Ann. Zool. Fennici 25:63–74. 1988

Large-scale spatial structure and population dynamics in arthropod predator-prey systems

Andrew D. Taylor

Taylor, A. D. 1988: Large-scale spatial structure and population dynamics in arthropod predator-prey systems. — Ann. Zool. Fennici 25:63–74.

It has been suggested that dispersal among local populations ("cells") may allow many predator-prey interactions to persist despite unstable local dynamics; this paper reviews the theoretical, laboratory, and field evidence for this hypothesis.

The possibility of regional persistence through dispersal has been shown repeatedly in two sorts of models: those describing within-cell densities explicitly and those ("cell-occupancy" models) which classify cells into a few density categories. Persistence in these models, when within-cell dynamics are unstable, requires: (1) a source of asynchrony among cells, which may be fixed differences among cells or some stochastic factor affecting them independently; (2) some within-population density dependence; (3) a large enough number of cells; (4) dispersal rates which are not high enough to synchronize the system; and (5) in cell-occupancy models, sufficient dispersal to offset the rate of extinctions. Important unanswered questions concern spatial heterogeneity, density-dependent dispersal, and the interaction of dispersal with within-cell density dependence.

Empirically, several laboratory predator-prey studies have shown a correlation between persistence and environmental complexity, and others have also suggested this. At least one natural plant-herbivore system (*Opuntia* and *Cactoblastis* in Australia) apparently persisted, despite local extinction, by dispersal. There is, however, little evidence concerning the role of dispersal in any natural predator-prey interactions; this can probably best be obtained through direct manipulation of dispersal itself.

Andrew D. Taylor, Department of Zoology, The Ohio State University, 1735 Neil Avenue, Columbus, Ohio 43210-1293, U.S.A.

1. Introduction

For many years some ecologists have argued that migration among local populations is important in the dynamics of regional populations, and especially in permitting their persistence in the face of unstable local fluctuations and extinctions (Andrewartha & Birch 1954, 1984, den Boer 1968, 1981). A particularly popular application of this regional dispersal-persistence hypothesis has been to predator-prey and parasitoid-host interactions (Nicholson 1933, Nicholson & Bailey 1935, den Boer 1968, Murdoch & Oaten 1975, Murdoch 1979, Murdoch et al. 1985, Morrison & Barbosa 1987).

This predator-prey version of the hypothesis is supported by a variety of indirect arguments. First, on the evidence of laboratory systems (Gause 1934, Huffaker 1958, Luckinbill 1973) and models (Nicholson 1933, Nicholson & Bailey 1935), it is generally accepted that simple predator-prey systems are particularly prone to unstable oscillations and extinction. Some authors argue further that the features thought to be required for local stability are not present in many natural interactions (Dempster 1983, Murdoch et al. 1984) or if present may not prevent extinction (Morrison & Barbosa 1987), or even that local extinctions in fact do occur (Murdoch et al. 1985); it is therefore concluded that some external factor, presumably migration among populations, must be responsible for the persistence of these systems. The feasibility, if not the reality, of regional persistence through dispersal is also supported more

directly by both an extensive body of theory and a number of suggestive laboratory studies, which will be reviewed in the following. Finally, an analogous regional dispersal process appears often to be involved in the coexistence of competing species (e.g. Horn & MacArthur 1972, Slatkin 1974, Hanski 1983); this indeed is implicit in the concept of "fugitive" early-successional plant species.

Unfortunately, however, there is as yet no direct evidence from natural predator-prey interactions concerning the role of population subdivision and dispersal in allowing persistence. In hopes of aiding and abetting the needed empirical studies, in this paper I will summarize what we do know, theoretically and empirically, about the regional dispersal-persistence hypothesis, and then briefly discuss how we might learn more.

2. Concepts and questions

It is first necessary to define the hypothesis more precisely. A crucial distinction is that between local and regional spatial units, which in multispecies interactions commonly are called "cells" and "systems". The present paper is concerned with the effects of the organization of local predator-prey cells into regional systems, and of dispersal within such systems; the effects of spatial heterogeneity and movement within local cells are discussed by Hassell (this issue). Local populations or cells are defined as the units within which occur reproduction, population regulation, and interactions such as predation, and within which the movements of most individuals are confined; there may well be further spatial structure within these. Regional populations or systems then are collections of local populations or cells linked by dispersal. In some models, where dispersal is only among neighboring cells, there may develop synchronized groups of neighboring cells; these are termed "hypercells" (Crowley 1981).

The importance of the distinction between local and regional phenomena is that the causes, dynamics, and effects of movement on these two scales, particularly with regard to predator-prey interactions, are quite different. At the local level the phenomena of interest typically concern the predatory interaction itself: the relevant movements are those affecting small-scale prey distribution and predator foraging over that distribution, and these directly affect the stability (see below) of local populations. Dispersal between local populations, on the other hand, can be thought of entirely independently of any interactions, and its primary dynamical effect is not local stabilization but regional persistence (see below). This dynamical difference is not lessened by the fact that the two scales may overlap, as discussed in Section 3.5 below, though this will complicate empirical definition of local and regional populations.

A secondary distinction is that between spatial subdivision and spatial heterogeneity. The preceding discussion of spatial population structure (cells and systems) did not specify whether cells differ from one another. Virtually all of the relevant theory, in fact, assumes that they do not: it considers subdivision, but not heterogeneity. As will be discussed in Sections 3.2 and 3.5 below, this assumption probably is conservative at the most general level, in that heterogeneity would further enhance persistence; more specific conclusions, concerning e.g. the effects of rates or density dependences of dispersal, however, may be strongly affected by whether cells differ.

Two types of dynamical measures also must be distinguished: stability and persistence. In this paper stability will be defined in the narrow sense, strictly applicable only to deterministic models, of populations returning to their equilibrium densities following small perturbations. Unless otherwise specified this will refer only to local populations; in some cases this within-cell stability will depend on whether the cell is isolated or is part of a system, but as is discussed later, this distinction generally is not important. At times the stability of the entire system — i.e. whether all its constituent populations are stable — will also be considered. In a system which is not regionally stable the proportions of cells in various states (e.g. occupied by prey or by predators) may nonetheless tend toward stable equilibrium values, even while the states of individual cells constantly change; to avoid confusion, however, I will not refer to this as stability, but rather as "macroscopic constancy". Persistence means simply that populations do not go extinct; this too can apply either locally or regionally, but unless otherwise stated will mean regional persistence.

