Given the potential importance of transients in changing our view of the explanations for virtually any dynamic phenomena in population biology, answers to the questions proposed here should be the subject of intense study.

References

1. May RM. Stability and Complexity of Model Ecosystems. Princeton: Princeton University Press, 1975.
2. May RM. Simple mathematical models with very complicated dynamics. Nature 1976; 261:459-467.
3. Hastings A, Hom C, Ellner S, et al. Chaos in ecology: Is mother nature a strange attractor? Annual Reviews of Ecology and Systematics 1993; 24:1-33.
4. Barbosa P, Schultz JC, eds. Insect Outbreaks. San Diego: Academic Press, 1987.
5. Berryman A. The theory and classification of outbreaks. In: Barbosa P, Schultz JC, eds. Insect Outbreaks. San Diego: Academic Press, 1987:3-30.
6. Hastings A, Higgins K. Persistence of transients in spatially structured ecological models. Science 1993; 263:1133-1136.
7. Lai YC, Winslow RL. Geometric properties of the chaotic saddle responsible for supertransients in spatiotemporal chaotic systems. Phys Rev Lett 1995; 74:5208-5211.
8. Ruxton GD, Doebelli M. Spatial self-organization and persistence of transients in a metapopulation model. Proc R Soc Lond B 1996; 263:1153-1158.
9. White A, Begon M, Bowers RG. Host-pathogen systems in a spatially patchy environment. Proc R Soc Lond B 1996; 263:325-332.
10. Tel T. Transient chaos. In: Hao B-L, eds. Experimental Study and Characterization of Chaos. Singapore: World Sci. 1990:149-211.
11. Kot M, Lewis MA, van den Driessche P. Dispersal data and the spread of invading organisms. Ecology 1996; 77:2027-2042.
12. Kaneko K. Spatiotemporal chaos in one- and two-dimensional coupled map lattices. Physica D 1989; 37:60-82.
13. Hastings A. What equilibrium behavior of Lotka-Volterra models does not tell us about food webs. In: Polis GA, Winemiller KO, eds. Food Webs: Integration of Patterns & Dynamics. New York: Chapman & Hall, 1996:211-217.
14. Lai YC. Persistence of supertransients of spatially chaotic dynamical systems in a noisy environment. Phys Lett A 1995; 200:418-422.
15. Allen JC, Schaffer WM, Rosko D. Chaos reduces species extinction by amplifying local population noise. Nature 1993; 364:229-232.
16. Hastings A, Powell T. Chaos in a three species food chain. Ecology 1991; 72:896-903.

CHAPTER 11

Spatial Pattern Formation in Ecological Systems: Bridging Theoretical and Empirical Approaches

Peter Turchin, John D. Reeve,
James T. Cronin and Richard T. Wilkens

Spatial Population Dynamics and Reaction-Diffusion Theory

The interplay between movement and spatial heterogeneity is of fundamental importance to population, community, and ecosystem dynamics. This is now widely appreciated by ecologists, as evidenced by high current interest in such topics as landscape ecology, metapopulation and patch dynamics, and ecological heterogeneity in general.[1-4] A central question in spatial ecological dynamics is what mechanisms are responsible for generating and maintaining spatial patterns in the distribution of organisms.[5] Formation of spatial pattern can occur as a result of both physical and biological processes.[6] Spatial patterns in ecology may form as a result of several possible mechanisms:[7] microhabitat preferences, congregation, dispersal in randomly fluctuating environments, multiple stable equilibria resulting from competitive interactions, and the interplay between biotic interactions and dispersal.

Interaction-dispersal models are well known to ecological theorists, and have been subject of much recent theoretical development, with mathematical approaches ranging from discrete cellular automata models (e.g., ref. 8) to continuum partial differential equations (e.g., refs. 9-10), or reaction-diffusion models (reaction refers to population interactions, often of the predator-prey kind, and diffusion to population redistribution of interacting species). Field evaluations of these models, by contrast, are almost unheard of[1] (but see refs. 10, 54 for a rare counter-example). This neglect is partly due to the esoteric

nature of the theory, but, more importantly, testing reaction-diffusion theory requires quantitative information about the dispersal rates of both predators and prey. Aquiring such data requires substantial investment of material, time, and mental resources. Nevertheless, we believe that it is extremely important to perform such tests, if we are to understand how spatial heterogeneity arises in ecological systems.

Pattern Formation by Diffusion–Driven Instabilities

Why is the reaction-diffusion theory relevant to empirical ecology? One of the most intriguing predictions from this theory is that an interplay between movement and species interactions can lead to formation of a spatial pattern in a homogeneous environment.[11,12] The basic idea of this "diffusion-driven instability" is due to Turing, and was first applied in an ecological context by Levin and Segel.[9] The classical mechanism for pattern formation in spatially-distributed predator-prey systems can be understood in terms of an activator–inhibitor system[12] that is characterized by the following features. First, the elevated density of prey (or the "activator" species) should have a positive effect on prey and predator population growth rate. The latter is easily satisfied (because all predators need to consume prey to survive and reproduce). The former can be satisfied if prey exhibit an Allee effect, or inverse density dependence (at low population densities, per capita rate of population change increases with prey density). Alternatively, certain kinds of predator functional response, such as the saturating functional response (type II) may yield the same effect. Second, the increased numbers of predators (the "inhibitor" species) should have a negative effect on both prey (this follows naturally from the nature of the interaction) and predator population growth rate (the latter requires some density dependence in predator population growth). Finally, predators should disperse substantially faster than the prey. Given these conditions, a spatially uniform equilibrium between prey and predators becomes unstable by the following mechanism. A small perturbation of prey density above the equilibrium leads to accelerated prey growth, and development of a prey outbreak. Predators respond by increasing their population density. In the absence of diffusion, predators will eventually bring prey density back to the equilibrium. However, if we add diffusion to the system, then predators will tend to "wander away" from the prey outbreak. This has two consequences. 1) Inside the prey patch, predator/prey ratio is lower than it would be without predator diffusion. Thus, predators become ineffectual at controlling prey, and prey density increases to the limit of prey carrying capacity. 2) In the area immediately adjacent to the prey patch, however, predator/prey ratios are high due to predators diffusing from the patch center. As a result, prey density there will be driven to a very low level. The end result is formation of patches of high prey density with very distinct borders.

