Stabilizing effects in spatial parasitoid–host and predator–prey models: a review

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Abstract

We review the literature on spatial host–parasitoid and predator–prey models. Dispersal on its own is not stabilizing and can destabilize a stable local equilibrium. We identify three mechanisms whereby limited dispersal of hosts and parasitoids combined with other features, such as spatial and temporal heterogeneity, can promote increased persistence and stability. The first mechanism, "statistical stabilization", is simply the statistical effect that summing a number of out-of-phase population trajectories results in a relatively constant total population density. The second mechanism involves decoupling of immigration from local density, such that limited dispersal between asynchronous patches results in an effect that mimics density-dependence at the local patch level. The third mechanism involves altering spatially averaged parameter values resulting from spatial heterogeneity in density combined with non-linear responses to density. Persistence in spatially explicit models with local dispersal is frequently associated with self-organized spatial patterning.
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1. Introduction

The idea that spatial subdivision and dispersal can promote persistence of otherwise unstable predator–prey or parasitoid–host interactions has been around for a long time. Nicholson (1933), in response to the instability of the now-classic Nicholson–Bailey model, suggested that asynchrony between a collection of unstable populations could lead to persistence on a regional scale when populations in local patches were prone to extinction or displayed unstable dynamics. The mechanism that he proposed was one of "large numbers of independent local systems of oscillations, all phases of oscillations being represented in the environment at any given time" (Nicholson, 1933, p. 163). Hosts increase in abundance on parasitoid-free patches, but then eventually are found by the parasitoid and are driven locally extinct. Simultaneous extinction of all asynchronous patches is unlikely.

Non-spatial parasitoid–host models have generally been variations of either the Nicholson–Bailey model in discrete time or the Lotka–Volterra predator–prey model in continuous time. Neither of these models, without the addition of specific stabilizing mechanisms, leads to a stable equilibrium of the host and parasitoid. The Lotka–Volterra model produces neutrally stable oscillations in which the amplitude of fluctuations depends on the size of perturbations from equilibrium. The Nicholson–Bailey model produces diverging oscillations until one or both species goes extinct. Stabilizing factors can include a host carrying capacity, parasitoid interference, an invulnerable age class, a Type III functional response (Lotka–Volterra formulation only), and non-random parasitoid attack (Murdoch and Oaten, 1975; Hassell, 1978, 2000).

In recent years there has been a proliferation of new spatial models. Some of these models have suggested mechanisms by which dispersal can promote stability and persistence of unstable host–parasitoid dynamics, other than the hide-and-seek on asynchronous patches suggested by Nicholson. Others have shown that spatial subdivision and dispersal can at times have a destabilizing

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effect, or have shown that it can lead to complex spatial patterning. In this review, we analyze the literature on the effects of spatial subdivision and dispersal in host–parasitoid models. We attempt to identify and clarify the various mechanisms whereby dispersal can enhance or interfere with persistence, or stabilize or destabilize the dynamics.

Before commencing the review of models, we need to comment on our usage of the terms "stability" and "persistence". Many of the models discussed in this review contain two spatial scales: a local scale and a global scale. The local scale, depending on the model formulation, may consist of an individual patch, or simply some collection of nearby individuals. The global, or system-wide, scale consists of all patches or individuals in the system. Stability at the local scale represents the situation in which the dynamics at the local scale return to equilibrium following a perturbation. If all patches are stable, then the entire system will also be stable. We will use the term persistence to mean simply that the populations of hosts and parasitoids do not go extinct. In many models, dispersal allows the populations to persist at the global scale in cases in which populations on an individual isolated patch would go extinct. In some cases connecting a local population that would not persist in isolation to other

Table 1
Common models of within-patch dynamics

(a) Discrete–time models

Host : $H_{t+1} = \lambda H_t f(P_t)$	λ = host finite rate of increase
Parasitoid : $P_{t+1} = cH_t[1 - f(P_t)]$	$f(P_t)$ = fraction of hosts that escape parasitism
	c = parasitoid conversion efficiency

Name	Parasitism function	New parameters
Nicholson–Bailey (Nicholson and Bailey, 1935)	$f(P_t) = \exp(-aP_t)$	a = parasitoid attack rate
Negative–Binomial (May, 1978)	$f(P_t) = \left(1 + \frac{aP_t}{k}\right)^{-k}$	k = clumping parameter of negative-binomial distribution
Hassell–Varley (Hassell and Varley, 1969)	$f(P_t) = \exp(-a(P_t)^{1-m})$	m = parasitoid interference coefficient

Example: A two-patch Nicholson–Bailey Model (e.g. Adler, 1993)

$H_1(t+1) = (1 - \mu_h)\lambda H_1(t)e^{-aP_1(t)} + \mu_h \lambda H_2(t)e^{-aP_2(t)}$, H_i = density of hosts in patch $i = 1,2$

$H_2(t+1) = (1 - \mu_h)\lambda H_2(t)e^{-aP_2(t)} + \mu_h \lambda H_1(t)e^{-aP_2(t)}$, P_i = density of parasitoids in patch $i = 1,2$

$P_1(t+1) = (1 - \mu_p)H_1(t)(1 - e^{-aP_1(t)}) + \mu_p H_2(t)(1 - e^{-aP_2(t)})$, μ_h = fraction of hosts in each patch that disperse to other patch each year

$P_2(t+1) = (1 - \mu_p)H_2(t)(1 - e^{-aP_2(t)}) + \mu_p H_1(t)(1 - e^{-aP_1(t)})$, μ_p = fraction of parasitoids in each patch that disperse to other patch each year

(b) Continuous-time models

Host : $\frac{dH}{dt} = g(H) - f(P)H$	$g(H)$ = host rate of increase without parasitoids
Parasitoid : $\frac{dP}{dt} = cf(P)H - dP$	$f(P)$ = per-capita host mortality due to parasitoids
	c = parasitoid conversion efficiency
	d = per-capita parasitoid death rate

Name	$g(H)$ and $f(P)$	New parameters
Lotka–Volterra (Lotka, 1920b, a; Volterra, 1926)	$g(H) = rH$ $f(P) = aP$	r = host intrinsic rate of increase a = parasitoid attack rate
Lotka–Volterra with upper limit for the host population (Crowley, 1981)	$g(H) = rH$ if $H < K$ $\quad = 0$ if $H \geqslant K$ $f(H,P) = aP$	K = host carrying capacity
Rosenzweig–MacArthur (Rosenzweig and MacArthur, 1963)	$g(H) = rH\left(1 - \frac{H}{K}\right)$ $f(H,P) = \frac{aP}{1+aT_hH}$	K = host carrying capacity T_h = handling time per prey

Example: A two-patch Lotka–Volterra Model (e.g. Jansen, 1995)

$\frac{dH_1}{dt} = rH_1 - aH_1P_1 - e_hH_1 + e_hH_2$, $\frac{dH_2}{dt} = rH_2 - aH_2P_2 - e_hH_2 + e_hH_1$,

$\frac{dP_1}{dt} = caH_1P_1 - dP_1 - e_pP_1 + e_pP_2$, $\frac{dP_2}{dt} = caH_2P_2 - dP_2 - e_pP_2 + e_pP_1$,

e_h = per-capita emigration rate of hosts from each patch.

e_p = per-capita emigration rate of parasitoids from each patch.

patches through dispersal can also lead to persistence on the local patch.

