0092-8240(96)00008-0

ABRUPT POPULATION CHANGES ALONG SMOOTH ENVIRONMENTAL GRADIENTS

■ WILLIAM G. WILSON* and ROGER M. NISBET
   Department of Ecology, Evolution, and Marine Biology,
   University of California at Santa Barbara,
   Santa Barbara, CA 93106, U.S.A.

■ ALEX. H. ROSS
   Marine Division,
   National Institute of Water and Atmospheric Research,
   P.O. Box 8602,
   Christchurch, New Zealand

■ CARLOS ROBLES and ROBERT A. DESHARNAIS
   Department of Biology,
   California State University at Los Angeles,
   Los Angeles, CA 90032, U.S.A.

Populations often exhibit abrupt changes in abundance associated with a smooth, continuous change in some component of their environment, with the abruptness usually attributed to inter-specific interactions or physical extremes. This paper presents a spatially explicit single-species population model in which intra-specific interactions alone are responsible for such an abrupt change. The essential mechanism involves cooperation in both colonization (through enhanced recruitment near other individuals) and mortality (protection through a "safety-in-numbers" interaction). Large fluctuations in population density would likely be observable near the transition region.

1. Introduction. Abrupt changes in abundance or species composition are common occurrences: for example, changes in the dominant species across the marine intertidal zone (Lewis, 1964; Menge, 1976; Keddy, 1985; Nielsen, 1987; Robles, 1987) or distribution of terrestrial plants with latitude and altitude (Noble, 1993; Slatyer and Noble, 1992). Many factors, including disturbance, predation, inter-specific competition and patchy recruitment (Dayton, 1971; Levin and Paine, 1974; Connell, 1975; Lubchenko and Menge, 1978; Marsh, 1986; Robles, 1987; Roughgarden *et al.*, 1988; Sato and Iwasa, 1993), influence spatial heterogeneity in sessile communities.

---

*Present address: Department of Zoology, Duke University, Durham, NC 27708, U.S.A.

Discontinuities in population density often correlate to abiotic boundaries, such as the correlation between ocean currents along the Western North American coast and the distribution of marine invertebrates (Efford, 1970). However, abrupt changes may also occur when organisms are distributed along *smooth* environmental gradients, such as the variation of temperature with altitude and latitude, atmospheric exposure time from the low tide line to the high tide line and toxicant concentration with distance from a point source.

A key feature in a sedentary organism's localized environment is the distribution of surrounding con-specific individuals, which admits the possibility of cooperative interactions. For example, a high population density increases the survivorship of the ribbed mussel Geukensia dimissa over the winter (Bertness and Grosholz 1985). A number of mathematical models regarding well-mixed, spatially homogenous populations incorporating intra-specific, cooperative interactions (e.g. Noy-Meir, 1975; May, 1977) exist in the literature: the spruce budworm model (Ludwig *et al.*, 1978) is a particularly well-known example. Certain parameter combinations in these models have two locally stable equilibria, and the long-term dynamics then depend on initial conditions. These models generally deal with mobile organisms, and mathematical derivations show that the consequence of diffusive movement is that for any given set of parameter values only one equilibrium is stable against large perturbations (Murray, 1989 and references therein). The proof is biologically instructive (see section 4). For example, think about a small patch of prairie in the midst of a forest, or vice versa, where both prairie and forest represent equilibrium states and diffusion represents seed dispersal. The proof considers movement of the interface separating these regions. There is a transition line (or hypersurface) in the system's parameter space where the interface switches from advancing into one spatial region (prairie overtaking forest) to advancing into the other spatial region (forest invading prairie). An environmental spatial gradient induces variation in rates of migration, reproduction and mortality as individuals respond to their local environment. Gradients that cut across the transition line would produce an abrupt change in density (a sharp tree line). Thus instead of multiple equilibria, the system would exhibit properties that resemble phase transitions (e.g. solid to liquid, ordered to disordered) in physical or chemical systems.

This paper shows that local cooperative interactions may lead to spatial density discontinuities in a single-species model of sedentary organisms. Our simplified general model was motivated by the population dynamics of marine mussels, yet the model is readily applicable to other biological systems. Mathematically, the model falls within the family of stochastic cellular automata (Wolfram, 1983), resembles "contact models" used exten-

sively in epidemiology and population genetics (Durrett, 1988) and shares features and the philosophy of the Ising model (Huang, 1987) in physics and chemistry. The latter is a mathematically accessible caricature of a phase transition that has helped identify common mechanisms in very different systems (ferromagnets, liquid–gas systems and binary alloys) by reinterpreting the Ising model's fundamental variables.