The following general model of predator–prey dynamics that could exhibit spatial pattern formation is often cited (e.g., ref. 12):

$$\frac{\partial N}{\partial t} = r(N)N - f(N)P + D_1 \frac{\partial^2 N}{\partial x^2}$$

$$\frac{\partial P}{\partial t} = cf(N)P - d(P)P + D_2 \frac{\partial^2 P}{\partial x^2}$$

1)

Here $r(N)$ is the per capita rate of prey population growth (assumed to be of the Allee kind), $f(N)$ is the functional response of predators to prey density, $d(P)$ is the predator death rate (assumed to be density-dependent—since predators must have an inhibiting effect on themselves for spatial patterns to form), and D_1 and D_2 are the diffusion coefficients of prey and predators, respectively. $N=N(x,t)$ and $P=P(x,t)$ are densities of prey and predators as a function of time and space (for simplicity, we have assumed that space is one-dimensional). This model, with its relatively complex structure may look somewhat intimidating. It is little appreciated, however, that stable nonuniform spatial patterns are easily generated even in some of the simplest predator-prey models to which space is added.

As an example, let us add simple diffusion to the predator-prey model analyzed by May[13]:

$$\frac{\partial N}{\partial t} = rN\left(1 - \frac{N}{K}\right) - \frac{CNP}{N+D} + D_1 \frac{\partial^2 N}{\partial x^2}$$

$$\frac{\partial P}{\partial t} = sP\left(1 - Q\frac{P}{N}\right) + D_2 \frac{\partial^2 P}{\partial x^2}$$

2)

This model assumes logistic population growth for prey, type II functional response by predators, and logistic growth by predators, with predator carrying capacity proportional to prey density. All the conditions for pattern formation *via* diffusion-driven instability are satisfied by this simple model. The activating effect of prey on predators and the inhibiting effect of predators on prey directly follow from the nature of the predator-prey interaction. The activating effect of prey on themselves is a side-effect of the functional response of type II: as prey density declines, the predators tend to kill an increasing fraction of prey. Finally, the inhibiting effect of predators on themselves is implicit in the logistic type of predator population growth. All that is required for pattern formation is that predator diffusion rate is greater than that of prey. Indeed, numerical solutions of this model indicate that spatially nonuniform stable patterns form for a wide variety of parameter values (this result was also proven analytically for a somewhat more complicated version of Equation 2 that included temperature-dependent prey growth rate and aggregation by predators; see ref. 14). An example of the spatial pattern forming for one particular combination of parameters is shown in Fig. 11.1.

Although simple, an aspatial version of Equation 2 was shown to be a good description for a real predator-prey system: a population interaction between voles and weasels in Northern Fennoscandia.[15–18] Weasels also happen to be much better dispersers than voles. Thus, one might be tempted to conjecture that the vole-weasel system may be prone to generating stable nonuniform spatial patterns! Actually, this is not the case in regions where vole populations cycle (e.g., Northern Fennoscandia), since weasels periodically drive vole populations in such locations to very low densities; however, it still may be a possibility in more southern regions, where vole dynamics are much more stable. Nevertheless, this remains a conjecture for now, as spatial dynamics of the vole-weasel system are poorly known, and we must look elsewhere for empirical tests of pattern formation as a result of diffusive instability.

Spatial Patterns in a Bark Beetle System

One possible testing ground of the reaction-diffusion theory is the system characterized by very pronounced spatial patterning that we have studied during the last 10 years. It consists of the southern pine beetle (SPB), *Dendroctonus frontalis*, its host plant, *Pinus taeda*, and one of its major predators, clerid beetle *Thanasimus dubius*. This system also fulfills the two necessary conditions for the diffusive instability in simple reaction-diffusion models, since predators disperse at a greater spatial scale than the SPB (J. Cronin, J. Reeve, R. Wilkens, and P. Turchin, manuscript), and even more importantly, it is characterized by a striking Allee effect.

The SPB is considered to be an aggressive bark beetle, because it attacks and kills live host trees (essentially acting as a tree predator). However, pines defend themselves against insects and pathogens by exuding resin.[19,20] Thus, a single beetle or a small group of beetles cannot succeed against a healthy host. To deal with host defenses, the SPB evolved an ingenious tactic—mass attack mediated by congregation pheromones, primarily, frontalin.[21-23] (A terminological note: *congregation* is a special kind of aggregation in which conspecifics gather together; thus SPB *congregate*, while their predators *aggregate* to elevated SPB densities; see ref. 24.) When pioneer beetles begin boring into a host tree, they release the congregation pheromone, which attracts more beetles, who release even more pheromone. Thus, the dynamics of mass attack are very similar to dynamics of an autocatalytic process. If enough beetles congregate on the tree (approximately two-thousand may be needed[25]), they collectively overwhelm tree defenses. This allows them to reproduce, and also causes death of the host. On the other hand, if the local density of beetles is insufficient and mass attack does not develop, then the pioneers are trapped and killed by the crystallizing resin. The overall effect of this unique biology is a highly nonlinear relationship between local population density and population growth rate, resulting in a very pronounced Allee effect (and, therefore, conducive to diffusive instability).