The hypothesis of the Introduction can now be put more precisely as: Dispersal among identical local populations enhances persistence of regional predator-prey systems in which isolated local cells would have unstable dynamics (perhaps even extinctions). Questions of interest, then, are: Can this occur? If so, how and when? How are the dynamics of individual cells affected, and in particular can they be made stable? How does this process interact with

local factors which lessen instability? What additional effects would result from heterogeneity among locations?

3. Theory

3.1. Types of models

Models of predator-prey interactions with regional and local population structure have been of two basic sorts: "cell occupancy" models, concerned with how many cells are in each of a few discrete categories of prey and predator densities at a given time, and "explicit within-cell" models which fully model within-cell densities and dynamics.

The cell occupancy models (Vandermeer 1973, Maynard Smith 1974, Hastings 1977, Zeigler 1977, Gurney & Nisbet 1978, 1982, Takafuji et al. 1983) share several fundamental assumptions: (1) all cells of a given occupancy state have identical dynamics, (2) only a few specified transitions among states are possible, and (3) some transitions occur only as a result of immigration, while (4) others are endogenous and inevitable, e.g. predators inevitably exterminate prey from any cell in which both occur. It generally also is assumed that predators cannot survive in cells lacking prey. (Vandermeer (1973) does not make this assumption, and as a result obtains aberrant results (Hastings 1977).)

The dynamics of a typical cell occupancy model (Maynard Smith 1974; see also Zeigler 1977, Takafuji et al. 1983) are shown schematically in Fig. 1. Due to the many states involved, and the large number of parameters — e.g. the times taken by each transition and the rates and distances of colonization by each species — models in the form of Fig. 1 have been studied by simulation. By considering only presence/absence (i.e. states *E*, *HC*, and *MD* in Fig. 1), in an infinite number of cells all equally accessible to each other, however, analytically tractable deterministic models can be obtained; an example (Zeigler 1977) is

$$\begin{aligned} dx/dt &= Hx(1-x-y) - Pxy \\ &= Hx(1-x) - (H+P)xy \qquad (1) \\ dy/dt &= Pxy - Cy. \end{aligned}$$

Here x is the proportion of cells containing only prey and y the proportion containing both prey and predators; $(1 - x - y)$ thus is the proportion of cells which are empty. H is a dispersal parameter describ-

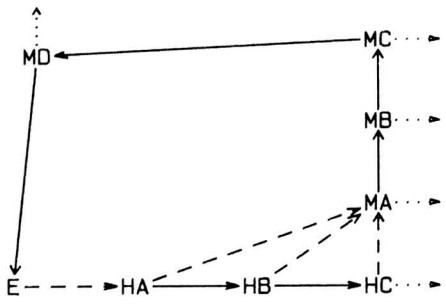

Fig. 1. Cell-occupancy states and transitions in the model of Maynard Smith (1974). Cells in state *E* are empty, cells in states *HA*, *HB* and *HC* contain prey only, in densities from few (*HA*) to many (*HC*), cells in states *MA*, *MB* and *MC* contain predators (in densities from few to many) and many prey, and state *MD* contains many predators but few prey. Transitions shown by dashed lines occur only by immigration, *E* and *HC* cells remaining as such until immigration occurs. Other transitions, shown by solid lines, occur automatically. Emigration is shown by dotted lines: prey from many-prey (*HC* and *M*-) cells and predators from many-predator-few-prey (*MD*) cells. Probabilities of immigration depend on how many cells from which the focal cell is accessible are producing migrants of the appropriate species.

ing the rate at which prey-only cells colonize empty cells, P is a similar parameter describing invasion of prey-only cells by predators from mixed cells, and C is the per-cell rate of extinction of mixed cells. An interesting point is that this model intentionally is formally equivalent to the Lotka-Volterra predator-prey model (Zeigler 1977). An implication not discussed by Zeigler (1977) is that the macroscopic system will be neutrally stable: x and y will show oscillations of a constant amplitude determined by initial conditions. This contrasts with the macroscopic constancy shown by simulations of the more complex model (similar to Fig. 1) from which the model of Eq. 2 was derived (Zeigler 1977).

The cell occupancy models were the first used to study the effects of regional dispersal. As such, they played an historically important role by confirming the possibility of the basic regional dispersal-persistence hypothesis. But their extremely simple structure prevents these models from realistically describing most natural interactions, from being used to investigate many factors which might enhance or impede the effect of regional dispersal, and from being readily comparable to theory concerning the dynamics of single cells.

The second class of models (Comins & Blatt 1974, Allen 1975, Chewning 1975, Hilborn 1975, Zeigler 1977, Crowley 1981, Nachman 1987a, b, Reeve 1988) largely remedies these shortcomings, by explicitly describing dynamics within as well as among cells. A representative model (Reeve 1988) assumes that there are n cells, with cell i containing $N_i(t)$ prey and $P_i(t)$ predators in generation t. Proportions μ_N of prey and μ_P of predators then leave each cell, and at time $t+h$ are evenly re-distributed over all cells, giving cell densities

$$\begin{aligned} N_i(t+h) &= (1-\mu_N)\,N_i(t) + (\mu_N/n)\sum_{j=1}^{n} N_j(t) \\ P_i(t+h) &= (1-\mu_P)\,P_i(t) + (\mu_P/n)\sum_{j=1}^{n} N_j(t). \end{aligned} \tag{2}$$

Predation and reproduction then occur in each cell between times $t+h$ and $t+1$, according to either the Nicholson-Bailey (1935) model:

$$\begin{aligned} N_i(t+1) &= f N_i(t+h)\, e^{-aP_i(t+h)} \\ P_i(t+1) &= N_i(t+h)[1 - e^{-aP_i(t+h)}] \end{aligned} \tag{3}$$

or May's (1978) negative-binomial model:

$$\begin{aligned} N_i(t+1) &= f N_i(t+h)[1 + aP_i(t+h)/k]^{-k} \\ P_i(t+1) &= N_i(t+h)\{1 - [1 + aP_i(t+h)/k]^{-k}\}, \end{aligned} \tag{4}$$

where f is the prey per-capita reproductive rate, a the Nicholsonian predator search rate, and k the clumping parameter of the negative-binomial distribution of encounters.