Section 2 introduces the model interactions as applied to a system of marine mussels. Section 3 presents a zero-correlation, or mean-field, model representing these interactions and demonstrates that this non-spatial approximation exhibits multiple equilibria. Section 4 considers spatial processes, comparing approximate zero-correlation results with measurements from a spatially explicit simulation. Results of the two approaches agree qualitatively, but this section discusses several factors that account for quantitative differences. Section 5 illustrates that a smooth, environmental spatial gradient produces an abrupt transition in population density.

2. Model Interactions. The model has general applicability to marine, aquatic or terrestrial systems of sedentary organisms, but this paper presents it for mussel dynamics to avoid unnecessary abstraction and cumbersome prose. The hypothetical mussels live in an arena represented by a two-dimensional grid of "cells," each cell being approximately the size of one large adult mussel. A state variable denoting *cell occupancy* characterizes each cell, having value 1 if at least one mussel (of any age or size) is present, and zero otherwise. The model is stochastic, with four interactions determining the probability per unit time that any given site's occupancy changes its state (see Fig. 1):

Open Recruitment. Local populations increase due to settlement of larvae from widely dispersed sources. Larval recruitment dynamics operate independently from local population dynamics, producing a constant influx of recruits. Open recruitment is a single-cell interaction: If a cell is unoccupied, it becomes occupied during a time interval Δt with probability $\beta \Delta t$, independent of its neighboring cells' states. Because cell occupancy is the state variable, recruitment to an already occupied cell has no effect in the model.

Patch Mortality. Local populations decrease when an occupied cell becomes unoccupied during a time interval Δt with probability $\delta \Delta t$. One mechanism leading to such a uniform removal is, for example, hydrodynamic lift (Denny, 1987; Denny *et al.*, 1985; Paine and Levin, 1981).

Figure 1. The left-hand side shows the rules for the mussel model. Cell states are 1 if occupied and 0 if empty. The rules, from top to bottom, represent *open recruitment, patch mortality, crowding and safety-in-numbers*. The right-hand side lists the contribution each process makes to the "zero-correlation" approximation of equation (1), where n is a cell's occupancy probability.

Crowding. Much successful settlement occurs in the shelter of established individuals, yet open recruitment only influences unoccupied cells. Recruits in occupied sites experience strong intra-specific competition as they grow. Near the edge of mussel clusters individuals can escape this competition by moving into unoccupied space and setting down bysal threads. The resulting cluster growth is incorporated in the model by assuming that an empty cell is colonized by a given occupied nearest neighbor cell during a time interval Δt with probability $\frac{1}{4}\chi \Delta t$.

Safety-in-Numbers. The most complex interaction represents mortality due to drag forces induced on individual mussels by the repeated actions of waves rolling into mussel beds (Denny, 1987; Denny *et al.*, 1985). Mussels on a cluster's edge, having less protection, experience higher drag forces than those located in the center. The model incorporates this feature by an occupied cell becoming empty during Δt with probability $\sigma \Delta t$ if neighbored by three or four empty cells. A mussel neighbored by two or more mussels is safe from this mortality, hence the term "safety-in-numbers." This could also represent edge-specific predation.

In spite of the model's conceptual simplicity, analytic methods cannot deduce its exact dynamics. Thus, this paper examines two approaches. The first is a straightforward computer simulation (see Appendix) incorporating spatial arenas with tens or hundreds of thousands of cells. If the "correlation length" (the effective range of correlated cell dynamics) is small

relative to the arena's size, simulations provide reliable estimates of exact dynamics of very large systems (e.g. Wilson and Vause, 1989). The second approach—the simplest analytic backdrop for simulation results—is a *zero-correlation* (Gurney and Nisbet, 1978a, b) or mean-field (Huang, 1987) *approximation*, which neglects spatial correlations in cell occupancies. The paper then investigates how spatial interactions modify the zero-correlation results.

