a smaller seed crop with higher allocation to auxiliary dispersal structures (cf. Schupp, this volume). If seed set is S_i in the absence of dispersal allocation, then with dispersal allocation, seed set will be $S_i \cdot A_s / (A_s + A_d)$ where A_s stands for seed allocation, A_d

stands for dispersal allocation and $A_s / (A_s + A_d)$ is the cost of dispersal calculated as the proportion of the reproductive allocation still going to seeds (i.e. the proportion of seed set retained with a given dispersal allocation). Assume that dispersal increases with allocation to dispersal according to some function $D = h(A_d)$. The inverse function, $A_d = g(D)$, gives the allocation required for a given level of dispersal. Using this formulation, the allocation cost of dispersal can be expressed as a function of the dispersibility, $f(D) = A_s / (A_s + g(D))$. By choosing the appropriate units, the allocation cost of dispersal can be reexpressed for mathematical convenience as $f(D) = 1 / (1 + g(D))$.

The allocation cost of dispersal is paid by dispersing and non-dispersing fruits alike. Thus the escape-from-crowding model can be reformulated to include both kinds of costs as follows:

$$G(D, \hat{D}) = \sum_{i=1}^m p_i \left(\frac{S_i f(D) (1 - D) + a D \bar{S} f(D)}{S_i f(\hat{D}) (1 - \hat{D}) + a \hat{D} \bar{S} f(\hat{D})} \right). \quad (30)$$

For comparison to the previous version of the model which only considers the mortality cost of dispersal we will recalculate the conditions under which a dispersing morph can invade a non-dispersing resident population:

$$\begin{aligned} \frac{\partial G}{\partial D} &= (-f(D) + f'(D)(1 - D)) \\ &\times \sum_{i=1}^m \frac{p_i S_i}{S_i f(\hat{D}) (1 - \hat{D}) + a \hat{D} \bar{S} f(\hat{D})} \\ &+ a \bar{S} (f(D) + D f'(D)) \\ &\times \sum_{i=1}^m \frac{p_i}{S_i f(\hat{D}) (1 - \hat{D}) + a \hat{D} \bar{S} f(\hat{D})}. \end{aligned} \quad (31)$$

where $f'(D)$ stands for $\partial f(D) / \partial D$. Thus,

$$\left. \frac{\partial G}{\partial D} \right|_{D=\hat{D}=0} > 0 \Rightarrow \frac{a \bar{S}}{H(S)} > 1 - f'(D). \quad (32)$$

where $H(S)$ stands for the harmonic mean of S_i . Thus the invasion conditions are the same as those for the previous case with no mortality costs but with an additional term representing the allocation costs of dispersal to the right of the 1. Since $f(D)$ is a decreasing function of D , $f'(D)$ is negative and the righthand side of (32) is greater than 1. Thus, the conditions for invasion of a dispersing morph are more restrictive than when only mortality costs are included.

It can also be shown that $D^* = 1$ will never be the ESS:

$$\begin{aligned} \left. \frac{\partial G}{\partial D} \right|_{D=\bar{D}=1} \geq 0 &\Rightarrow - \sum_{i=1}^m \frac{p_i S_i f}{a \bar{S} f} \\ &+ \sum_{i=1}^m \frac{p_i a \bar{S} f'}{a \bar{S} f} + \sum_{i=1}^m \frac{p_i a \bar{S} f}{a \bar{S} f} \geq 0 \\ &\Rightarrow \frac{1}{a} - \frac{f'}{f} \leq 1. \end{aligned} \quad (33)$$

Since $a \leq 1$ and $f' < 0$, this expression can never be satisfied and $D^* = 1$ will never be the ESS. Cohen & Motro (1989) reached similar conclusions in an analysis of a sib-competition model with no spatial or temporal variance.

The nature of density dependence

We have frequently used some fairly awkward and vague expressions such as 'sib-density effects' or 'escaping the effects of density' instead of simpler expression such as 'sib competition'. This is because the models actually treat general density effects which could just as readily be due to density-dependent predation as to competition.

For analytical simplicity the previous models assumed constant yield or exact density compensation. This corresponds quite well to the empirically observed phenomenon of constant-yield competition in plants (Kira *et al.* 1953). Yet there are many ways in which density effects could operate, and the details will affect the quantitative predictions of the models. For the escape-from-

crowding model, the value of dispersal is in arriving at patches in which residents did poorly in the previous year, but which will experience favorable conditions in the present year. If one or a few plants cannot realize all of the potential yield of a patch, selection favoring dispersal will be weaker than in the simple analytical constant-yield models. Many models of constant-yield competition include the possibility that yield is not fully realized at the lowest densities (reviewed in Watkinson 1980). The degree to which this low-density effect is important will depend on the relationship between plant size and patch size (i.e. single individuals of species with small maximum plant size will not be able to fully realize the potential yield of a large patch).