Other explicit within-cell models describe predation and reproduction by other standard predator-prey models (Comins & Blatt 1974, Chewning 1975, Zeigler 1977, Crowley 1981), or variants of these (Allen 1975, Hilborn 1975), or by a detailed system-specific model (Nachman 1987a, b). All of them, however, differ from the cell occupancy models in one important way: dynamics within each cell can continue, cycling through high and low densities, in the absence of immigration. Local extinction can occur in many of the models, but in none is it inevitable or dynamically important; in Reeve's (1988) model the within-cell submodel (Eq. 4) could even be stable.

Another difference between the two classes of models, which will be important below, is in the scaling of their dispersal parameters. In the explicit within-cell models, dispersal parameters (e.g. the μ's in Eq. 2) are proportions of populations per generation (or something similar), as might be measured in a field study. In the cell occupancy models, on the other hand, colonization parameters (e.g. H and P in Eq. 1) are quantities such as the mean proportion (or number) of empty cells colonized by migrants from a given cell during the lifetime of that cell; this is a much longer time scale than a single generation, while the numbers of colonized cells may be much lower than the number of migrants.

As with the cell occupancy models, the principal goal of most studies of explicit within-cell models has been to explore the effects of the regional population structure (e.g. cell number, dispersal patterns and rates) (Hilborn 1975, Zeigler 1977, Crowley 1981, Reeve 1988). In addition, however, several authors have examined the effects of spatial heterogeneity (Chewning 1975) or the effects of various dispersal schemes on the maintenance of spatial heterogeneity (Comins & Blatt 1974, Zeigler 1977), analyzed deterministic stability (Allen 1975, Chewning 1975, Crowley 1981, Reeve 1988), or compared models with varying degrees of within-population stability (Reeve 1988).

Despite these differences in form and analysis, however, all the models of both types consistently conclude that regional persistence can be enhanced by dispersal among cells. How and when this possibility may be realized will be the focus of most of the following discussion.

3.2. Asynchrony and variability

For regional persistence to be enhanced by dispersal, when the dynamics of individual cells are unstable, the fluctuations of the cells must be asynchronous. If instead the fluctuations are synchronous (and identical in all cells, as assumed by almost all the regional-population models), there will be no net flow to or from any population, and hence no buffering of the fluctuations: the collection will behave exactly like a single cell. The effects of the various factors considered below will be seen to depend largely on their effects on population synchrony.

Maintenance of asynchrony in the face of the homogenizing effect of dispersal requires one or both of two things: (1) permanent differences among cells which make synchronization impossible, or (2) random factors which affect each cell differently at any

given time — thus desynchronizing them — but on average do not differ among cells over time. The first of these factors, temporally-constant spatial heterogeneity, has received little attention, most workers concentrating instead on the much simpler case of a homogeneous environment. Preliminary indications, however, are that spatial heterogeneity can indeed enhance persistence. Maynard Smith (1974) modeled a system in which within-cell dynamics remained unstable but different cells fluctuated at different frequencies due to fixed environmental differences, and concluded that dispersal would tend to synchronize the cells at some intermediate frequency and thus preclude any enhancement of persistence; unfortunately, no evidence was presented for this assertion. Chewning (1975), on the other hand, presents general conditions under which a system of heterogeneous, unstable local interactions can be stabilized by migration, and gives a simple example (a system of two internally Lotka-Volterra cells with rate parameters in one cell twice those of the other) in which this occurs. Alternatively, spatially-biased migration — a tendency for prey or predators, or both, to migrate toward some locations — can stabilize a system of identical Lotka-Volterra interactions (Comins & Blatt 1974); Comins & Blatt (1974) speculate that asynchronous cycles may be involved, though a refuge effect is also possible. Hassell (this issue) also presents models in which spatial biases in immigration are stabilizing, though these might be more appropriately interpreted as representing within-cell patchiness. It should be noted that the outcome in the models of Comins & Blatt (1974), Chewning (1975) and Hassell (this issue) is not merely increased persistence but actual stability, both locally and regionally. Since any real system inevitably will contain heterogeneity both in within-cell dynamics and in migration, the effects of this heterogeneity (and its interaction with other factors of system structure discussed below) clearly deserve much more study.

In all the other models of subdivided predator systems, which assume all cells to have the same internal and dispersal dynamics, the one structural factor which crucially affects the outcome is whether the models are deterministic or stochastic. The reason for this is that in this case, unlike those studied by Comins & Blatt (1974), Chewning (1975) and Hassell (this issue), dispersal cannot produce regional stability: deterministic analyses of very general models conclude that a system of identical cells can never be more stable than an isolated cell would be and might even be less stable (Allen 1975, Crowley 1981, Reeve 1988), though loss of stability requires fairly extreme conditions (Reeve 1988). Persistence of homogeneously subdivided, locally unstable models therefore is possible only with some stochastic factor to maintain asynchrony, and is correlated with the amount of this stochastic variability (Table 1; Crowley 1981, Reeve 1988).

Table 1. System persistence in an explicit within-cell model as a function of prey and predator per-capita dispersal rates and environmental variability (from Reeve 1988). Systems contained 100 cells, with dynamics described by Eqs. 2 & 3 with f a lognormal random variable with mean 2 and coefficient of variation (CV) as shown. Results shown are the number of generations (mean ± 1 S.E. for 5 replicates) until extinction (defined as either prey or predator population, for the entire system, dropping below 0.001 × its equilibrium density); a maximum of 500 generations were simulated.