In this review we first make the point that random dispersal on its own cannot stabilize an unstable equilibrium on a local patch, and in some cases it can actually destabilize a stable equilibrium. However, many papers have shown that spatial structure and limited dispersal ability of individuals can enhance the persistence of parasitoid–host systems, and when combined with other features, such as spatial or temporal heterogeneity, can lead to a stable equilibrium at the local scale in every patch, and therefore also at the global scale.

We next discuss three stabilizing mechanisms that are common to a number of models (see also Murdoch et al., 2003, Chapter 10). The first two of these mechanisms result from out-of-phase fluctuations in different parts of the environment. These are:

(A) "Statistical stabilization". This is not really stability in the sense of leading to a system-scale stable equilibrium, but simply the fact that the summing over out-of-phase fluctuations across the environment will result in a relatively constant total density.

(B) Decoupling immigration from local density in systems with out-of-phase fluctuations and weakly coupled dynamics. Random dispersal between areas at different points in their trajectories can result in the immigration rate into each patch being negatively correlated with the density in the local patch. This can result in real stability.

(C) Non-linear spatial averaging. The third mechanism is associated with static self-organized spatial patterning. Spatial heterogeneity in density, combined with non-linear responses to density, can result in the values of demographic parameters experienced by individuals in a population differing from those that would occur if the system were well-mixed. In some cases altering the values of parameters can shift the system from an unstable to a stable region of parameter space (e.g. the Rosenzweig–MacArthur model, Table 1) and therefore be stabilizing.

Many of the models involve aspects of more than one of these stabilizing mechanisms.

2. Scope of this review

To find papers for inclusion in this review, we scanned the tables of contents of the following journals for the years 1980 to 2002: *American Naturlaist Ecology*, *Ecology Letters*, *Ecological Modeling*, *Ecological Monographs*, *Journal of Animal Ecology*, *Journal of Mathematical Biology*, *Journal of Theoretical Biology*, *Nature*, *Oikos*, *Oecologia*, *Proceedings of the Royal Society of London Series B*, *Science*, and of course *Theoretical Population Biology*. We also included all of the relevant classic papers that are frequently cited from years prior to 1980. We searched for papers that dealt with the long-term dynamics of spatially explicit host–parasitoid or predator–prey models, including patch models, lattice models, individual-based models, and reaction–diffusion models (our classification of models is described below). We included those models that were either intended to represent host–parasitoid interactions, or that could be easily interpreted as such. We have concentrated here on one-host/one-parasitoid species models, and have excluded papers that modeled exclusively multiple host or parasitoid species. For lack of space, we do not thoroughly review the vast literature on reaction–diffusion models, but instead concentrate on a few key results for comparison with other model frameworks.

In this review we have chosen to concentrate on models that follow the density or number of individuals in the population, and exclude patch-occupancy models that follow the fraction of occupied patches (e.g. Hastings, 1977; Gurney and Nisbet, 1978; Sabelis and Diekmann, 1988), and discrete-event models in which the patches pass through a series of a few discrete states (e.g. Maynard Smith, 1974; Zeigler, 1977). Some of the individual-based models, which we do review, follow grids of cells that are either full or empty, and therefore these models might be interpreted as patch-occupancy models. The individual-based models that we have included, however, also incorporate rules describing the movement of individuals, rather than just the colonization or extinction of patches. We have not included papers that investigate models analogous to spatial models, but in another context. For example, we have not included Smith and Mead's (1974) age-structured model, which is very similar in form to some of the continuous-time patch models of a spatially structured system (Murdoch et al., 2003). In addition we have decided not to review papers that deal primarily with simulation models of particular systems.

We have chosen to exclude papers that model only the effects of aggregation of parasitoids to regions of high density or independent of host density within a population. Such models that look at the effects of variability between hosts in the risk of parasitism is a very large literature in itself, and we do not have space to review it here (see e.g. Hassell and May, 1973, 1974, 1988; Hassell, 1978; May, 1978; Chesson and Murdoch, 1986; Murdoch and Stewart-Oaten, 1989; Hassell and Pacala, 1990; Hassell et al., 1991b; Pacala and Hassell, 1991; Rohani et al., 1994a). These aggregation models consider the movement of parasitoids as they search for hosts within a population. We instead review models of the effects of dispersal at a larger spatial scale. The

distinction that we make here is that we include only models in which the entire population of hosts and/or parasitoids is not "well-mixed" each generation. Some of the formulations of the models that we discuss here, especially the patch models, are termed host–parasitoid metapopulations by some authors, with local subpopulations of hosts and parasitoids linked together by dispersal. Other authors, however, limit the use of the term metapopulation to describe only the dynamics of a single species (Holyoak et al., in review). Here we avoid the metapopulation terminology and instead talk about within-patch (or local) and system-wide (or global) dynamics.

3. Classification of models

3.1. Reaction–diffusion models

In reaction–diffusion models, both time and space are continuous variables (Okubo and Levin, 2001). Density functions describe the current density of the population of each species across space. The reaction part of the reaction–diffusion model describes the interaction between species. This can take on the form of a simple Lotka–Volterra predator–prey interaction, or any other type of continuous-time consumer–resource model. The diffusion part of the model describes the movement of each species through space. In true diffusion models, the individuals of each species move randomly in all directions, so the net rate of flow is from high to low density areas. Other types of movement rules have also been modeled in this partial differential equation framework. Because this literature is immense and has been discussed extensively in other papers and books (e.g. Okubo, 1980; Murray, 1989; Hastings, 1990; Pascual, 1993; Holmes et al., 1994; Okubo and Levin, 2001), we do not review these models here but summarize their results for comparison with the results of other models.

3.2. Individual-based models

In the individual-based models covered here, the population is composed of discrete individual prey and predators that can move around the landscape, eating, reproducing and dying. The most common way in which this is implemented is to divide space into a discrete lattice of cells, each just large enough to hold one prey and/or one predator. Time is frequently also divided into discrete time steps during which individuals have some probability of moving, giving birth, and dying according to some set of rules. An alternative representation involves continuous time and space, in which, instead of following the state of individual lattice cells, the model follows the position and state of individual

prey and predators in space. The individual-based approach allows the authors to study the effects of demographic stochasticity along with the movement of individual prey and predators (de Roos et al., 1991; Molofsky, 1994; Donalson and Nisbet, 1999).