Another possibility is that density effects may occur with undercompensation such that total yield is higher at higher densities. With undercompensation, there will be less reason to escape crowded patches and selection favoring dispersal will be weaker than in the constant-yield model. If density effects are overcompensating such that total yield is lower at higher densities, the selection for dispersal to escape crowding or sib interactions will be stronger. For example, overcompensating density-dependent predation is the mechanism in the 'escape-from-density' version of Janzen's escape hypothesis. These or other specific density relationships could be inserted into computer-simulation versions of the models presented above.

In order to evaluate the importance of sib-density effects in natural systems, the life stage at which density-dependent limitation is occurring must be known. The reasoning behind this statement is as follows. The mechanism favoring dispersal in this model is that there are diminishing returns from retaining more and more non-dispersing seeds in the parental patch. The diminishing returns are due to the fact the mutant's seeds represent a large fraction of seeds in the parental patch. N is the number of individuals per patch at the limiting life-stage and escape from sib-density effects is only an important function of dispersal when N is small. Since seedling densities may be orders of magnitude higher than

adult densities, N will be much larger if safe sites for seedling establishment are limiting rather than canopy sites for adults. Thus, the life stage at which density-dependent limitation occurs is critical for determining the influence of sib interactions.

The consequences of the breeding system and genetic structure

While the ideas presented here have been predominantly ecological, there are some significant junctures with population genetics. First of all, as mentioned above, on average 1/2 of the offspring of a cosexual plant are produced via male function. These seeds are produced on the mates of the parent in question and consequently the dispersal distance for 1/2 of the offspring depend intimately on the breeding system. This particular juncture between the breeding system and the dispersal biology of plants is critical to an understanding of the population-dynamic consequences of dispersal and it has not been explicitly recognized in much of the seed dispersal literature.

Secondly, the function of dispersal for escape from high concentrations of relatives depends on the genetic structure of the population. Several studies have demonstrated how the strength of the sib-interaction effect depends on the genetic relationship between the individual with the mutant dispersal type and the seeds being left behind (Frank 1986; Taylor 1988). Frank (1986) used Price's (1972) method for analyzing selection in subdivided populations. He showed that $D^* = (\rho - (1 - a)) / (\rho - (1 - a)^2)$ where ρ is Hamilton's (1972) regression coefficient of relatedness of the genotype controlling dispersal (the mother plant in our case) onto a member of the cohort of offspring chosen randomly before dispersal. ρ is a measure of the relatedness of individuals within a patch. It declines with increasing number of individuals per patch and increases with the level of inbreeding. Thus variation in the breeding system will affect the ESS value of dispersal in a sib-interaction model. Under our assumption that

the proportion of dispersing offspring is determined by the mother and with the added assumption that there is no genetic correlation among the mothers in a site or the pollen they have received, Frank's result is equal to ours (26), i.e. $\rho = 1/N$. Since spatial genetic structure is expected to accumulate in metapopulations with fractional dispersal, genetic correlations are likely to exist among plants in a site. This favors greater dispersibility and (26) becomes a lower bound on the ESS dispersal rate. Taylor (1988) formulates the problem in inclusive fitness terms, emphasizing the 'altruistic' nature of dispersal in a sib-interaction model. Dispersing seeds have the same expected reproductive success as non-dispersing ones, yet they must pay the mortality cost of dispersal. In exchange, they improve the prospects of success for those seeds left behind, some of which are their relatives. This phrasing of the problem permits the examination of parent/offspring conflict over offspring dispersal (the mother wants more), with all of the potential intricacies created by the different genetic constitutions of the different seed and fruit tissues (cf. Queller 1983).