		μ_P		
μ_N	CV (f)	0.01	0.10	1.00
0.01	0	457.4 ±42.6	403.0 ±63.6	
	0.71	500.0 ± 0	500.0 ± 0	
	1.41	500.0 ± 0	500.0 ± 0	
0.10	0	49.6 ± 5.4	22.4 ± 2.9	
	0.71	312.4 ±85.4	131.4 ±23.7	
	1.41	500.0 ± 0	500.0 ± 0	
1.00	0			15.0 ± 0.5
	0.71			24.4 ± 0.8
	1.41			17.4 ± 2.2

The desynchronizing stochastic factors can be of a variety of forms. In cell-occupancy models the dispersal process is probabilistic in space (even though by assuming an infinite number of cells a deterministic regional model may be obtained): at any time a species colonizes only some of the cells available to it, thereby creating differences among the cells. In models with explicit within-cell dynamics, asynchrony and persistence have been produced by stochastic environmental variation in population parameters (Crowley 1981, Reeve 1988), and by demographic stochasticity either within small populations (Nachman 1987a) or in dispersal among cells (Hilborn 1975). In the first case, persistence times are somewhat dependent on which parameter is made variable, but this effect is generally minor compared with that of the amount of variability (Reeve 1988). An exception is large variability in the attack rate, which leads, with complete mixing, to deterministic stability (Bailey et al. 1962).

Whatever the source of variability, maintenance of asynchrony requires that different cells be affected differently. Demographic stochasticity or probabilistic colonization will always do this, but environmental variability may not: environmental factors acting on the scale of an entire system of cells (e.g., regional weather patterns) will affect individual cells synchronously (Crowley 1981), while at the other extreme factors acting on a scale much smaller than an individual population or a hypercell will be largely averaged out at the population level (Crowley 1981).

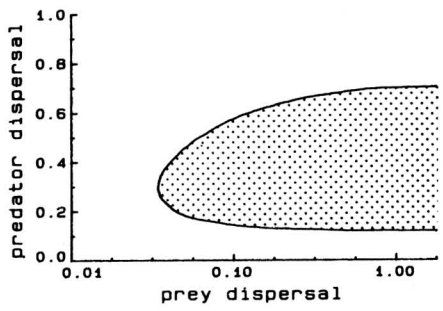

Fig. 2. Persistence of a cell-occupancy model as a function of prey and predator dispersal parameters, from Zeigler (1977: Fig. 5). Dispersal parameters are probabilities of colonization of empty neighboring cells, analogous to H and P in Eq. 1 in text; see Zeigler (1977) for model details and other parameter values.

3.3. System structure: cell number, and dispersal rates and distances

A consistent finding is that the persistence-enhancing effect of between-population dispersal is stronger in systems with more cells (Maynard Smith 1974, Hilborn 1975, Zeigler 1977, Crowley 1981, Nachman 1987b, Reeve 1988). This result is sometimes viewed as a statistical effect, of an average taken over an increased number of independent units (Crowley 1981). A perhaps more meaningful interpretation is that increasing system size decreases the likelihood of synchronization (Crowley 1981). This interpretation is clearest for models with limited dispersal distances in which synchronized hypercells develop: if the system is large enough to contain several hypercells it can persist, while if it is the size of a hypercell it cannot (Crowley 1981, Nachman 1987b). In the special case in which local extinction is important, a reduced risk of simultaneous extinction (the population-dynamics meaning of den Boer's (1968) "spreading of risk in space") is essentially a reduced risk of synchrony.

It appears that above a certain level, a further increase in system size may have little effect on persistence (Nachman 1987b, Reeve 1988). Various models differ substantially on this point, however: Nachman (1987b) and Reeve (1988) find little difference above about 25 cells, but Zeigler (1977) reports that stabilization was greater in a system of 900 cells than one with 100 cells.

The dispersal rates of prey and predators clearly also could be important for regional persistence. The two types of models, however, produce apparently quite different conclusions concerning what rates most favor persistence. In cell-occupancy models persistence generally requires both prey and predator dispersal rates to be fairly high, but the predator rate not too high (Fig. 2; Maynard Smith 1974, Zeigler 1977). For Zeigler's (1977) model in Eq. 1, for instance, the conditions for persistence are equivalent to those for positive equilibria in the analogous Lotka-Volterra system: "predation" (invasion of prey cells from mixed cells) must be fast enough for each mixed cell to replace itself, and prey cell "reproduction" (colonization of empty cells) must be fast enough, and predation slow enough, for prey cells to replace themselves before being invaded by predators. Two exceptions to this conclusion should be noted, however. First, Hastings (1977) did not obtain a minimum prey dispersal rate for persistence, possibly because he assumed prey in mixed cells as well as prey-only cells could colonize empty cells. Conversely, Takafuji et al. (1983) found that high prey dispersal could lead to macroscopic instability of cell-occupancy proportions, and thus perhaps extinction in a natural interaction, when there is strong local competition among prey.

In the explicit within-cell models, in contrast, there is not a lower limit on dispersal rates below which persistence is lost, and in fact persistence is greatest at the lowest non-zero dispersal rates examined (Table 1; Crowley 1981, Nachman 1987b, Reeve 1988). These models do, however, show an upper limit for prey as well as predator dispersal rates, above which cells become synchronized and the system does not persist (Crowley 1981, Nachman 1987b, Reeve 1988); this synchronizing effect of dispersal is particularly well treated in Crowley's (1981) analysis of the effect of dispersal on a single cell linked to a stable pool. The effect of dispersal rates on persistence will also, of course, depend on a number of factors, in particular the amount of desyn-

chronizing variability (Table 1; Crowley 1981, Nachman 1987b, Reeve 1988) and the within-cell effectiveness of the predators (Takafuji et al. 1983, Nachman 1987b).