3.3. Patch and lattice models

In patch and lattice models, the dynamics of the host and parasitoid populations in each patch are modeled explicitly. Several of the commonly used forms for the within-patch dynamics are shown in Table 1. In discrete-time patch models, once per generation a fraction of hosts and/or parasitoids leave each patch and disperse to other patches. Discrete time models must include assumptions about the order of events during the season (e.g. host reproduction then parasitism then dispersal, or host reproduction then dispersal then parasitism, etc.). Whereas in continuous time models, hosts and parasitoids generally leave each patch continuously at some rate and disperse to other patches. In continuous time models, all processes occur concurrently, so no assumption about the order of events is necessary.

In patch models, the spatial location of each patch does not matter, and dispersal is said to be global, such that all patches in the system potentially can receive dispersers from every other patch. In lattice models, the patches are arranged in 1-D, 2-D, or 3-D arrays, and dispersal can be local, such that individuals leaving a patch can arrive at only the nearest n patches (where n is frequently 4, 8, etc.). The properties of the patch and lattice models reviewed here are listed in Table 2.

See Durrett and Levin (1994) for a comparison of different spatial modeling approaches, and Keeling et al. (2002) for a technique for deriving some analytical results from spatial stochastic individual-based models using moment equations.

4. Dispersal per se is not stabilizing

Dispersal per se does not stabilize an unstable predator–prey system. A general result of reaction–diffusion models is that diffusive movement of individuals cannot stabilize an otherwise unstable predator–prey interaction. If the non-spatial model has an unstable equilibrium, then the spatially homogeneous equilibrium in spatial reaction–diffusion models will also be unstable (Levin, 1974; Murray, 1975; Hastings, 1990; Okubo and Levin, 2001). Similar results have been found with patch models. Allen (1975) showed analytically for discrete-time models with Nicholson–Bailey within patch dynamics and nearest neighbor dispersal, that the instability of the equilibrium in the local patch implies instability of the ensemble of patches. Reeve (1988) repeated this analysis with a Negative-Binomial

Table 2
Summary of key features and main results of patch and lattice models reviewed here

(2.1) Patch and lattice models with identical patches

Discrete-time

Reference	Dimension	# patches	Analytical/simulation	Parasitism function[a]	Dispersal[b]	Main results
Allen, 1975	1, 2, or 3	Any	Analytical	Nich–Bailey	NN, CF	Dispersal between identical patches cannot stabilize N–B dynamics
	1	Any	Analytical	Hassell–Varley	NN, CF	Dispersal has no effect on stability if hosts and parasitoids move at same rate; equilibrium can be destabilized if hosts and parasitoids move at very different rates
	2	5 × 5	Simulation	Hassell–Varley	NN, CF	Spatial rings when parasitoids move faster than hosts
Reeve, 1988, 1990	n/a	Any	Analytical	Neg–Binomial	Global, CF	Dispersal has no effect or can be destabilizing when hosts and parasitoids move at different rates
Hassell et al., 1991; Comins et al., 1992	2	Up to 30 × 30	Simulation	Nich–Bailey or Neg–Binomial	NN, CF	Complex spatial dynamics, system-wide dynamics bounded and with reduced variability
Adler, 1993	n/a	2 or 50	Simulation	Nich–Bailey	Global, CF	Different initial conditions can potentially lead to persistent oscillations with only 2 patches
Rohani and Miramontes, 1995	2	Up to 128 × 128	Simulation	Nich–Bailey	NN, parasitoids aggregation	Intermediate levels of parasitoid aggregation lead to lowest population variability
Rohani et al., 1996	1	10	Both	Neg–Binomial	NN, CF	Dispersal has no effect, or is destabilizing when *H&P* movement is asymmetric
Taylor, 1998	n/a	100	Simulation	Nich–Bailey or Neg–Binomial	Global, CF	Persistence in systems of unstable patches for limited ranges of parameters, with different initial conditions in the different patches
Rohani and Ruxton, 1999	1	Up to 30	Both	Neg–Binomial host d-d, host threshold, refuge, or inverse d-d parasitism	NN, CF	Dispersal can destabilize local equilibrium with identical densities in all patches, resulting in stable pattern formation or asynchronous dynamics between patches. Factors that stabilize local patches reduce likelihood of dispersal-induced instabilities
Kean and Barlow, 2000	2	30 × 30	Simulation	Nich–Bailey, Neg–Binomial or Type II FR, host d-d	NN, CF	Lattice models could lead to lower host density than non-spatial models
McCann et al., 2000	1	3	Both	Nich–Bailey, Type II FR, host d-d, or Allee effect	NN, CF	Stable pattern formation when logistic host population growth combined with Type II FR or Allee effect

Table 2 (*Continued.*)

(2.2) Patch and lattice models with identical patches
Continuous-time

Reference	Dimension	# patches	Simulation/ analytical	Parasitism function	Dispersal[b]	Main results
Levin, 1974	n/a	2	Analytical	LV, d-d host birth, allee effect	Global, CR	Dispersal in some cases can destabilize equilibrium
Crowley, 1981	n/a	Any	Analytical	LV, Type II FR, host ceiling	Global, CR	Dispersal cannot stabilize equilibrium
Nisbet et al., 1992	n/a	2	Both	LV	Global, CR	System is neutrally stable, like individual patch
Jansen, 1995	n/a	2	Both	LV	Global, CR	System is neutrally stable like individual patch, but different initial conditions can lead to lower amplitude fluctuations
	n/a	2	Both	Rosenzweig–MacArthur	Global, CR	Cycles with different densities in the two patches possible
Weisser and Hassell, 1996	n/a	2, any	Analytical	Lotka–Volterra	Global, CR or d-d parasitoid emigration rate	A pool of dispersers is stabilizing
Weisser et al., 1997	n/a or 1	4, any	Analytical	Lotka–Volterra	Global, or NN through dispersal pool, CR	A pool of dispersers is stabilizing
	n/a	2	Analytical	LV, Type II FR	Predators only, global or NN dispersal pool, CR	Can result in stable equilibrium, lead to stable limit cycles, or unstable limit cycles
de Roos et al., 1998	n/a	5, any	Both	Rosenzweig–MacArthur	Prey: Global, CR Predators: constant density across space	Constant predator density and non-linear functional response lead to multiple equilibria and spatial pattern formation
Jansen, 2001	n/a	2	Both	Rosenzweig–MacArthur	Global, CR	Asynchronous dynamics for intermediate levels of parasitoid dispersal. Different patches have different constant densities for high levels of dispersal
Neubert et al., 2002	n/a	2, any	Both	LV	Global, CR, distribution of travel times	Transit time of hosts and/or is almost always stabilizing