Colonization, escape from parents, and directed dispersal revisited

In this paper we have discussed four fundamental population-dynamic functions of dispersal which differ from the three functions of dispersal which are cited in the majority of papers on seed dispersal: colonization, escape, and directed dispersal (Howe & Smallwood 1982). The four population-dynamic functions presented here are based on theoretical results. They are conceptually distinct and have some fundamental population-dynamic reality. For example, risk reduction requires global temporal variance, escape from crowding requires spatial and local temporal variance, while escape from sib-density effects requires only spatial structure and varying concentration of relatives. Colonization, escape, and directed dispersal refer to a mix of ideas that have had important impact on empirical research in

dispersal ecology (the ‘case making’ approach of Howe, this volume). Thus the escape hypothesis is built around Janzen (1970) and Connell’s (1971) hypothesis regarding seed predation in relation to distance from tropical trees. The colonization hypothesis has its roots in early ideas on plant life histories in successional environments (Salisbury 1942; Baker & Stebbins 1965). The directed dispersal hypothesis emerged directly from studies of the natural history of scatterhoarders, mistletoes, and ant-dispersed plants (Vander Wall & Balda 1977; Docters van Leeuwen 1954; Handel 1978).

The colonization hypothesis relates most directly to the escape-from-crowding models. Only limited success is possible in densely occupied patches and occupied patches will eventually disappear. Windfall reproductive profits can be obtained from successful dispersal to empty or sparsely occupied patches.

The escape hypothesis is often phrased in terms of either *distance*-dependent success or *density*-dependent success. Distance dependence refers to negative effects of a long-lived parent (usually a tree) either in terms of competitive suppression or attraction of species-specific predators. A strict distance-from-parent effect is perhaps best considered in terms of the escape from crowding model with perennality, i.e. occupied safe sites or canopy sites are unavailable for regeneration until the occupant dies. Curiously, perennality does not necessarily favor increased dispersibility in the models presented here. This is because, in our models, all habitable patches are potentially occupied by adults and the probability of adult mortality creating an open site is the same everywhere.

Density-dependent versions of the escape hypothesis are often phrased in terms of what we would call an escape-from-crowding or sib-interaction model with over-compensating density dependence. At the highest densities, mortality is so intense that absolute regeneration is lower than at intermediate densities.

Directed dispersal represents a distinct mechanism (i.e. ‘predictive dispersal’) from the other three which all can operate with random dispersal.

If a plant can vary its dispersal into a patch or habitat depending on the likelihood of its success in that patch (perhaps by utilizing an appropriate vector or morphology) then it is clearly fitness enhancing to do so.

Interactions between theoretical and empirical studies

Gaining an empirical understanding of the fitness consequences of fruit dispersal is a daunting task. Where do seeds go? What are their fates throughout the life of the plants? How do different density/frequency/distance dependent factors affect the fitness of dispersing seeds? What are the costs involved in dispersal, both in terms of allocation to dispersal structures and in terms of dispersal related mortality? How does all of this vary in space and time? Understanding all of these factors and processes for any single species seems all but hopeless.

One approach to understanding these difficult to measure phenomena is through conceptual exploration of the phenomena under consideration, and that is the approach taken in this chapter so far. But ultimately, the ideas must be ‘unpacked’ and interfaced with empirical results for specific systems. Some interesting work has been done in this area with modelling frameworks other than those advocated here. Murray (1988) has investigated three neotropical gap-dependent plants using data on dormancy, dispersal, and forest dynamics in a simulation model aimed at determining the fitness consequences of bird dispersal. Alvarez-Buylla & García-Barrios (1991) has integrated field data on patch structure and seed behavior into models of the metapopulation dynamics of the pioneer rainforest tree, *Cecropia obtusifolia*. Olivieri & Gouyon (1985) used a metapopulation approach to model the evolution of dispersibility of the temperate weeds, *Carduus pycnocephalus* and *C. tenuiflorus*. They successfully predicted trends with population age in the ratios of dispersing and non-dispersing seed morphs. Horvitz & Schemske (1986) utilize a metapopulation model to predict the fitness conse-

quences of dispersal and dormancy for *Calathea ovandensis* a tropical herb.

The models presented here can provide a powerful guide to empiricists trying to determine the fitness consequences of dispersal. The models are simple to use and calculations with field data are easily carried out. The data needed to fit to the models is among the easiest to collect and the models point the way to answering difficult questions about seed dispersal.

In the section on escape from crowding we showed that the expected rate of increase in site occupancy of an individual with a mutant dispersal type is given simply by the sum over all patch types of the ratio for each patch type of the reproductive success of mutant and resident seeds (15). The reproductive success in each patch type of mutant and resident seeds can be estimated from field data on seed rain in different patches or quadrats and inserted in the following equation:

$$G(D, \hat{D}) = \sum_{\substack{\text{quadrats} \\ \text{or patches}}} \left(\frac{\text{mutant seed density}}{\text{resident seed density}} \right). \quad (34)$$

As before, $G(D, \hat{D})$ gives the expected fitness of an individual adopting the dispersibility represented by the numerator in a population with the dispersibility represented by the denominator under the assumptions of the escape-from-crowding model.