These differences between the cell-occupancy and explicit within-cell models in the relationship between dispersal and persistence (i.e. Fig. 2 *vs.* Table 1) may be in part artifacts of the different measures of dispersal in the two types of models (see Section 3.1), in that low per capita dispersal (measured in an explicit within-cell model) might produce a high rate of colonization of empty cells (measured in a cell-occupancy model). There are also, however, two biologically meaningful explanations for this disagreement. First, the asynchrony in cell-occupancy models, involving differences among cells in the presence and absence of species, is grosser than that in the explicit within-cell models. In other words, the probabilistic colonization in the former models is a more effective desynchronizer than the various sources of variability in the latter. Prey dispersal in particular cannot ever synchronize cell-occupancy models: even if all cells are colonized by prey, synchrony would require also that all (or none) have been invaded by predators. The second point is that since local extinction is not inevitable in the discrete within-cell models, a cell's populations often can persist without immigrants and thus with low rates of dispersal.

Much of the preceding discussion has not distinguished between the dispersal rates of prey and predators. It is sometimes believed, perhaps due to Huffaker's (1958) empirical studies, that persistence requires prey dispersal rates to be greater than those of the predator. Although, as noted above, cell-occupancy models cannot persist if predator dispersal is too high, it is in fact not the case in either type of model that prey must disperse more than predators (Maynard Smith 1974, Hastings 1977, Zeigler 1977). In particular, both Nachman (1987b) and Reeve (1988; see Table 1) found that prey dispersal produced synchrony and destroyed persistence at lower levels than did predator dispersal. Vandermeer (1973) does conclude that low predator dispersal is desirable, but this is due to his unusual assumption that predators can survive without prey.

What is fairly clear is that low predator dispersal, relative to prey dispersal, will produce relatively large mean prey densities (Maynard Smith 1974, Zeigler 1977, Reeve 1988). As an aside, decreasing the predator's reproductive capacity, or increasing that of the prey, will also increase mean prey densities. Growth rates may also affect persistence, although the preliminary evidence is contradictory: in a deterministic analysis Crowley (1981) found that a higher ratio of prey to predator reproductive rates reduces the tendency toward synchronization, while in stochastic simulations with a fixed predator reproductive rate Reeve (1988) found persistence to be negatively correlated with the prey growth rate.

Prey and predator dispersal rates also appear to have different effects on the amplitude of local population fluctuations (Reeve 1988): low prey dispersal increases the local prey fluctuations, as unstable local oscillations are less buffered by immigration, while low predator dispersal appears to decrease local prey fluctuations, apparently by allowing a local numerical response and thus more effective regulation of the prey. Low dispersal rates of both species presumably also will increase the spatial variability of densities, though data on this have never been reported.

Closely related to the question of dispersal rates is that of dispersal distance. Two basic migration schemes have been explored: "stepping-stone" migration in which only nearby cells are accessible from any given cell, and "island" migration in which all cells are equally accessible. It seems, intuitively, that stepping stone migration would have a weaker synchronizing effect than island migration. Unfortunately, most studies have considered only one migration scheme, and the two studies which have explicitly compared migration patterns reached opposite conclusions. Thus Nachman's (1987b) simulations suggest that increasing the proportion of long-distance migration decreased system stability and persistence, but Maynard Smith (1974) reports that an island model persisted slightly longer than an otherwise equivalent stepping-stone model. Unfortunately, Nachman's (1987b) simulations were neither replicated nor compared quantitatively, while Maynard Smith (1974) presents no data at all; this issue clearly needs further study.

Intuition also suggests that the density dependence of migration might be important, but is not clear about what its net effect would be. If emigration increases or immigration decreases with increasing cell density, this will tend to stabilize within-cell dynamics but would also strengthen the synchronizing effect of dispersal; conversely, aggregative migration might retard synchronization (Maynard Smith 1974) but would increase local instability. This question again has received little rigorous attention: Nachman's (1987b) model is the only one with density-dependent dispersal, and its analysis of the effect of

this is so incomplete that no conclusions can be drawn. It may also be that there is an interaction between density-dependent dispersal and spatial heterogeneity: the stabilizing influence of density-dependent dispersal might be strengthened, and thus prevail, when cells have substantially different dynamics (see Hassell, this issue).

3.4. Within-population stability

As indicated by the preceding discussion, almost all work on multicell systems has incorporated the assumption that local interactions, if isolated, would be unstable or go extinct. From this it has been found that persistence does not require within-population stability *per se*. This does not imply, however, that locally stabilizing, density-dependent factors are irrelevant in these systems; on the contrary, local density dependence remains necessary for persistence, while inter-population dispersal often is not.

It seems likely that a system of populations linked by dispersal but entirely lacking density-dependent regulation necessarily will eventually either increase to infinity or decrease to zero; in the real world this would mean either extinction or some density regulation (e.g. resource limitation). If there is no delayed or immediate density-dependence in predation, the dynamics of the prey become equivalent to a single-species model, with predation subsumed into the net reproductive rate. The analyses of Klinkhamer et al. (1983, Metz et al. 1983) suggest that the long-term growth of such a system will be intermediate between that of a single population (determined by the mean log growth rate; Lewontin & Cohen 1969) and that of an infinite system of identical cells with complete mixing every generation (determined by the mean growth rate, the log of which will be greater than the mean of the log growth rates). It will therefore ultimately either explode or go extinct unless the effect of the mixing (and perhaps of mortality during dispersal) is such that it produces a mean growth rate of exactly zero, an unlikely circumstance of no natural relevance.

Extinctions need not occur in the multicell predator-prey models, and explosions never do, because they all contain, implicitly or explicitly, some sources of density-dependent population regulation. Specifically, all coupled predator-prey models necessarily contain density dependence in both populations: in prey mortality through the predator's numerical response (although this may be delayed, so that the function describing within-generation predation is density independent, as in Eqs. 3 & 4), and in predator reproduction through competition for prey. These factors prevent unregulated population explosions; in discrete-time models delays in these density dependences may destabilize within-cell equilibria but they still restrain populations, and in some models (e.g. Reeve 1988; see Eqs. 3 & 4) predator or parasitoid competition is immediate and thus stabilizing (Taylor 1988). The models of Hilborn (1975), Takafuji et al. (1983), and Nachman (1987a, b) also include direct prey density dependence, in the latter two through depletion of the food supply.