(2.3) Patch and lattice models with spatial and/or temporal variability

Reference	Dimension	# patches	Source of variability	Parasitism function	Dispersal[b]	Main results
Murdoch and Oaten, 1975	n/a	2	Spatial differences in H birth, P death or attack rates	Lotka–Volterra	Global, CF	Dispersal between patches with different parameters can be stabilizing
Crowley, 1981	n/a	1, 10, 100	Spatial differences in H birth rate and P death rate	Type II FR, host ceiling	Global, CF	Persistence highest at intermediate level of dispersal
Holt, 1984, 1985	n/a	2	Prey on only one patch	Type I, II or III FR	Global, CR	Random dispersal to a predator sink can be stabilizing
Hassell and May, 1988	n/a	5, any	Fixed spatial differences in fraction of H and/or P immigrating	Nich–Bailey	Global, CF emigrate, patch-specific fraction immigrate	Fixed spatial differences in fraction of H or P can be stabilizing or at least enhance persistence
Reeve, 1988, 1990	n/a	100	Spatial and temporal variability in H birth rate, or P attack rate	Neg–Binomial	Global, CF	Persistence highest at low levels of dispersal and high levels of environmental variability
	n/a	100	Spatial and temporal variability in H birth rate, or P attack rate	Neg–Binomial	Global, CF d-d host emigration, P aggregation	Persistence time decreased by density-dependent migration or parasitoid aggregation
Hassell et al., 1991; Comins et al., 1992	2	Up to 30×30	Spatial and temporal differences in H births	Nich–Bailey or Neg–Binomial	NN, CF	Reduces likelihood of crystal lattice structures, little effect on spatial spirals and spatial chaos, except easier to start
Ives, 1992	n/a	2, or 2 to 5	Fixed spatial differences in H birth or dispersal rate	Type II FR	Global CR for hosts, P aggregation	Increasing parasitoid aggregation can be either stabilizing or destabilizing when the host dispersal rate differs between the patches and the average host dispersal rate is low
Murdoch et al., 1992	n/a	2	Fixed spatial differences in H births	Lotka–Volterra	Global, CR	Different prey birth rates in the two patches is stabilizing
	n/a	2	Fixed spatial differences in H births	Lotka–Volterra	Global, d-d H immigration	Strong density-dependence in host emigration rate is destabilizing because it synchronizes dynamics in two patches
	n/a	2	Fixed spatial differences in H births	Lotka–Volterra	Global, P aggregation	Weak P aggregation to patches of high H is stabilizing, but strong aggregation is destabilizing because it synchronizes dynamics in two patches

Table 2 (*Continued.*)

(2.3) Patch and lattice models with spatial and/or temporal variability

Reference	Dimension	# patches	Source of variability	Parasitism function	Dispersal[b]	Main results
Adler, 1993	n/a	2 or 50	Spatial and temporal variability in H growth rate, emigration rates or attack rates	Nicholson–Bailey	Global, CF	Variability decreased the probability of persistence. Variability allows the two patches started at different conditions to come into phase
Holt and Hassell, 1993	n/a	2	One patch is a complete refuge from parasitism. H birth rate can differ between the patches	Nicholson–Bailey	H only Global, CF	Coupling to a refuge from parasitism that is a sink for the host has a stabilizing effect. Coupling to a refuge that has a relatively high host of increase can allow the host to escape regulation by the parasitoid Coupling to a refuge with a very low rate of increase can lead to extinction in both patches
	n/a	2	P attack rate is very high in one patch, P conversion efficiency can differ between the two patches	Nicholson–Bailey	Global, CF	High levels of dispersal between the two patches is stabilizing. Differences in P's conversion efficiency between the patches can be stabilizing in a weakly coupled system
Jansen, 1995	n/a	2	Temporal variability in predator death rate	Lotka–Volterra	Global, CR	Temporal variability decreases amplitude of fluctuations
Wilson and Hassell, 1997	2	100×100	Integer-based model, demographic stochasticity in host birth rate, parasitism function, and migration	Nicholson–Bailey	NN, CF	Demographic stochasticity decreases probability of persistence. Spatial spirals and spatial chaos can occur

Reference				Parasitism[a]	Dispersal[b]	Results
Taylor, 1998	n/a	100	Spatial and temporal variability in H birth rate, or P attack rate, or P conversion efficiency	Nicholson–Bailey or Negative Binomial	Global, CF	Persistence occurred with low levels of dispersal of H and P and with high levels of environmental variability. Persistence easier with variability in H birth rate and P attack rate than for variability in P conversion efficiency
	n/a	100	Fixed spatial variability in H birth rate, P attack rate, or P conversion efficiency	Nicholson–Bailey or Negative Binomial	Global, CF	Persistence occurred with low levels of dispersal of H and P and with high levels of environmental variability
Namba et al., 1999	n/a, 1 or 2	2 or many	Prey carrying capacity differs between patch	Lotka–Volterra with host carrying-capacity	Predator only NN, CF	A predator sink patch can lead to extinction of the system

[a] *Parasitism abbreviations*: FR = Functional response; d-d = density-dependence in host rate of increase; LV = Lotka–Volterra local patch dynamics; Nich-Bailey = Nicholson–Bailey local patch dynamics; Neg-Binomial = Negative-binomial local patch dynamics.
[b] *Dispersal abbreviations*: NN = Nearest-Neighbor, dispersing hosts and/or parasitoids are distributed among nearest neighboring patches; Global = Global Dispersal, dispersing hosts and/or parasitoids are distributed equally among all patches in the system; CF = Constant Fraction, in discrete-time models, each time period a constant fraction of hosts and/or parasitoids leave each patch; CR = Constant Rate, in continuous-time models, hosts and/or parasitoids continuously emigrate from each patch at a constant per-capita rate.

parasitism function and global, rather than local, dispersal between identical patches, and found that dispersal between patches could not stabilize an otherwise unstable equilibrium at either the patch or global scales (see also Rohani et al., 1996).

In continuous-time systems with Lotka–Volterra local patch dynamics linked together by dispersal at a constant rate, the only possible equilibrium was the symmetrical equilibrium with equal densities in each patch (Nisbet et al., 1992; Jansen, 1995). Any differences due to initial conditions between the patches eventually disappeared, and the overall system cycled in a neutrally stable manner (although the amplitude of the cycles was potentially smaller than those in a non-spatial model, as will be discussed below; Jansen, 1995). This result again carries over from continuous-time, continuous-space reaction–diffusion models with Lotka–Volterra local dynamics (Murray, 1975). All solutions become spatially uniform, with densities in the entire system oscillating in neutrally stable cycles. In a more complicated continuous-time predator–prey system with a saturating Type II functional response and an absolute upper limit on the prey density, Crowley (1981) showed analytically that a spatial system of identical patches linked by global dispersal had the same local stability properties as an isolated single patch system.

When the dispersal rates of host and parasitoids are very different, random dispersal between patches can actually destabilize the homogeneous equilibrium with identical densities in each patch (Allen, 1975; Reeve, 1988), potentially resulting in persistent, but spatially heterogeneous, dynamics (Levin, 1974; Allen, 1975; Rohani et al., 1996; Rohani and Ruxton, 1999).