Various combinations of actual and hypothetical seed distributions can be inserted in the numerator and denominator of (34). For example, the spatial distribution of seeds that would occur if all seeds fell under the parent plant could be used, or the distribution assuming uniform dispersal. One could then ask what the fitness advantage would be of a mutant individual with uniform dispersal in a resident population with the actual dispersibility. Alternatively, one could ask what the fitness advantage of a mutant with the actual dispersibility might be in a population without dispersal. Many other questions could be generated in a similar fashion. For example, what would be the fitness consequence of a change in

the slope of the dispersal curve (c.f. Mary Willson chapter).

The actual data required include: 1) the spatial distribution of reproductive plants (and preferably an estimate of their fecundity), 2) the spatial distribution of seed fall (without necessarily knowing where each seed comes from) or the spatial distribution of seeds on the soil shortly after dispersal, and 3) an estimate of the dispersal curve. Actually, the calculations could be made with either 2) or 3) but if both are available, some cross-validation of the data are possible (e.g. it should be possible to generate 2) from 1) and 3) since applying the dispersal curves to the adult distributions should yield something close to the actual spatial distribution of seeds).

One practical consideration that comes up in calculating (34) is what to do with 0's in the denominator. This represents the situation in which a mutant dispersing seeds would have complete access to the resources of a patch in which there are no residents. The field biologist would have to determine, for the particular patch size assumed, what, N , the maximum site occupancy in that patch type would be. N represents an upper limit on the gain in site occupancy in a particular patch. The calculation would be to sum over patch types either N or the ratio of mutant divided by resident seed density, whichever is smaller.

Elements of sophistication can be progressively added to such a model. The fitness gain accrued by changing morphology could be calculated if measurements were available on the impact of dispersal morphology on seed spatial distributions or on the shape of the dispersal curve (c.f. Augspurger, this volume). Allocation costs of changing morphology could be estimated (Augspurger, this volume) as could mortality costs. At each step of model building one can test the impact of modifying assumptions about unknown aspects of the plant biology (such as the detailed nature of density dependence) or one could set out to measure the unknown aspect.

Exercises of this nature provide first-order empirical estimates of the fitness consequences of dispersal and also focus attention on the biological issues in need of further study to increase

realism and sophistication of understanding. Another useful aspect of this approach is the focus it places on studying all aspects of the dispersal problem in the same experimental system. Currently bits and pieces of information are available for a variety of plant species, but for any single species, large gaps remain.

In this section we have emphasized the model for escape from crowding. The other functions could be explored as well. To investigate the role of risk reduction, measurements for a large number of patches would have to be made in a variety of year types. Sib-interaction models require a knowledge of the partition of seeds in a patch into those produced *in situ* and those coming from elsewhere (c.f. (23)). Evidence on the genetic structure of populations could be incorporated as well. The role of directed dispersal can be estimated by quantifying the non-randomness of deposition patterns and the increased demographic success in the target patch types relative to random patches.

Conclusion

In summary, the field is in an interesting position where a fairly sophisticated set of theoretical ideas exists that can be applied to the question of the fitness consequences of seed and fruit dispersal in spatially and temporally varying plant metapopulations. If this theory is carefully 'unpacked' and applied to frugivory problems it can provide a framework for thinking about the processes involved in the evolution of seed dispersal. We have tried to flesh out some of the implications for real dispersal systems, yet many interesting issues remain untouched. For example, different dispersal syndromes have systematic differences in allocation and mortality costs and in the evenness and directedness of dispersal. Thus dispersal vectors should differ in their relative contributions to risk reduction, escape from crowding, escape from sib interactions, and directed dispersal. Some dispersal mechanisms involve the concentrated clumping of seeds and the details determine the degree of risk reduction, escape from crowding,

and escape from sib interactions achieved. We will have to leave these and many other implications of these ideas to the reader's imagination.

The models presented here should also be very useful for guiding and simplifying empirical research efforts aimed at estimating the fitness consequences of fruit and seed dispersal. Considerable insights can be achieved with relatively easy-to-collect data and the models point the way to add progressive layers of realism by probing more deeply in to an array of critical biological processes.

Acknowledgements

The following people contributed with either helpful discussions or comments on drafts: W. A. Mitchell, G. A. Fox, M. Martinez-Ramos, J. X. Becerra. The research was supported in part by NSF grants BSR-8516971 and BSR-9107324.

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