As mass attack proceeds, and the tree begins to fill up with beetles, they start releasing a repelling pheromone (verbenone) that eventually inhibits late-arriving beetles from entering the tree.[26,27] If there are enough of them, late arrivals may switch the focus of mass attack to a nearby host tree, in which case the whole process of activation-inhibition is repeated. A contiguous group of host trees killed by the SPB is called a "spot infestation". Spot infestation growth initially occurs as a result of adult beetles attracted to the spot by congregation pheromones. When a new generation is produced (which in summer takes one month), some proportion of them disperses, while others contribute to further infestation growth.[28,29]

We have a number of reasons to believe that natural enemies, in particular the predatory clerid beetle *Thanasimus dubius*, play an important role in SPB dynamics, and possibly could affect the spatial pattern of spot growth. First, *T. dubius* adults are among the most abundant natural enemies of SPB, appearing on the attacked trees simultaneously with the arrival of adult SPB, and both adult and larval clerids prey on SPB.[30-32] Second, clerids appear to be a significant source of adult SPB mortality during mass-attack. This was determined in laboratory experiments where we simulated a mass attack by adding varying numbers of SPB and clerids to a caged pine bolt, with numbers chosen to create a natural range of predator and SPB densities.[33] At densities typical of
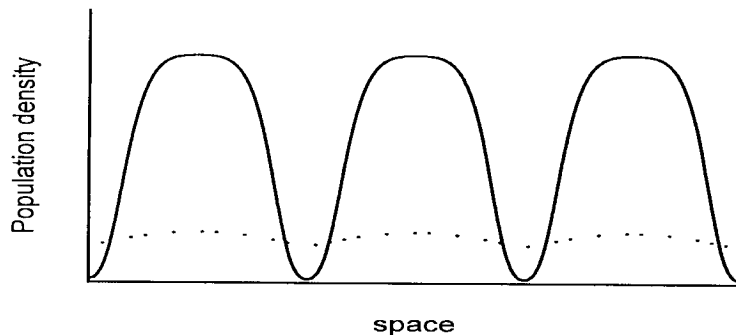
Fig. 11.1. A stable spatially-nonuniform pattern forming in a predator-prey model with diffusion (solid curve–prey density, dotted curve–predator density). The curves are a numerical solution of Eq. 2 with parameters $r=5$, $s=3$, $K=1$, $D=0.05$, $C=10$, $Q=1$, $D_1=0.001$, and $D_2=0.1$, using the method of lines as implemented in the DMOLCH routine of the IMSL Math Library. We assumed reflecting boundary conditions.

the field, clerids caused substantial mortality, up to, or even exceeding 50% (Fig. 11.2). This result suggests that predation by clerids may slow attack of the host tree, possibly even decreasing the number of SPB attackers below the threshold density, thus causing mass attack to fail.

At the spatio-temporal scale of entire National Forests and years, there is evidence that *T. dubius* responds numerically to changes in SPB density.[34] In a long-term survey of the densities of adult SPB and clerids, we observed that as SPB populations changed from endemic levels in 1989-1990 to a mild outbreak in 1993, followed by collapse in 1994, trap catches of clerid predators paralleled these changes in prey density.[33] Given that adult clerids seem to cause substantial SPB mortality, we expect this predator to help suppress SPB outbreaks.

Applying Reaction-Diffusion Theory to the SPB-Clerid Interaction

An extremely patchy pattern of SPB population distribution within relatively homogeneous forest stands suggests that biotic interactions may play an important role in determining the amplitude and the scale of spatial heterogeneity in this system (Fig. 11.3). However, the classical reaction-diffusion models are not directly applicable to our empirical system. We treat the predictions of the standard reaction-diffusion theory as metaphors, while building a specific spatial predator-prey model tailored to the pines-bark beetle-clerid predators system. Our working hypothesis for how spatial pattern arises in this system can be described as follows.
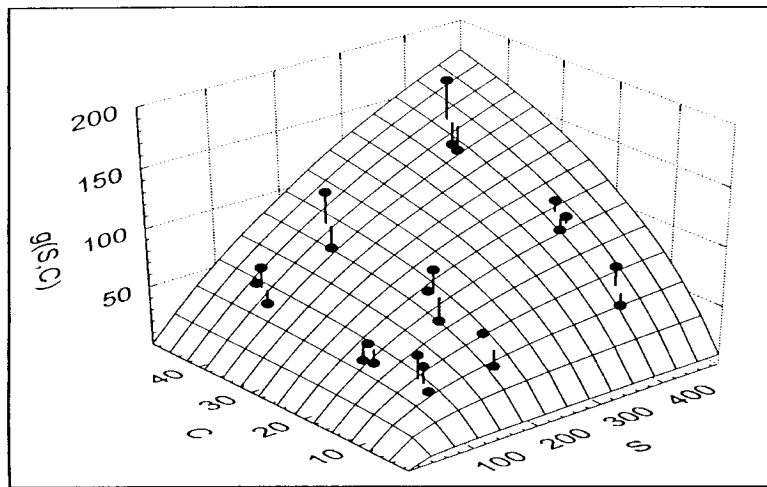
Fig. 11.2. The effect of SPB (S) and clerid (C) density on the number of SPB killed before entering experimental bolts, $g(S,C)$. The curve was fitted assuming a ratio-dependent functional response $g(S,C) = GSC/(S+DC)$.