5. Spatial mechanisms resulting in stability or increased persistence

Although random dispersal on its own cannot stabilize an otherwise unstable parasitoid–host equilibrium, many papers have revealed situations in which spatial structure can lead either to indefinite persistence in systems that would otherwise have unbounded dynamics, or in some cases lead to true stability at the local and system-wide levels. In reviewing the papers, three different classes of stabilizing or persistence-promoting mechanisms emerged (Murdoch et al., 2003, Chapter 10). The distinction between these three mechanisms is to some degree artificial, and many models contain aspects of more than one of these mechanisms. The first two mechanisms result from asynchronous dynamics in different parts of the environment, the third is a consequence of spatial heterogeneity combined with non-linearities in the interaction. Thus, spatial heterogeneity is a crucial component of all three of these mechanisms. We will

now describe these three mechanisms in more detail and review the models that illustrate their stabilizing or persistence-promoting effects.

5.1. Statistical stabilization from out-of-phase fluctuations

The simplest way in which space can promote persistence in consumer–resource interactions is the mechanism proposed by Nicholson (1933), in which the populations in different regions of space are at different points in their unstable consumer–resource fluctuations. Not all areas are simultaneously at high or low densities. If the prey population is driven to low densities or driven extinct by the predator in some areas, it can be recolonized from other areas. This can occur only when there is restricted movement of individuals, so that the dynamics on different parts of the environment can remain asynchronous. "Statistical stabilization" is not really stability as it is normally defined. It is simply the statistical result that if you sum up the population densities in a number of asynchronous, out-of-phase, trajectories, the total density across the region has a much lower temporal variability than an individual patch. Through this mechanism the population density in each area continues to fluctuate, but the total population density across the region is relatively constant, and persistence of the system is enhanced. Spatial subdivision and limited dispersal ability of individuals is necessary for this mechanism to allow the dynamics in different spatial regions to remain asynchronous.

This mechanism is well illustrated by the individual-based models of the research group of McCauley, de Roos, and Wilson, who first coined the term "statistical stabilization" (de Roos et al., 1991; McCauley et al., 1993, 1996; Wilson et al., 1993). Their individual based models divide space into a square lattice of cells, with each cell holding at most one prey and one predator individual. The rules describing the interaction between individuals generally approximate either a Rosenzweig–MacArthur or Lotka–Volterra model, and in the different models prey and predators can be sedentary, move locally (to one of the neighboring cells), or move globally. In their first paper, de Roos et al. (1991) showed that when prey did not move, the model with global dispersal of predators resulted in dynamics equivalent to the non-spatial Rosenzweig–MacArthur model. For parameters in the unstable region of the Rosenzweig–MacArthur model, the spatial model with global dispersal was also unstable, displaying synchronous system-wide fluctuations in density. However, when the predators had limited local dispersal, the magnitude of the fluctuations in total density at the system-wide scale was greatly reduced. The dynamics in individual parts of the lattice continued to fluctuate, but

dynamics in different sections of the lattice were asynchronous, such that at the scale of the entire lattice the densities of prey and predators were relatively constant through time. They defined the "characteristic spatial scale" as the spatial scale below which the dynamics resembled a well-mixed system. Above this scale the dynamics in different parts of the system were only weakly coupled.

Wilson et al. (1993) used the same approach as de Roos et al. (1991) to study an individual-based version of the Lotka–Volterra predator–prey system. They found again that, with limited dispersal of prey and predators, total population densities could show relatively constant densities, due to averaging across asynchronous dynamics in different portions of the system. This is perhaps surprising because it is in contrast to the results of reaction–diffusion models including Lotka–Volterra predator–prey dynamics with diffusive movement. As mentioned above, in reaction–diffusion models the only long-term system-wide dynamics possible is synchronous, neutrally stable fluctuations of the sort observed in the non-spatial system (e.g. Okubo, 1980; Hastings, 1990). Wilson et al. (1993) concluded that this discrepancy was due to the fact that their simulations divide time and space into discrete steps (if the time steps and grid size are made vanishingly small, they recover the homogeneous solution), and due to demographic stochasticity from following discrete individuals, which provides a source of continuous local perturbation to the system.

Not all movement has the same effect on the stability of the system in these individual-based models. Wilson et al. (1993) and McCauley et al. (1993, 1996) found that prey movement had a stronger effect on stability than did predator movement. With high levels of prey movement, prey could temporarily escape predator control, leading to a spatial version of prey escape cycles (see e.g. de Roos et al., 1990) and larger scale fluctuations. McCauley et al. (1996) found that a similar asymmetry existed between stages of the predator. Because adults not only ate prey but also reproduced, it was the adult age class that caused "overcompensation," in which predators overshot prey control, leading to cycles. Adult movement, therefore, had larger effects than juvenile movement, led to greater synchrony across the lattice, and increased fluctuations or instability (McCauley et al., 1996). See also Wilson (1996, 1998) for further analysis of this result.

Donalson and Nisbet (1999) also studied an individual-based spatial version of the Lotka–Volterra predator–prey model, but with a discrete-event simulator. Their model followed the position, direction, and velocity of each prey and predator moving in a two-dimensional square spatial arena. They compared their results to a stochastic birth–death implementation of the Lotka–Volterra model, which contained the effects of demographic stochasticity from

limited numbers of individuals, but not the effects of explicit space. They found that for small systems with few individuals, both the non-spatial and spatial models went extinct rapidly. For large systems, large amplitude oscillations in density swept as waves across the system, but the system as a whole persisted.

The statistical stabilization mechanism also contributes to the long-term persistence in many patch and lattice models. Global dispersal between identical patches can reduce the amplitude of fluctuations in total density, and can enhance system-wide persistence in both continuous-time (Crowley, 1981; Jansen, 1995, 2001) and discrete-time patch models (Reeve, 1988, 1990; Adler, 1993; Taylor, 1998). However in these models our second mechanism of "decoupling immigration from local dynamics", described next, must also play a role, because the population fluctuations in individual patches are also reduced.

Local dispersal between identical patches in lattice models frequently results in complex spatial dynamics, e.g. spiral waves, spatial chaos, and persistent spatial differences in density (Hassell et al., 1991a; Comins et al., 1992; Rohani et al., 1994b; Rohani and Miramontes, 1995). One consequence of spatial pattern formation, is that the dynamics in different parts of the system remain asynchronous, so that the amplitude of fluctuations in total density, summed across the system, is generally lower than that of an individual patch, i.e. "statistical stabilization". For example, Hassell et al. (1991a) and Comins et al. (1992) linked together identical patches with unbounded Nicholson–Bailey local dynamics in a two-dimensional array with local dispersal of hosts and parasitoids to nearest neighboring patches. The Nicholson–Bailey model in an isolated patch of course produces only non-persistent diverging oscillations, eventually leading to the extinction of the parasitoid or the host and then the parasitoid. Hassell et al. (1991a) and Comins et al. (1992) found that when the patches were linked with local dispersal, a system with a large number of patches could persist, and display a wide range of complex spatial dynamics (see also Kean and Barlow, 2000). Wilson and Hassell (1997) showed that demographic stochasticity in an integer-based version of the Hassell et al. (1991a) model led to a greater chance of extinction at the system level, especially when the parasitoid attack rate was high (which leads to low densities of hosts and parasitoids). The model could still produce the complex patterns of spatial cycles and spatial chaos; however, the fixed spatial patterns (their "crystal lattice" structures) were eliminated by demographic stochasticity.