Two fundamental assumptions of the cell-occupancy models — that all populations of a certain category produce the same number of migrants, and that migration into occupied cells has no effect — produce additional density dependence, as shown by the prey self-damping term in Eq. 1. The first of these assumptions implies that all populations in a given category are at the same density (i.e. some stable carrying capacity; Hastings 1977) or else that the probability of an individual leaving a cell decreases linearly with cell density. The second assumption in essence constitutes competition for space.

Not only is some density regulation necessary for regional persistence, but stronger within-population regulation enhances persistence and constancy. Thus persistence is usually somewhat greater and fluctuations smaller if within-cell dynamics are only moderately unstable rather than severely so (e.g. described by Eq. 4 with $k = 2$ rather than by Eq. 3; Reeve 1988). If local interactions are in fact stable (e.g. $k = 0.5$ in Eq. 4), of course, neither dispersal or asynchrony may be needed for persistence, even with considerable random environmental variability (Reeve 1988; cf. Morrison & Barbosa 1987).

It should be noted that even when individual behavior is density independent the net flow in or out of individual cells will be density dependent (per capita emigration will be constant, but per capita immigration will be inversely related to density). In the field, if only cell densities or net flows can be observed, this phenomenon would be indistinguishable from classical density-dependent regulation of the local populations. In dispersal-linked systems, however, it moves cells toward the system mean rather than toward an equilibrium, and thus increases synchronization, though obviously not always sufficiently to prevent persistence.

3.5. Unanswered questions

As summarized here, current theory is consistent on the main points: low to moderate dispersal within a system of cells, combined with some factor(s) preventing synchronization of cell fluctuations and some density regulation, can indeed enhance the persistence of the system. Many specific questions, however, remain unanswered. The most important of these, noted above, concern the effects of spatial heterogeneity and of density-dependent dispersal. Also, although it is clear that within-cell stability and between-cell sources of persistence are in general complementary rather than opposed, it would be valuable to know more about their interaction. In particular, the suggestion that population subdivision and dispersal can destabilize a locally stable interaction (Allen 1975, Crowley 1981, Reeve 1988) needs to be investigated in the sort of stochastic models which so far have shown persistence — only if this is done will it be known whether regional dispersal always enhances persistence, as is usually assumed.

Many secondary questions concerning system structure also remain which are relevant in determining in which real systems we should expect regional dispersal to be dynamically important. Among these are: (1) the system sizes (numbers of cells) at which inter-cell processes become significant and the sizes at which they attain their maximal effect; (2) the discrepancy between cell-occupancy and explicit-density models concerning the range of dispersal rates favoring persistence; (3) the effects of the relative dispersal and reproductive rates of prey and predator; and (4) the effects of island vs. stepping stone migration. It would also be useful to know more about how regional dispersal affects within-cell dynamics, since the latter are what typically are observed. Furthermore, the answers to many of these questions undoubtedly are interrelated, and better understanding of these interrelations would be valuable.

A number of aspects of natural systems which are likely to be relevant have not been studied at all. All existing models assume a very simple spatial structure: cells have no explicit internal spatial heterogeneity, are of the same scale for both predators and prey, are identical (with a few exceptions as noted above), and are all part of a single system (i.e. there is no subdivision of the system into distinct subsystems of cells). In nature a hierarchical organization seems likely; for instance, if "cells" are plants and the "system" is a field (or a greenhouse, as in Nachman 1987a, b), there also will almost certainly be some movement among fields on a farm, among farms in a region, etc. Interactions between levels of such a hierarchy might be interesting, although there is no reason to expect they would negate the conclusions of current models. More interestingly, the scales of population structure of the two species might differ. For instance, some herbivores (e.g., many homopterans) are much less mobile than their natural enemies, so that several local populations of prey would fall within the foraging range of individual predators, that is, within a single local predator population (in which case the definition of local and regional populations becomes difficult); the opposite asymmetry is also possible. Hassell (this issue) presents preliminary results for systems with either one predator population and several completely isolated prey populations, or the reverse, and with immigration rates differing among cells. In this case not only persistence but regional stability is possible, and predator aggregation in areas of high prey density might be even more favorable to stability (Hassell, this issue). Further study of these issues, especially in systems containing more than one local population of the more mobile species, and with only partial isolation of populations of the other species, should prove very informative.

Almost all current models also assume unnaturally simple biotic structures with only two species. The tight coupling of prey and predator dynamics is essential to the within-cell dynamics, and so presumably to system behavior. Adding additional species in either trophic level, and thereby weakening this coupling, would almost certainly have a significant effect, although what it would be is unclear; the dynamics of multispecies predator-prey systems are complex and incompletely understood even in homogeneous systems. Vandermeer's (1973) model, in which predators lacking prey die off exponentially rather than immediately, indeed might be resurrectable as a model for a generalist predator which has a constant supply of alternative, though not fully adequate, prey in every cell. Seen in this way, his conclusion that persistence is possible even with very low predator dispersal rates becomes sensible, and it is reassuring that this model otherwise agrees with those which followed it.

Two other models — Takafuji et al. (1983) and Nachman (1987a, b) — expand the biotic structure vertically rather than horizontally, by including the quality of the prey's host plants. In this case the principal effect is not to decouple the interaction but

simply to create overcompensating competition among the prey, leading to prey extinction even without predators if prey emigration is too low (so that local abundances become excessive and then crash). Beyond this condition on prey emigration, the specific effects of this modification are not clearly defined, but it does not seem likely to affect the basic conclusions concerning system persistence summarized in this paper.

More fundamentally, a more powerful and general modeling approach is needed. The combination of detailed internal dynamics, dispersal, and stochasticity has so far had to be studied by simulation. Unfortunately, general conclusions on, e.g., the interactions among structural factors, are difficult to obtain by simulation. If theory is to go much beyond where it is today, and especially if it is to establish a coherent general framework, it will almost certainly have to develop analytic methods in place of the current reliance on simulation.