The initial impulse for pattern formation in the SPB system is provided by congregation and a nonlinear (accelerating) per capita growth rate of prey population that results from mass attack. Congregation and mass attack by themselves should lead to formation of an aggregated pattern of SPB attack, but there are several reasons to believe that these two processes may not provide a complete explanation of SPB spatial dynamics.

In the absence of predator-mediated feedback, prey clumps should simply continue to expand until they take over all available space, and all resources are exhausted. Many SPB spots indeed stop expanding only when they approach an area of locally sparse host-tree density (e.g., a hardwood bottom), but many other spots die out, seemingly without any apparent reason.[35] On the larger spatial scale (National Forests), resource depletion cannot account for collapse of SPB outbreaks, since SPB kill at most 1-2% of available hosts, even during the worst outbreaks in recorded history.[36] This is in contrast with other aggressive bark beetles in North America. For example, outbreaks of mountain pine beetles are curtailed by severe depletion of the host population.[37] Losses of host trees exceeding 60% are not unusual,[38] with 100% mortality among the largest-diameter trees.[39] Another contrast between these two closely related species is that mountain pine beetle infestations are diffuse, with killed and surviving trees interspersed together. SPB spots, by contrast, are very compact, and typically there are no live host trees left within the spot boundaries.

The attack proceeds in a very orderly fashion, in which trees are attacked sequentially (Fig. 11.3). This pattern of expansion is especially regular on those occasions when very large SPB spots develop (e.g., Fig. 11.3 and more recent spots in Texas wildernesses, J. Reeve, personal observation). Based on these observations, coupled with general insights from the diffusion-reaction theory and specific observations of clerid impact on SPB, we have hypothesized that clerids play an important role in enhancing the degree of spatial patchiness, as well as imposing greater spatial regularity on SPB spot dynamics.

In the classical reaction-diffusion theory, reviewed above, the spatial pattern forms because predators are characterized by higher diffusion rate than prey. Predators produced within the patch rapidly diffuse out, resulting in a spatial "halo" of predator density around the prey patch. Low predator/prey ratios in the patch center allow prey to increase, while high predator/prey ratios around the patch boundaries suppress prey density, causing a stable spatial pattern to form. Our hypothesized mechanism for pattern formation in the SPB system is somewhat different:

1) The numerical dynamics of clerid populations occur at a longer temporal scale: SPB generation time is only one month in summer, while clerid generation time is about a year (actually, clerids exhibit a distributed developmental delay, with some emerging 0.5 year after mass attack, and the rest emerging after 1, 1.5, or even a 2 year diapause).[40] Thus, when considering short-term (within-season) dynamics of SPB spot formation, we can ignore predator reproduction (this does not, however, prevent accumulation of predators within older infestations due to continual immigration).

2) Clerids exhibit spatial aggregation to prey clumps, because of their behavioral response to the SPB congregation pheromone, frontalin.[41] This aggregation response produces a halo of enhanced predator density around prey patch periphery leading to an increase in the clerid/SPB ratio with distance from the spot. (Fig. 11.4).

3) Unlike static patches forming in classical models, the spatial pattern in the SPB system is highly dynamic. This happens because as SPB successfully mass-attack pine trees, these trees are removed from the population of susceptible hosts. Thus, in order to survive, a spot infestation must continuously expand, leaving "scorched earth" behind it.

4) Finally, the spatial matrix within which SPB spot growth occurrs is highly heterogeneous. Spatial variation in two variables is particularly interesting, because it strongly affects the spatial pattern of SPB distribution. First, host density is highly variable in space. This is partly due to a highly fragmented nature of the forest resulting from a variety of human activities. As importantly (if not more so), there is a considerable spatial variation in the ratio of pine trees to nonhost hardwood species. Stands with a high hardwood component are much less likely to develop a SPB spot.[42] Second, host trees vary widely in their capacity to resist SPB attack. Resistance is related to the species and the physiological status of the tree.[20,43-45] In addition, stochastic point events, such as lightning strikes,[46] may create single trees that are particularly susceptible to SPB attack. In fact, the majority of SPB spots in summer are initiated by a lightning strike.[47,48]

Given the above features of the study system, we emphasize again that we regard the classical theory of pattern formation in homogeneous space as a metaphor at best. The most interesting concept from the classical theory, which

Fig. 11.3. Outbreak dynamics in the Four Notch Area, TX. Scale: 1:30000. Extent of SPB infestation on June 8, 1983 (black), July 7, 1983 (darker gray), and July 21, 1983 (lighter gray).
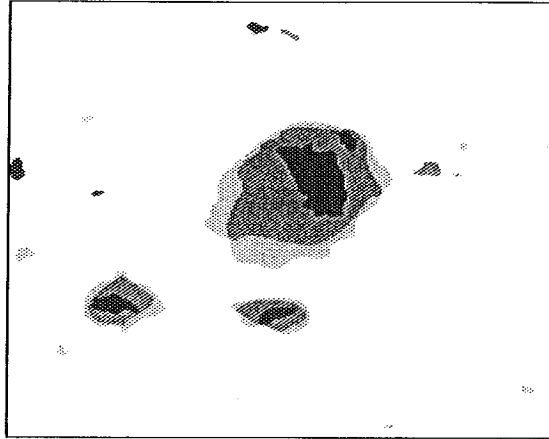
we think may be directly applicable to our system, is that predator spatial dynamics may be such that there will be a "halo" of predators arising around prey patches, and as a result, prey population growth will be inhibited just outside a prey congregation.