5.2. Immigration uncoupled from local dynamics

5.2.1. Immigration uncoupled from local dynamics

The second stabilizing mechanism commonly seen in spatial host–parasitoid (or predator–prey) models can be easily illustrated by considering a model of a single patch with constant immigration from outside the system. A simple example of this is the Lotka–Volterra predator–prey model with a constant immigration rate of prey:

$$\text{Host (or prey)}: \quad \frac{dH}{dt} = rH - aHP + R.$$

$$\text{Parasitoids (or predators)}: \quad \frac{dP}{dt} = caHP - dP,$$

where, as in the standard Lotka–Volterra model, r is the host intrinsic rate of increase, a is the attack rate, c is the conversion efficiency, and d is the parasitoid death rate. R is the constant immigration rate of hosts. This model has a stable equilibrium for any value of $R > 0$. The constant input of hosts is a relatively large contribution to the host population when the host population is small, and a relatively small contribution when the host population is large. Thus the "relative immigration rate", i.e. immigration relative to the local population size, decreases as a function of local population size. This causes $\left(\frac{1}{H}\right)\frac{dH}{dt}$ to be a decreasing function of the host density in the patch (for any given density of P), and therefore has a stabilizing effect. In simple consumer–resource models, a constant input of either the resource or consumers is stabilizing in this way (Crowley, 1981; Reeve, 1988; Murdoch et al., 2003). There has been some disagreement in the literature about whether a constant input of individuals from outside a local patch should be referred to as "density-dependence" (e.g. Sale and Tolimieri, 2000; Berryman, 2002). This discussion has been especially relevant to defining "density dependence" for open populations. We would like to sidestep that argument here and simply say that it causes the relative growth rate of the local patch, $\left(\frac{1}{H}\right)\frac{dH}{dt}$, to decrease with increasing H, and therefore is stabilizing.

An immigration rate that is not constant, but is to some degree uncoupled from the dynamics of the local population, such as immigration from another asynchronous patch, can be stabilizing in the same way. When patches with asynchronous dynamics are linked through dispersal, the per-individual input of individuals into each patch can become a decreasing function of the local density in each patch. For example, if the dynamics in the patches are out-of-phase, then not all patches will reach low population densities at the same time, therefore the patches currently at their troughs will receive a relatively high per-individual input from other patches. In many of the patch models discussed in this article, the dynamics in the local patch are stabilized through this mechanism, also resulting in stable system-wide dynamics (e.g. Crowley, 1981; Murdoch et al., 1992; Holt and Hassell, 1993; Jansen, 1995; Weisser and Hassell, 1996; Weisser et al., 1997; Jansen, 2001; Neubert et al., 2002).

There are two key features necessary for this mechanism to work. First, there must be some finite, but limited amount of dispersal between the patches. Dispersal must be high enough to link the patches together, but low enough so that the patches are not synchronized (Reeve, 1988; Murdoch et al., 1992). Second, there must be some feature that leads to persistent differences in the dynamics between patches. In the next several subsections we review the types of features that promote persistent differences between the patches, and therefore stability, or at least increased persistence, through this mechanism.

5.2.2. Processes that promote uncoupling of immigration from local dynamics

(a) *Fixed spatial differences between patches*: If fixed differences between the parameters in the patches cause the dynamics on the different patches to be asynchronous or have differently shaped trajectories, then any random or constant rate of dispersal between the patches can translate into a per-individual input of individuals into each patch that is a decreasing function of local density (Murdoch et al., 2003).

In continuous time, Murdoch and Oaten (1975) first noted that linking two Lotka–Volterra patches with different demographic parameters could stabilize the dynamics in each patch. They also found counter-examples, however, in which dispersal between the patches could be destabilizing, especially when both prey and predators dispersed. Murdoch et al. (1992) showed in a two-patch Lotka–Volterra system in which the patches differed only in prey birth rate, that as the difference in birth rate increased, the stability of the equilibrium increased (as measured by the magnitude of the dominant eigenvalue). The increase in stability was accompanied by an increase in the level of asynchrony between the densities on the two patches.

In discrete time, Taylor (1998) modified a model of Reeve (1988) in which patches with Nicholson–Bailey or Negative-Binomial local dynamics were linked together through random dispersal. Taylor added fixed spatial differences to the patches (each patch was assigned a random host birth rate and parasitoid attack rate at the start of the simulation, which it retained for the duration of the simulation), and showed that persistence was most likely with low dispersal rates and high levels of spatial environmental variability.

Sometimes such fixed spatial differences can be framed in terms of source–sink dynamics. Holt (1984, 1985) investigated a two-patch source–sink model with non-dispersing prey, and randomly dispersing predators. For prey the two patches were equivalent, although one patch was a sink habitat for predators. He found similar stabilizing results, particularly if dispersal rates were not too high. In a similar model, Namba et al. (1999) found stabilizing effects of sink habitats depen-

dent on the dispersal of the predator and the ratio of the prey carrying capacity in the source relative to the sink. Higher dispersal rates required higher source carrying capacities so that the predators would not all be drained into the sink. Holt and Hassell (1993) investigated a parallel host source–sink situation with parasitoids confined to one patch and found again that coupling to a sink could have a stabilizing effect. Stabilization resulted because the sink provided a buffer during times of high parasitism rate in the source.

Fixed spatial differences can also lead to refuge effects. Hassell and May (1988) studied a model in which hosts and/or parasitoids redistributed themselves unevenly each generation. If all hosts and parasitoids leave each patch every generation, then this becomes the within-population parasitoid aggregation models of Hassell and May (1973, 1974), not covered here. If parasitoids were sedentary, and all hosts dispersed each generation, then persistent patch-specific immigration differences could have a stabilizing effect when host reproductive rates were low. Parasitoids survived only in the patches with high host immigration, and the other patches acted as refuges. If parasitoids redistributed each generation, and hosts remained fixed, then enhanced persistence was possible if parasitoids failed to find all of the patches each generation.

(b) *Differences in initial conditions*: Differences in the initial conditions between patches linked by limited dispersal can, at least initially, result in the relative immigration into each patch decreasing with local density. This is not sufficient to result in stability, but it can reduce the amplitude of fluctuations in the patches (Murdoch et al., 2003), and promote persistence.