3. Zero-Correlation Analysis. An exact analytic analysis accounts for stochasticity and variability at the scale of individual cells. The simpler zero-correlation approximation assumes there is no variability and that lattice cells are statistically identical and independent. Although this is a gross simplification, its attractive feature is its analytical approachability. The general prescription for deriving the zero-correlation model for the dynamics of a chosen cell state is to sum over all possible configurations the product of each configuration's probability and its transition probability to the chosen cell state. The analysis performed here uses "occupied" as the chosen cell state and defines $n(t)$ as the occupancy probability at time t, equal to the fraction of occupied sites in a large system. The result is a first order differential equation with a contribution from each interaction described in the previous section (Fig. 1). Hence the contribution due to open recruitment is the probability of an unoccupied cell $(1 - n)$ multiplied by the transition rate β. Similar reasoning yields the term for patch mortality. The crowding term is the probability of two neighbor sites being in opposite states $n(1 - n)$ multiplied by the transition rate $(\frac{1}{4})\chi$, summed over the four nearest neighbors, yielding $\chi n(1 - n)$. The safety-in-numbers interaction is the sum of the probability of an occupied site being surrounded by four empty sites $n(1 - n)^4$ and the probability of having just one neighbor (with four possible locations for the occupied cell) $n(4n)(1 - n)^3$, multiplied by the transition rate σ, yielding $\sigma n(1 - n)^3(3n + 1)$. These terms, with the appropriate signs, give the rate of change for the occupancy probability:

$$\frac{dn}{dt} = \beta(1 - n) - \delta n + \chi n(1 - n) - \sigma n(1 - n)^3(3n + 1). \qquad (1)$$

The dynamical system defined by Equation (1) has equilibrium properties similar to those of many physical and biological systems possessing phase transitions (e.g. Schlögl, 1972; Haken, 1983; Huang, 1987; Murray, 1989). Figure 2a illustrates these properties, showing that for some parameter values, Equation (1) has three positive equilibria, two of which, n_L and n_H, are locally stable to small perturbations. The middle equilibrium, n_U, is locally unstable. However, the right-hand side of Equation (1) has three
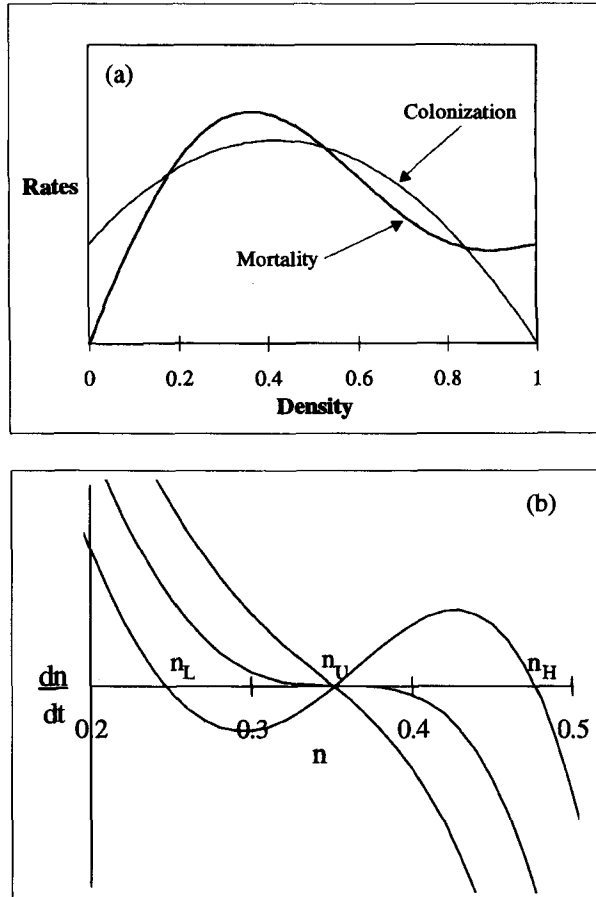
Figure 2. (a) The zero-correlation colonization (light line) and mortality (heavy line) terms of equation (1) demonstrate the possibility of multiple equilibria (the intersections of the colonization and mortality curves). (b) The total population rate of change takes three basic shapes along a line through parameter space with constant equilibrium density n_U. The middle curve marks a "critical point" on the surface that separates this model's parameter space into a region with one stable equilibrium and a region with two stable equilibria.

general forms, shown in Fig. 2b. In one region of parameter space, the single equilibrium n_U is stable. In the remaining region, corresponding to the colonization and mortality curves shown in Fig. 2a, there are the three equilibrium points discussed above, with the intermediate equilibrium n_U being unstable. Systems with initial densities less than n_U evolve to the lower equilibrium n_L and, likewise, systems with initial densities greater than n_U evolve to the upper equilibrium n_H, implying that initial conditions determine the long-term population densities. The combination of

parameter values used to generate the middle curve in Fig. 2b marks a "critical point" in parameter space (Huang, 1987).