We will not describe here the specific model of spatial dynamics in the host tree-bark beetle-clerid predator system that we have developed, because we are not yet finished with obtaining parameter estimates for it (for the parts that have already been completed see refs. 29, 33, 49-51). More importantly, we expect that functional forms assumed by us initially (especially those dealing with clerid movement) will eventually be modified in light of additional data that we are currently collecting. However, it is of great theoretical interest to examine the spatiotemporal dynamics characterizing some greatly simplified variants of our model. In particular, what behaviors might be expected in systems with the biological features 1 and 2 listed above (predators respond to prey clumps by aggregating in space, but there is no numerical response)? A simple model encapsulating these features might be written as follows:

$$\frac{\partial N}{\partial t} = rN\left(1 - \frac{N}{K}\right) - \frac{CNP}{N+D} + D_1 \frac{\partial^2 N}{\partial x^2}$$

$$\frac{\partial P}{\partial t} = \frac{\partial^2}{\partial x^2}(\mu(N)p) \qquad\qquad 3)$$

The prey equation is the same as in model (2). The predator equation has only a redistribution term, written in the Fokker-Planck form.[24,51] Here $\mu(N)$ is the *motility*, which is assumed to be a function of local prey density, $N(x,t)$. The
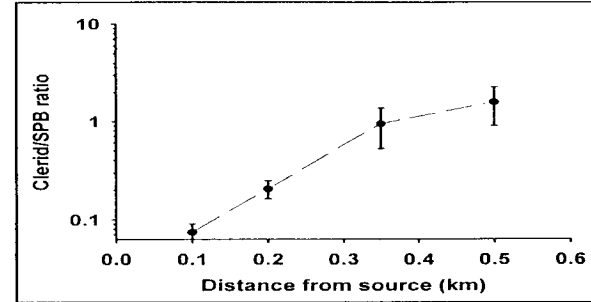
Fig. 11.4. The ratio of clerids to SPB as a function of distance from an active spot infestation.

motility is likely to be high where prey are absent, and low where prey are abundant. Assuming that motility approaches some low value asymptotically as prey density is made very high, suggests the following explicit form for it:

$$\mu(N) = \frac{D_2(N+\delta)}{\varrho N + \delta}$$

where parameter D_2 is the diffusion rate of predators in the absence of prey, ϱ is the ratio of motility in the absence of prey (D_2) to the asymptotic motility at very high prey density, and δ specifies how fast prey density saturates the predator aggregation response.

The long-term dynamics of the aspatial analog of Eq. 3 are not complicated. Depending on how many predators are present, they either drive the prey to extinction (this is a consequence of assuming type II functional response), or, if predator density is low, then the only predator effect is to slightly depress prey density below K. Numerical simulations of the Equation 3 suggest that the spatial system has the same long-term dynamics—either prey are driven to extinction everywhere, or they achieve the "outbreak" status everywhere. In either case, only spatially-uniform long-term solutions appear to be possible. We are, however, interested in the transient behavior of the system. In the short run, a variety of spatiotemporal patterns are possible. If we start with an initial clump of prey in the center—a "spot infestation"—then the most common behavior is that the initial prey patch either expands (Fig. 11.5a), and eventually occupies the whole space, or it shrinks and is eventually extinguished by predators (not shown). Contrast this behavior with what happens in the absence of predators (Fig. 11.5b). In this case, the initial patch both expands much more rapidly, and there is prey growth outside the patch that leads to a quick establishment of outbreak conditions over the whole spatial domain. Thus, predators are capable of suppressing prey outside the "spot," at the same time slowing spot expansion, or even, if the predator density is high, extinguishing the infestation altogether. Although regular infestation expansion or shrinkage are the most common behaviors, even more complex patterns are possible for some parameter values. For example, predators may aggregate so efficiently, that they successfully extinguish the initial prey infestation

Fig. 11.5. (a) Spatiotemporal dynamics predicted by model (3) with parameters $r = 5$, $K = 1$, $D = 0.05$, $C = 4$, $D_1 = 0.001$, $D_2 = 0.1$, $\delta = 0.1$, and $\varrho = 100$. Boundary conditions are reflecting. The initial distribution of predators is uniform, $P(x,0) = 0.1$, and prey have a clump in the center, $N(x,0) = 0.01 + [4x(1-x)]^{50}$; $0 < x < 1$. Prey density is shown for times $t = 1, 2, 3, 4, 5$. (b) Same as (a) except that the predator density is set to 0.

a Predators present

b Predators absent

space
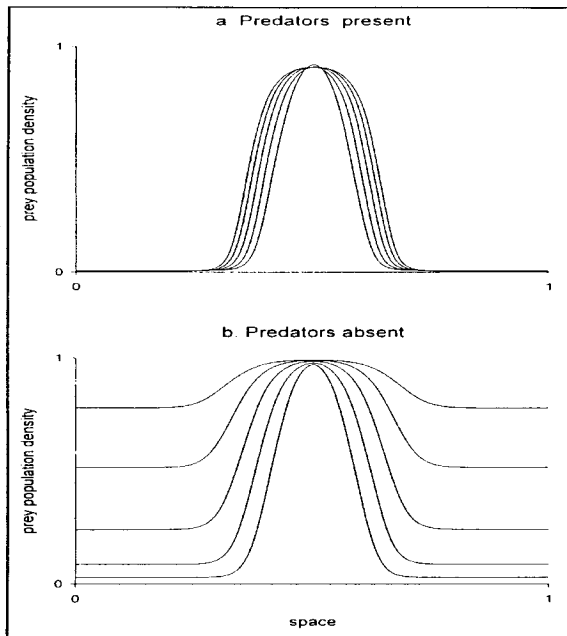
(Fig. 11.6). However, so few predators are left in the peripheral areas that prey infestations spontaneously arise there. Next, predators aggregate in the peripheral areas, and begin reducing prey infestations there. This, however, allows prey to outbreak in the center, and eventually the prey outbreak spreads over the whole space.