In continuous-time, Jansen's (1995) system of 2 linked patches, with Lotka–Volterra local dynamics and differing only in initial conditions, displayed neutrally stable oscillations, in the same way as unlinked patches. Any differences due to initial conditions disappeared through time, and the patches eventually became identical. However, with small differences in initial conditions between the patches and low levels of dispersal, connected patches can display smaller amplitude neutrally stable cycles than unconnected patches. This reduction in the amplitude of the fluctuations comes about because, up until the point that the dynamics on the two patches come into phase with each other, the per-individual input of individuals into each patch is negatively correlated with local density. Jansen (1995, 2001) also found that when the dynamics in each of the identical local patches is of the Rosenzweig–MacArthur form, rather than Lotka–Volterra, the total densities of the overall metapopulation are often much less variable than the densities in isolated patches. In the case of the Rosenzweig–MacArthur model, this can occur because the system can settle into a state in which the two patches are not symmetrical,

and the densities in the two patches fluctuate in an out-of-phase manner. Crowley (1981) found a similar result in his model with a Type II functional response and an upper limit on host density.

In discrete-time, Adler (1993) found that a system of only 2 identical patches with Nicholson–Bailey local dynamics, linked together with low levels of dispersal, could produce bounded oscillations for some combinations of initial conditions, provided the initial conditions in the two patches were not too similar to each other. When the system persisted, the 2 patches oscillated out of phase with each other, with the different patches showing similar, although slightly different, cycles from each other. This allowed the per-individual input of individuals into each patch to be negatively correlated with local density, resulting in persistent within-patch dynamics, rather than the unbounded dynamics of an isolated Nicholson–Bailey patch. In systems of multiple patches with Nicholson–Bailey local dynamics, differing only in initial conditions, Reeve (1988) found that low rates of dispersal between patches with Nicholson–Bailey local dynamics could substantially extend the time to extinction; see also Adler (1993) and Taylor (1998).

In a number of models, limited *local* dispersal between patches that are identical except for their initial conditions results in system-wide persistence, accompanied by spatial pattern formation (e.g. Hassell et al., 1991a; Comins et al., 1992; Rohani et al., 1994b). In these models, uncoupling immigration from local dynamics plays a role in persistence. For example in Hassell et al. (1991a) and Comins et al.'s (1992) two-dimensional arrays of identical patches with Nicholson–Bailey local dynamics, persistence and complex spatial dynamics do not occur if all of the patches are started with identical initial conditions (Adler, 1993). Differences in the initial conditions are necessary to initiate asynchrony between the patches. Asynchronous dynamics causes the relative immigration into each patch to be a decreasing function of local density, leading to bounded population dynamics in each patch.

(*c*) *A pool of dispersers, or a transit time between patches*: All of the models discussed so far have assumed that a disperser that leaves one patch immediately enters another patch. Neubert et al. (2002) found that a transit time between patches (that is, a delay between leaving one patch and entering another patch) is almost always stabilizing. Weisser and Hassell (1996) and Weisser et al. (1997) showed that entering a "pool" of dispersers before settling on a new patch stabilized the equilibrium of the local patches and the collection of patches. A pool of dispersers effectively creates another patch type with a fixed spatial difference (see above) from the patches in which the dynamics occur. In addition, a pool of host dispersers can potentially serve as a refuge from parasitism (e.g. Holt and Hassell, 1993), which can be

stabilizing. If individuals cannot reproduce while dispersing, the dispersal pool can act as a low quality patch or sink, which can also be stabilizing (Holt, 1984, 1985; Namba et al., 1999).

(*d*) *Spatial and temporal variability in parameters*: The differences between patches do not necessarily have to be permanent to cause uncoupling of immigration from local dynamics to contribute to the persistence of predator–prey systems. A number of authors have shown that the combination of temporal environmental variability that affects different areas of the environment differently, and movement between patches can enhance persistence. The key to persistence is the maintenance of asynchrony between the dynamics on the patches.

Crowley (1981) added temporal variability to the prey birth rates and predator death rates in a discrete time system with global dispersal. He found that the system was most stable with a high degree of environmental noise, low dispersal, and a large number of patches in the system. All three of these factors promoted the maintenance of asynchrony between the dynamics on different patches. Reeve (1988, 1990) and Taylor (1998) showed similar results with global dispersal between patches with Negative-Binomial local dynamics and variability in host fecundity or parasitoid attack rate. The densities in individual patches still fluctuated, but did not show the extreme diverging oscillations seen in the non-spatial version (Reeve, 1988). Environmental variability combined with a low degree of migration allowed the dynamics in the different patches in the system to remain asynchronous. However, with a high degree of migration, the patches could come into synchrony, and the collection as a whole could simultaneously go extinct. Taylor (1998) found that variability in the parasitoid conversion efficiency, a parameter that does not stabilize the non-spatial Negative-Binomial model, also increased system persistence. Hassell et al. (1991a) and Comins et al. (1992) found that adding temporal variability to host fecundity made spatial chaos and spiral waves easier to generate. Temporal variability, however, destroyed the fixed spatial patterns of their "crystal lattice" structure.

In contrast, Adler (1993) found that temporal variability in host reproduction, parasitoid attack rates, and migration rates in a two-patch system with Nicholson–Bailey local dynamics, decreased the probability of persistence. Environmental variability provided a mechanism for the two patches to become synchronized and simultaneously go extinct.

Jansen (1995) investigated briefly the effect of temporal variability without spatial variability. While keeping the parameters equivalent across patches, he varied the predator death rate through time in a two-patch model with Lotka–Volterra local dynamics. He found that this tended to bound the amplitude of the fluctuations, and keep the system from reaching

extremely low densities, compared to a non-spatial Lotka–Volterra system with the same level of noise added to the predator death rate.

5.2.3. Processes that increase coupling between immigration and local dynamics

Maintenance of spatial asynchrony is crucial for the mechanism of uncoupling of immigration from local dynamics to work. Therefore processes that synchronize the dynamics between patches can interfere with this mechanism, and therefore can be destabilizing.

(*a*) *Parasitoid aggregation*: In some cases aggregation of parasitoids to local areas of high host density can be stabilizing, but strong aggregation can also even out differences in density between patches, and this synchronizing effect can be destabilizing.

In continuous time, Murdoch et al. (1992) showed in a two-patch Lotka–Volterra system with consistent differences between the parameters in the two patches that weak aggregation by predators to the patches with higher host density tended to have a stabilizing effect, but strong levels of aggregation were destabilizing. The destabilizing effect was associated with synchronization of the dynamics in the two patches. Ives (1992) investigated a two-patch continuous-time model in which the parasitoids in each patch had a Type II functional response. Increasing aggregation was always stabilizing when host reproductive rates were different or when average host movement rates were high. However, for lower levels of host dispersal, increasing the strength of parasitoid aggregation from low to moderate levels was stabilizing, but further increases were destabilizing. Weisser and Hassell (1996) found that a host density-dependent parasitoid dispersal rate further stabilized their model containing a pool of parasitoid dispersers.