4. Implications of Space. Section 1 noted that previous studies have established that asymptotic dynamics of single-species models with multiple equilibria *and* diffusion are independent of initial conditions (Murray, 1989). Simple diffusion added onto a zero-correlation model yields a partial differential equation of the form

$$\frac{\partial n}{\partial t} = g(n) + D\frac{\partial^2 n}{\partial x^2}, \tag{2}$$

where $n(x, t)$ is the occupancy probability at location x and time t, and D is the diffusion coefficient. The function $g(n)$ describes local birth and death processes having the properties shown in Fig. 2b. Murray's (1989) proof continues with the assumption of an initial configuration in which the origin $x = 0$ represents a boundary separating high- and low-density equilibrium regions. The question is then whether this boundary moves in the positive direction, negative direction or stays motionless at the origin. The direction of boundary movement determines the dominant phase and, after some analysis, gives the condition

$$\int_{n_L}^{n_H} g(n)\,dn \begin{cases} > 0, & \text{high-density phase invades low density phase,} \\ = 0, & \text{motionless boundary,} \\ < 0, & \text{low-density phase invades high density phase.} \end{cases} \tag{3}$$

Figure 2b represents the integral (3) as the sum of the "negative" area (the integral from n_L to n_U) and the "positive" area (the integral from n_U to n_H). The point along a line in parameter space where these two areas are equal marks a transition between the phases n_L and n_H. This equal-area construction has been in use for over a century in phase transition models of physical systems (see Klein, 1974, who cites Maxwell, 1875, for the first derivation).

Figure 3 compares results from the zero-correlation model and the spatial simulation. The zero-correlation plot (Fig. 3a) only shows the dominant equilibrium, chosen by the equal-area construction, in regions of multiple equilibria. Figure 3b presents equilibrium densities measured from a spatially explicit simulation (see Appendix). Both cases fix the recruitment rate β and patch mortality rate δ while varying the crowding rate χ and safety-in-numbers rate σ. Predictions from the zero-correlation approximation and equal-area construction provide a qualitative match to the simulation results. Clearly there is significant quantitative disagreement,
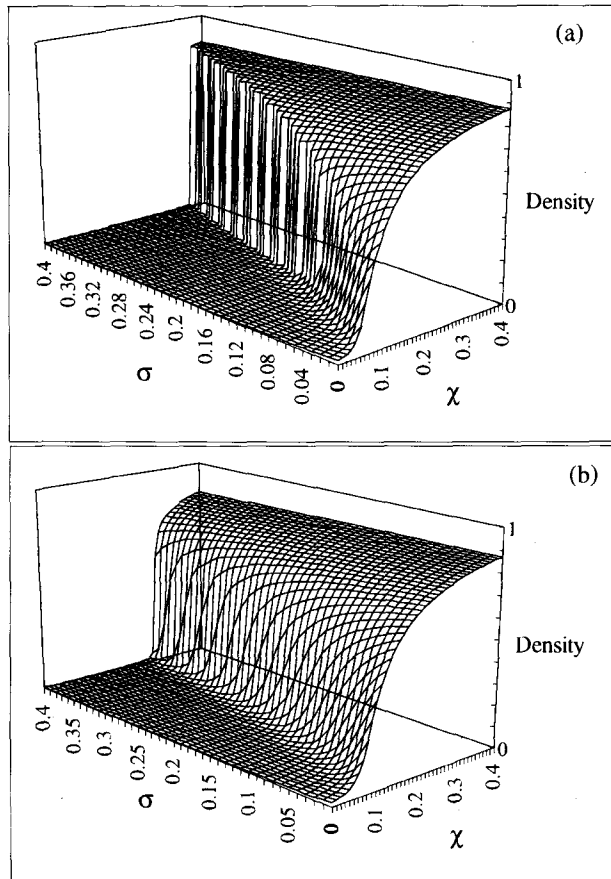
Figure 3. (a) Equilibrium mussel density n determined from equation (1) and the equal-area construction (3) as a function of crowding rate χ and drag rate σ with recruitment rate $\beta = 0.001$ and patch mortality rate $\delta = 0.05$. (b) Results from a spatial simulation using a 128^2 lattice. Zero-correlation and simulation results agree qualitatively, in spite of clear quantitative differences.