The implication of these results is that if nontrivial spatiotemporal patterns can so easily arise even in such a simple equation as 3, then the more complex SPB model is likely to have an even richer array of potential dynamical behaviors. It remains to be seen, however, whether the model will exhibit complex spatial dynamics for the parameters estimated in field experiments, and what will be the role of clerid predation in it.

Conclusion

The origin of patchiness in the distribution of organisms is a central question in ecology. Despite a high degree of interest in this issue, however, most current approaches are descriptive and phenomenological, while there is a dearth of mechanistic studies examining how spatial pattern in organism
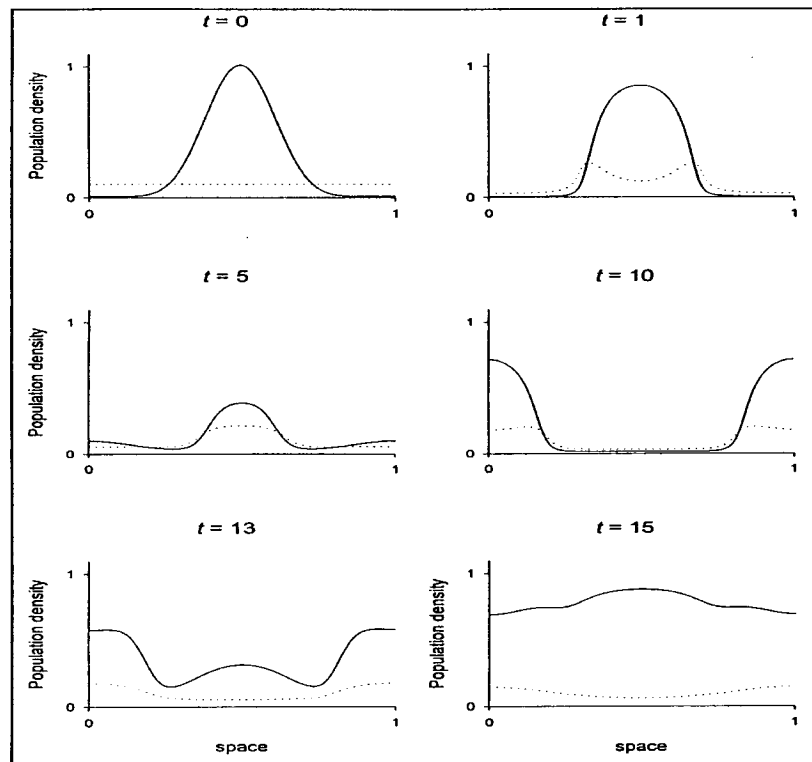
$t = 0$

$t = 1$

$t = 5$

$t = 10$

$t = 13$

$t = 15$

space   space

Fig. 11.6. Spatiotemporal dynamics predicted by Eq. 3 with parameters $r = 5$, $K = 1$, $D = 0.05$, $C = 7$, $D_1 = 0.001$, $D_2 = 0.1$, $\delta = 0.1$, and $\varrho = 100$. Boundary conditions are reflecting. The initial distribution of predators is uniform, $P(x,0) = 0.1$, and prey have a clump in the center, $N(x,0) = 0.01 + [4x(1-x)]^{10}$; $0 < x < 1$. Prey (solid line) and predator (dotted line) densities are shown for times $t = 0, 1, 5, 10, 13$, and 15.

distribution arises, and is maintained. For example, a current emphasis in landscape ecology is on description and quantification of spatial patterns, rather than on elucidating mechanisms responsible for them. Reaction-diffusion theory suggests one of the most interesting mechanisms for ecological pattern formation, yet this theoretical prediction has virtually not been tested in the field. A rare counterexample is provided by the work of Harrison and coworkers[53-55] on spatially localized persistent outbreaks of western tussock moth.

We have tried to develop two themes in this chapter. First, we have attempted to demystify reaction-diffusion theory and one of its most interesting predictions for spatial ecology. Spatial patterns in initially homogeneous space can arise when movement terms are added to most familiar predator-prey models that are a standard fare now in textbooks, such as the model of May[13] that we discussed here, and the Rosenzweig-MacArthur[56] model with an extra term for predator self-limitation.[57]

Our second theme is that results from the "classical" reaction-diffusion theory should not be taken literally, but rather as a guide to developing hypotheses and models more closely tailored to specific biological systems. The classical models for pattern formation in predator-prey systems employ simple diffusion as a description of the population redistribution process, and many field ecologists distrust such models of "random movement." Yet the basic message does not depend on specific details of models. For example, Wollkind et al[14] mathematically investigated the dynamics of a more complex model, which was developed for a temperature-dependent predator-prey mite system on fruit trees, and they showed under what conditions spatial patterns can form in that model. Aggregation of predators to prey was not part of the classical reaction-diffusion theory, yet it provides another mechanism by which a predator "halo" can arise around a prey patch (however, some forms of predator aggregation could also act as a powerful homogenizing force see ref. 12).