In discrete time, Reeve (1988) found that adding predator aggregation reduced the persistence time of his system of discrete-time patches because it increased the degree of synchrony between the patches. Rohani and Miramontes (1995) expanded the lattice model of Hassell et al. (1991a) and Comins et al. (1992) to include parasitoid aggregation to patches of high host density. They found that moderate levels of parasitoid aggregation promoted self-organization of spiral structures, but high levels of parasitoid aggregation inhibited spiral formation. Given that the overall population variability is low when the system persists in spiral waves, this suggests that moderate levels of parasitoid aggregation are also beneficial to the persistence of the host–parasitoid metapopulation. Rohani and Miramontes (1995) also found that multiple movement events by parasitoids within a host generation severely reduced the region of parameter space in which spiral waves occurred. With multiple movement events, the system was dominated by spatial disorder and was less likely to persist.

(*b*) *Directed host movement*: Movement of hosts from areas of high host density to low host density is potentially stabilizing, however it can also promote between-patch synchrony. Murdoch et al. (1992) looked at the effect of directed host movement, such that the immigration rate of hosts decreased as the relative host density in that patch increased, in a two-patch model with Lotka–Volterra local dynamics and different prey birth rates in the two patches. They found that strong directed host movement of this type could be destabilizing, because it caused the densities in the two patches to become more synchronized.

Krivan (1998) investigated the case with two Lotka–Volterra patches, in which the prey in one patch had a higher risk of predation, but also a higher intrinsic rate of increase. Prey moved between patches in such a way that maximized their instantaneous per-capita growth rate. He found that this optimal behavior by prey led to bounded predator–prey oscillations rather than neutrally stable cycles.

5.3. Non-linear spatial averaging

The third stabilizing mechanism appears in a relatively restricted set of models in which static self-organized spatial patterns occur. In these situations the spatially heterogeneous distribution of individuals, combined with a non-linear response to density, can cause the parameter values experienced by individuals to be different from those in a well-mixed system. In models in which a shift in the value of a parameter can cause the long-term dynamics of a system to go from limit cycles to a stable equilibrium (e.g. reducing the predator conversion efficiency in a Rosenzweig–MacArthur model), this can have a stabilizing effect (Murdoch et al., 2003).

This mechanism is well-illustrated by the spatial Rosenzweig–MacArthur model studied in the individual-based model of McCauley et al. (1996) and the patch model of de Roos et al. (1998) with limited prey dispersal and an essentially constant system-wide predator density (see also Gurney and Vetch, 2000, for a reaction–diffusion version of this model). The predator in the model has a Type II functional response, and the predator density is constant throughout the system. The combination of these two factors means that the predator will be most efficient at suppressing the prey in parts of the system that are already at low prey density. In this way, any spatial differences in the initial prey density can be amplified, with the prey driven extinct in parts of the environment, and the prey reaching high densities in other areas. The system settles into a heterogeneous state in which some patches have high prey densities and other patches are empty of prey.

The spatially constant predator density means that each predator is spending an equal amount of time in every patch, regardless of prey density. The predator's Type II functional response means that in the high prey density patches it spends a high proportion of its time handling prey, and the predator is wasting time in the low density patches. Thus, the heterogeneous distribution of prey causes the predator to be less efficient at converting prey into new predators than it would be if each patch was at the system-wide average, intermediate prey density. Reducing the predator conversion efficiency is stabilizing in the non-spatial Rosenzweig–MacArthur model, and in the spatial model it can lead to the spatially heterogeneous distribution of patches of high and low prey density becoming stabilized in space and time. The system is most stable when there is a high fraction of patches empty of prey. de Roos et al. (1998) found that the outcome depended on the initial conditions, with the system either settling on a spatially heterogeneous solution or a spatially homogeneous solution (see also Jansen, 1995, 2001).

This mechanism also appears to be acting in McCann et al.'s (2000) models aimed at explaining the spatially localized outbreaks observed in western tussock moth on lupines in California (see also Wilson et al., 1999, for more system-specific models). In a 3-patch model with local dispersal, they looked for the conditions that lead to the potential for multiple equilibria in the parasitoid–host interaction, which could result in the system in different patches settling on different within-patch equilibrium states. They found that the necessary conditions were high levels of parasitoid dispersal and low levels of host dispersal combined with either (a) a logistic host population growth rate with an Allee effect with either a linear or Type II parasitism function, or (b) a logistic host population growth rate with a Type II parasitism rate. Each of these situations introduces a non-linear response to host density that allows the host to have a relatively low per-capita growth rate at both low and high host densities.

In each of the models in this section it is assumed that the parasitoids (or predators) are acting in a suboptimal way, spending significant fractions of their time in parts of the environment with low host (or prey) density. McCann et al. (2000) found that the spatial patterning could persist with low, but not high, levels of aggregation by parasitoids to areas of high host density.

mechanisms. Spatial heterogeneity is a crucial component of all three of these mechanisms. In many of the models illustrating these mechanisms, self-organized complex spatial patterning is an outcome. This can include patterns in which regions of high or low density remain fixed in space and time in the absence of any underlying heterogeneity (e.g. Hassell et al., 1991a; Comins et al., 1992; McCauley et al., 1996; de Roos et al., 1998; Wilson et al., 1999; McCann et al., 2000). We have ignored the complementary literature that looks at whether underlying spatial heterogeneity in demographic parameters can be amplified or masked by the predator–prey interaction (McLaughlin and Roughgarden, 1991, 1992).

Another type of complex spatial pattern that can arise in the models is spiral waves, in which waves of high host density sweep through the environment followed by waves of high parasitoid density, leaving troughs of low host density in their wake (e.g. Hassell et al., 1991a; Comins et al., 1992; Rohani et al., 1994a; Rohani and Miramontes, 1995; Gurney et al., 1998; Donalson and Nisbet, 1999). In many of the lattice models, the waves can be arranged into one or more spirals emanating out from relatively spatially fixed foci. In other cases, transient spirals form and dissolve, resulting in spatial chaos (Hassell et al., 1991a; Comins et al., 1992). In this review we have stressed how these complex spatial patterns may result in persistent spatial patterns when the predator–prey interaction in an isolated patch would not persist. We have ignored the literature on spatial patterns that can occur during invasion of a novel predator into a new region of space (e.g. Sherrat et al., 1995, 1997; Gurney et al., 1998).

Seventy years ago, Nicholson (1933) proposed that otherwise unstable parasitoid–host interactions may be able to persist on a regional scale if there were limited dispersal between spatially separated local populations. The overall conclusion from this review is that the recent explosion of modeling effort in this area has, for the most part, proven Nicholson to be right. A number of models have shown repeatedly that different permutations of Nicholson's mechanism of "large numbers of independent local systems of oscillations, all phases of oscillations being represented in the environment at any given time" (Nicholson, 1933, p. 163) promote persistence.

6. Discussion

In this review we have concentrated on stabilizing mechanisms that come about when the spatial dimension and limited dispersal of individuals are added to host–parasitoid and predator–prey models. We have identified three stabilizing, or persistence promoting,

Acknowledgments

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