4. Empirical evidence

4.1. Laboratory

Several laboratory studies have examined the relationship between population subdivision and dispersal and the persistence of predator-prey systems. The first and best known was Huffaker's (1958) classic mite study, in which the two species persisted longest in the most complex environment. However, this study was completely unreplicated, and the system in the complex environment lasted through three oscillations (all others persisted for only one oscillation) only by virtue of single prey females surviving population crashes; it may have been pure chance that this occurred in the most complex system. Further studies (Huffaker et al. 1963) did show persistence for two or more oscillations in both replicates of a complex system. However, when the size of the cells in this system was increased but their number and spatial arrangement kept the same all three replicates went extinct after only one oscillation, calling into question the robustness of the preceding result. In combination these two studies do suggest greater persistence in more complex systems, but the lack of replication raises some doubts about this, and the complexity of the differences between trials — especially the alteration of within-cell dynamics as well as system structure — makes mechanistic interpretation difficult.

Takafuji (1977), working with another mite system, also reported greater persistence in a patchy system when dispersal was made more difficult. However, neither of his systems persisted beyond one oscillation; the increased persistence in the more subdivided system was due simply to slower predator dispersal from the single location at which they were introduced. In further experiments Takafuji et al. (1983) obtained longer persistence, with some recolonization of patches, when dispersal was reduced and initial distributions were uniform, but no consistent effect of cell number; the first of these results again was unreplicated.

The most satisfactory laboratory evidence of persistence due to subdivision and dispersal comes from a study by Pimentel et al. (1963), in which a parasitic wasp consistently persisted longer with either of two of its hosts in systems containing more cells or with reduced parasite dispersal, than in smaller systems or ones with freer dispersal. V. Taylor & M. Hassell (unpubl.; see Hassell, this issue) similarly obtained increased persistence, and perhaps even stability, in a very subdivided parasitoid-host system compared with a homogeneous system; in this case, however, the "cells" were far smaller than the scale of even a local population, so this might be interpreted better as a study of within-cell patchiness.

Although some of them have serious weaknesses, taken as a whole these laboratory studies do support the hypothesis that spatial subdivision and dispersal can enhance the persistence of predator-prey systems. Being quite artificial, however, they do not say much about whether and when this occurs in nature. Well designed manipulations of a larger, more natural but still controlled system, such as the greenhouse mites studied by Nachman (1987a, b), would tell us much more about these questions, but they ultimately must be answered in the field.

4.2. Field

At present nothing is known directly about the role of regional population structure and dispersal in the persistence of natural predator-prey systems. Broadening the scope of inquiry to plant-herbivore interactions, however, produces one clear example of system persistence due to large-scale population subdivision: the biological control of *Opuntia* by *Cactoblastis cactorum* in Australia. Following the initial destruction by the moth of the massive cactus infestation, the system settled into persistent dy-

namics in which small, widely dispersed clumps of cactus lasted long enough to reproduce and disperse before being found and destroyed by the moth (Dodd 1959). Another plant-herbivore system, the ragwort-cinnabar moth interaction studied by van der Meijden (1979), also is cited as an example of regional persistence through dispersal, but this is not really appropriate since the "populations" are small, closely situated, and generally annual clumps of plants, equivalent to resource units rather than local populations.

Returning to predator-prey systems, Murdoch et al. (1985) suggest that extinction of local populations may occur in several cases of successful biological control. If this were true, it would be compelling evidence of the importance of migration for the persistence of those populations. Unfortunately, this line of argument has one major weakness: it is often difficult to prove extinction. Thus in several cases Murdoch et al. (1985) can conclude only that there is no evidence of a stable equilibrium. It also appears that several of the cases of Murdoch et al. (1985) may not involve true local populations in the sense of the hypothesis they, and the present paper, are concerned with: two concern patches of mosquito larvae, which clearly are not populations since immigration and recolonization is a necessary part of the life cycle, and other cases may represent "sink" populations which are maintained simply by immigration from a stable source population. Even in the two strongest cases, the olive scale and the cottony-cushion scale, almost all the extinctions appear to be on a spatial scale (individual trees) which is probably smaller than the foraging range of the natural enemies, and Huffaker et al. (1986) argue that extinction of olive scale even on this scale is very rare. Furthermore, Murdoch et al. (1984) describe one apparent extinction of olive scale from an entire grove, but the absence of recolonization over the following 12 years suggests that dispersal in fact is not capable of maintaining populations at this spatial scale. At present, then, we have some suggestions of local extinction in these systems, but little proof.

How might conclusive evidence of the role of inter-population dispersal in predator-prey interactions be obtained? The evidence and arguments of Murdoch et al. (1985) certainly point in a useful direction, and careful documentation of local extinctions, while subject to the difficulties just mentioned, would be very informative. The question of local extinctions, however, is pertinent only to the extreme version of the regional dispersal-persistence hypothesis represented in the cell-occupancy models. Investigation in nature of the more general hypothesis represented in the explicit within-cell models would ideally involve description of the internal dynamics of a set of local populations and of movements among them; even data on densities and immigration and emigration for a single local interaction would be valuable. A much simpler procedure, however, would be to alter migration in and out of some local populations, and compare their dynamics with those of control populations; this would be the most direct and conclusive way possible for addressing the basic question of what the effects of dispersal are on local populations, though it would provide less mechanistic information than would direct measurement of dispersal. Studies of one or the other sort are badly needed.

Acknowledgements. I am grateful to John Reeve for access to unpublished work and for much useful discussion, and especially to Ilkka Hanski and Tony Ives for many helpful comments on the manuscript.

References

Allen, J. C. 1975: Mathematical models of species interactions in time and space. — Amer. Nat. 109:319–342.

Andrewartha, H. G. & Birch, L. C. 1954: The distribution and abundance of animals. — 782 pp. Chicago University Press, Chicago.

—"— 1984: The ecological web: more on the distribution and abundance of animals. — Chicago University Press, Chicago.

Bailey, V. A., Nicholson, A. J. & Williams, E. J. 1962: Interactions between hosts and parasites when some individuals are more difficult to find than others. — J. Theor. Biol. 3:1–18.

den Boer, P. J. 1968: Spreading of risk and stabilization of animal numbers. — Acta Biotheor. 18:165–194.

—"— 1981: On the survival of populations in a heterogeneous and variable environment. — Oecologia 50:39–53.