When discussing pattern formation by diffusive-driven instability, the focus is typically on static spatial patterns. Yet nothing in the real world is static. This means that we do not want to focus exclusively on long-term behavior of our models, but need to also consider "transient" spatiotemporal patterns. Reaction-diffusion theory predicts a rich variety of complex spatiotemporal patterns, ranging from relatively simple ones, such as invasion fronts and solitary travelling waves, to much more complex ones such as spiral waves and spatiotemporal chaos (for reviews see refs. 58, 59). Equally striking results have been obtained in discrete analogs of reaction-diffusion models—coupled-map lattices.[8] Are at least some of these complex spatiotemporal dynamical patterns found in field populations? The resolution of this question is an exciting direction in current empirical research in spatial ecology.

References

1. Turner MG, Gardner RH. Quantitative Methods in Landscape Ecology: The Analysis and Interpretation of Landscape Heterogeneity. Springer Verlag 1991b.
2. Hanski I, Gilpin ME. Metapopulation Biology. Academic Press 1991.
3. Levin SA, Powell TM, Steel JH. Patch Dynamics. Berlin:Springer-Verlag 1997.
4. Kolasa J, Pickett STA. Ecological Heterogeneity. Springer-Verlag 1991.
5. Levin SA. The problem of pattern and scale in ecology. Ecology 1992; 73:1943-1967.
6. Deutschman DH, Bradshaw GA, Childress WM, et al. Mechanisms of patch formation. In: Levin SA, Powell TM, Steel JH, eds. Patch Dynamics. Berlin:Springer-Verlag 1993:184-209.
7. Roughgarden J. Patchiness in the spatial distribution of a population caused by stochastic fluctuations in resources. Oikos 1997; 29:52-59.

8. Hassell MP, Comins HN, May RM. Spatial structure and chaos in insect population dynamics. Nature 1991; 353:255-258.
9. Levin SA, Segel LA. Hypothesis for origin of planktonic patchiness. Nature 1976; 259:659.
10. Kareiva P, Odell GM. Swarms of predators exhibit "preytaxis" if individual predators use area restricted search. Am Nat 1987; 130:233-270.
11. Kareiva P. Population dynamics in spatially complex environments: theory and data. Phil Trans R Soc Lond B 1990; 330:175-190.
12. Holmes EE, Lewis MA, Banks JE, Veit RR. Partial differential equations in ecology: spatial interactions and population dynamics. Ecology 1994; 75:17-29.
13. May RM. Stability and Complexity in Model Ecosystems. 2nd ed. Princeton:Princeton Univ Press, 1974.
14. Wolkind DJ, Collings JB, Barba MCB. Diffusive instabilities in a one-dimensional model system for a mite predator-prey interaction on fruit trees: dispersal motility and aggregative preytaxis effects. J Math Biol 1991; 29:339-362.
15. Hanski I, Hansson L, Henttonen H. Specialist predators, generalist predators, and the microtine rodent cycle. J Anim Ecol 1991; 60:353-367.
16. Hanski I, Turchin P, Korpimäki E, Henttonen H. Population oscillations of boreal rodents: regulation by mustelid predators leads to chaos. Nature 1993; 364:232-235.
17. Hanski I, Korpimäki E. Microtine rodent dynamics in northern Europe: parameterized models for the predator-prey interaction. Ecology 1995; 76:840-850.
18. Turchin P, Hanski I. An empirically-based model for the latitudinal gradient in vole population dynamics. Am Nat 1997; 149:842-874.
19. Vité JP, Wood DL. A study on the applicability of the measurement of oleoresin exudation pressure in determining susceptibility of second growth ponderosa pine to bark beetle infestation. Contrib Boyce Thomp Inst 1961; 21:67-78.
20. Hodges JD, Elam WW, Watson WF, Nebecker NE. Oleoresin characteristics and susceptibility of four southern pines to southern pine beetle (Coleoptera: Scolytidae) attacks. Can Entomol 1979; 111: 889-896.
21. Vité JP, Renwick JAA. Insect and host factors in the aggregation of the southern pine beetle. Cont Boyce Thomp Inst 1968; 24:61-63.
22. Kinzer GW, Fentiman AF, Page TF, et al. Bark beetle attractants: identification, sunthesis and field bioassay of a new compound isolated from *Dendroctonus*. Nature 1969; 221:477-478.
23. Payne TL, Coster JE, Richerson JV, et al. FIeld response of the southern pine beetle to behavioral chemicals. Env Ent 1978; 7:578-582.
24. Turchin P. Quantitative Analysis of Movement. Sunderland, MA:Sinauer Associates, 1997 in press.
25. Goyer R, Hayes J. Understanding the southern pine beetle. Forests and People. 1991; 4:10.