Chewning, W. C. 1975: Migratory effects in predator-prey models. — Math. Biosci. 23:253–262.

Comins, H. N. & Blatt, D. W. E. 1974: Prey-predator models in spatially heterogeneous environments. — J. Theor. Biol. 48:75–83.

Crowley, P. H. 1981: Dispersal and the stability of predator-prey interactions. — Amer. Nat. 118:673–701.

Dempster, J. P. 1983: The natural control of populations of butterflies and moths. — Biol. Rev. 58:461–481.

Dodd, A. P. 1959: The biological control of prickly pear in Australia. *in* Biogeography and ecology in Australia. — Monogr. Biologicae 8:565–577.

Gause, G. F. 1934: The struggle for existence. — 163 pp. The Williams and Wilkins Company, Baltimore.

Gurney, W. S. C. & Nisbet, R. M. 1978: Predator-prey fluctuations in patchy environments. — J. Anim. Ecol. 47: 85–102.

—"— 1982: Modelling fluctuating populations (Ch. 10). — 360 pp. John Wiley & Sons, Chichester.

Hanski, I. 1983: Coexistence of competitors in patchy environment. — Ecology 64:493–500.

Hastings, A. 1977: Spatial heterogeneity and the stability of predator-prey systems. — Theor. Popul. Biol. 12:37–48.

Hilborn, R. 1975: The effect of spatial heterogeneity on the persistence of predator-prey interactions. — Theor. Popul. Biol. 8:346–355.

Horn, H. S., MacArthur, R. H. 1972: Competition among fugitive species in a harlequin environment. — Ecology 53:749–752.

Huffaker, C. B. 1958: Experimental studies on predation: Dispersion factors and predator-prey oscillations. — Hilgardia 27:343–383.

Huffaker, C. B., Kennett, C. E. & Tassan, R. L. 1986: Comparisons of parasitism and densities of Parlatoria oleae (1952–1982) in relation to ecological theory. — Amer. Nat. 128:379–393.

Huffaker, C. B., Shea, K. P. & Herman, S. G. 1963: Experimental studies on predation: complex dispersion and levels of food in an acarine predator-prey interaction. — Hilgardia 34:305–330.

Klinkhamer, P. G. L., de Jong, T. J. & Metz, J. A. J. 1983: An explanation for low dispersal rates: a simulation experiment. — Neth. J. Zool. 33:532– 541.

Lewontin, R. C. & Cohen, D. 1969: On population growth in a randomly varying environment. — Proc. Nat. Acad. Sci. (U.S.A.) 22:461–469.

Luckinbill, L. S. 1973: Coexistence in laboratory populations of Paramecium aurelia and its predator Didinium nasutum. — Ecology 54:1320–1327.

May, R. M. 1978: Host-parasitoid systems in patchy environments: a phenomenological model. — J. Anim. Ecol. 47:833–844.

Maynard Smith, J. 1974: Models in ecology (Ch. 6). — 136 pp. Cambridge University Press, Cambridge.

van der Meijden, E. 1979: Herbivore exploitation of a fugitive plant species: local survival and extinction of the cinnabar moth and ragwort in a heterogeneous environment. — Oecologia 42:307–323.

Metz, J. A. J., de Jong, T. J. & Klinkhamer, P. G. L. 1983: What are the advantages of dispersing; a paper by Kuno explained and extended. — Oecologia 57:166–169.

Morrison, G. & Barbosa, P. 1987: Spatial heterogeneity, population "regulation" and local extinction in simulated host-parasitoid interactions. — Oecologia 73:609–614.

Murdoch, W. W. 1979: Predation and dynamics of prey populations. — Fortschr. Zool. 25:295–310.

Murdoch, W. W., Chesson, J. & Chesson, P. 1985: Biological control in theory and practice. — Amer. Nat. 125:344–366.

Murdoch, W. W., & Oaten, A. 1975: Predation and population stability. — Adv. Ecol. Res. 9:1–131.

Murdoch, W. W., Reeve, J. D., Huffaker, C. B., & Kennett, C. E. 1984: Biological control of scale insects and ecological theory. — Amer. Nat. 123:371–392.

Nachman, G. 1987a: Systems analysis of acarine predator-prey interactions. I. A stochastic simulation model of spatial processes. — J. Anim. Ecol. 56:247–265.

—"— 1987b: Systems analysis of acarine predator-prey interactions. II. The role of spatial processes in system stability. — J. Anim. Ecol. 56: 267–281.

Nicholson, A. J. 1933: The balance of animal populations. — J. Anim. Ecol. 2:132–178.

Nicholson, A. J. & Bailey, V. A. 1935: The balance of animal populations, Part I. — Proc. Zool. Soc. Lond. 1935: 551–598.

Pimentel, D., Nagel, W. P. & Madden, J. L. 1963: Space-time structure of the environment and the survival of parasite-host systems. — Amer. Nat. 97:141–166.

Reeve, J. D. 1988: Environmental variability, migration and persistence in host-parasitoid systems. — Amer. Nat. [in press].

Slatkin, M. 1974: Competition and regional coexistence. — Ecology 55:128–134.

Takafuji, A. 1977: The effect of the rate of successful dispersal of a phytoseiid mite, Phytoseilus persimilis (Acarinia: Phytoseiidae) on the persistence in the interactive system between the predator and its prey. — Res. Popul. Ecol. 18:210–222.

Takafuji, A., Tsuda, Y. & Miki, T. 1983: System behaviour in predator-prey interaction, with special reference to acarine predator-prey system. — Res. Popul. Ecol. Supplement 3:75–92.

Taylor, A. D. 1988: Parasitoid density dependence and the dynamics of host-parasitoid models. — Amer. Nat. [in press].

Vandermeer, J. H. 1973: On the regional stability of locally unstable predator-prey relationships. — J. Theor. Biol. 41:161–170.

Zeigler, B. P. 1977: Persistence and patchiness of predator-prey systems induced by discrete event population exchange mechanisms. — J. Theor. Biol. 67:677–686.

Received 25.IX.1987, revised 6.I.1988
Printed 6.V.1988