26. Renwick JAA, Vité JP. Bark beetle attractants: mechanism of colonization by *Dendroctonus frontalis*. Nature 1969; 224:1222-1223.
27. Renwick JAA, Vité JP. Systems of chemical communication in *Dendroctonus*. Contrib Boyce Thompson Inst 1970; 24:283-292.
28. Coulson RN. Population dynamics. In: Thatcher RC, Searcy JL, Coster J, Hertel GD eds. The Southern Pine Beetle. USDA Tech Bull 1980; 1631:71-105.
29. Cronin JT, Turchin P, Hayes J, Steiner CA. MS. Intra- and inter-infestation dispersal by the southern pine beetle. Ecology, in review.
30. Fiske WF. Notes on insect enemies of wood boring Coleoptera. Proc Entomol Soc Wash 1908; 9:23-27.
31. Thatcher RC, Pickard LS. The clerid beetle, *Thanasimus dubius*, as a predator of the southern pine beetle. J Econ Ent 1966; 59:955-957.
32. Dixon WN, Payne TL. Attraction of entomophagous and associate insects of the southern pine beetle to beetle- and host-produced volatiles. Env Ent 1979; 8:178-181.
33. Reeve JD. Predation and the dynamics of the southern pine beetle, *Dendroctonus frontalis*. Oecologia, 1997 in press.
34. Reeve JD, Ayres MP. Lorio PL. Host suitability, predation, and bark beetle population dynamics. In: Cappuccino N, Price P, eds. Population Dynamics: New Approaches and Synthesis. NY: Academic Press, 1997, in press.
35. Hedden RL, Billings RF. Southern pine beetle: Factors influencing the growth and decline of summer infestations in east Texas. For Sci 1979; 25:547-556.
36. Price TS, Doggett C, Pye JM, Holmes TP. A history of southern pine beetle outbreaks in the southeastern United States. Southern Forest Insect Working Group Report 1992:67.
37. Raffa KF. The mountain pine beetle in western North America. in Berryman AA, ed. Dynamics of Forest Insect Populations: Patterns, causes, Implications. NY:Plenum Press, 1988:506-530.
38. Harris JWE, Dawson AF, Brown RG. Evaluation of mountain pine beetle damage using aerial photography taken with a hand-held 70-mm camera, Gold Bridge-Clinton, BC, 1981. Info Rep BC-X-245.
38b. Victoria, BC, Canada: Canadian Forestry Service, Environment Canada. 1983:15.
39. Mitchell RG, Preisler H. Analysis of spatial patterns of lodgepole pine attacked by outbreak populations of the mountain pine beetle. For Sci 1992; 37:1340-1408.
40. Reeve JD, Simpson JA, Fryar JS. Extended development in *Thanasimus dubius* (F.) (Coleoptera: Cleridae), a predator of southern pine beetle. Journal of Entomological Science 1996; 31:123-131.
41. Vité JP, Williamson DL. *Thanasimus dubius*: prey perception. J Ins Physiol 1970; 16:233-239.
42. Lorio PL, Sommers RA. Central Louisiana. In: Coster JE, Searcy JL, eds. Site, Stand, and Host Characteristics of Southern Pine Beetle Infestations. Technical Bulletin USDA: Washington, DC 1981; 1612:23-29.

43. Hodges JD, Lorio PL. Carbohydrate and nitrogen fractions of the inner bark of loblolly pines under moisture stress. Can J Bot 1969; 47:1651-1657.
44. Lorio PL. Growth-differentiation balance: A basis for understanding southern pine beetle-tree interactions. For Ecol Mgt 1986; 14:259-273.
45. Lorio PL, Stephen FM, Paine TD. Environment and ontogeny modify loblolly pine response to induced acute water deficits and bark beetle attack. Forest Ecology and Management 1995; 73:97-110.
46. Hodges JD, Pickard LS. Lightning in the ecology of the southern pine beetle, *Dendroctonus frontalis* (Coleoptera: Scolytidae). Can Entomol 1971; 103:44-51.
47. Lorio PL, Yandle DO. Distribution of lightning-induced southern pine beetle infestations. So Lumberman 1978: Jan 1.
48. Coulson RN, Hennier PB, Flamm RO, et al. The role of lightning in the epidemiology of the southern pine beetle. Z Angew Entomol 1983; 96:182-193.
49. Turchin P, Thoeny WT. Quantifying dispersal of southern pine beetles with mark-recapture experiments and a diffusion model. Ecol Appl 1993; 3:187-198.
50. Turchin P, Odendaal FJ. Measuring the effective sampling area of a pheromone trap for monitoring population density of southern pine beetle (Coleoptera: Scolytidae). Environ Entomol 1996; 25:582-588.
51. Turchin P, Simmons G. Movements of animals in congregations: an Eulerian analysis of bark beetle swarming. In: Parrish J, ed. Three-Dimensional Aggregations 1997 in press.
52. Okubo A. Diffusion and ecological problems: mathematical models. Heidelberg-Berlin-New York:Springer-Verlag, 1980.
53. Harrison S. Persistent, localized outbreaks in the western tussock moth (*Orgyia vestuta*): the roles of resource quality, predation and poor dispersal. Ecological Entomology 1997 in press.
54. Brodmann PA, Wilcox CV, Harrison S. Mobile parasitoids may restrict the spatial spread of an insect outbreak. J Anim Ecol 1997; 66:65-72.
55. Wilson WG, Harrison SP, Hastings A, McCann K. Exploring stable patternformation in models of tussock moth populations. 1998 Submitted.
56. Rosenzweig ML, MacArthur RH. Graphical representation and stability conditions of predator-prey interaction. Amer Natur 1963; 97:209-223.
57. Bazykin AD, Berezovskaya FS, Denisov GA, Kuznetsov YuA. The influence of predator saturation effect and competition among predators on predator-prey system dynamics. Ecol Modelling 1981; 14:39-57.
58. Murray JD. Mathematical Biology. Springer-Verlag: Berlin 1993:2.
59. Grindrod P. Patterns and waves. The Theory and Applications of Reaction-Diffusion Equations. Oxford, UK:Clarendon Press, 1991.