particularly in the sharpness and location of the boundary between low and high population densities and in the precise location of the critical point, that stepping beyond a mean-field approximation to a pair approximation would partly alleviate (Matsuda *et al.* 1992; Harada and Iwasa, 1994). However, several issues remain when accounting for this quantitative mismatch: two surround assumptions regarding derivation of the equal-area construction, and others involve general properties of phase transitions. The remainder of this section addresses each in turn. The quantitative effect due to each of these remains unclear.

Local Mixing.   Propagation of a phase boundary requires a degree of local mixing or interactions that allow neighboring regions in different equilibrium phases to affect each other. Murray's equal-area construction uses simple density-independent diffusion to provide local mixing, but the simulation model of *sessile* organisms has no explicit diffusion, relying instead on interaction terms, with cell transitions dependent on neighboring cell states, to propagate the phase boundary. Non-linear effects due to the safety-in-numbers interaction further complicate applicability of analytic results assuming simple diffusion. This interaction involves many neighbors, and the analytic counterpart is a density-dependent diffusion term along with the simple diffusion used in Murray's analysis. Such an addition might just replace the simple equal-area construction (3) with a more complicated condition yet retain the important feature that one phase or the other dominates at each point in parameter space. If this argument is correct, the non-linearity present in the simulations would produce a phase transition boundary at a location different from that predicted by the equal-area construction.

Metastability.   Applicability of the equal-area construction also relies on a subtler assumption. Consider a system of mussels represented by the mussel model with parameter values having a dominant low-density equilibrium. Open recruitment ensures that there will be a low mussel density everywhere in a uniform habitat. Now imagine slowly changing a parameter just enough to cross the discontinuous phase transition line such that the high-density equilibrium becomes dominant. The entire system remains in the low-density phase while crossing the line; hence the system is now metastable. Before the dominant phase takes hold, a "small" region of the high-density phase must appear spontaneously. The macroscopic times required for the appearance of a spontaneous, large perturbation leads to many interesting features in phase transitions, such as hysteresis, or super-heating and super-cooling. The equal-area condition (3) determines the dominant phase by calculating which way the boundary moves and makes no explicit predictions about these times. Simulation results, however, show that a single mussel occasionally becomes a "keystone individual," representing a very localized region of high-density phase, that begets the high-density equilibrium. This, coupled with open recruitment, infers that adequate local perturbations are always present in the system, and the most important question involves which phase dominates.

Divergent Properties.   Finally, we have observed, but not presented, that the mussel model's critical point exhibits many of the same features

observed near critical points of common physical phase transition models. Particularly, both the relaxation time (the time needed to achieve steady state) and the correlation length (the distance over which local perturbations have an influence) diverge at the critical point (Huang, 1987; Wilson and Vause, 1989). The implications for simulation studies are two-fold. First, long relaxation times require long transient periods, and thus more computing effort, before making measurements of steady-state properties. Second, long correlation lengths require large lattices: Near the critical point a "small" lattice's edges influence the system and the simulation no longer approximates an infinite system. In these situations simulation results are unreliable. The issue is not one of quantitative agreement between mean-field and simulation results, rather recognition that both are poor approximations of an infinite system's true steady-state solution. Generally, if steady-state results are the goal, then critical points present a problem, but if transient phenomena are also interesting (Hastings and Higgins, 1994), then critical points in biological systems could be fascinating.

5. Dynamics along a Smooth Environmental Gradient. Smooth spatial variation of some environmental feature may produce a proportionate variation in one or more of the biological interactions represented by the model parameters. For example, a gradient in wave action would mean a parameter gradient in the "safety-in-numbers" term. Figure 3 suggests that sharp changes in population density should be anticipated along a smooth environmental gradient that crosses the phase transition. The simulation model demonstrates this phenomenon when the parameter σ increases linearly with position (Fig. 4). The transition in mean patch occupancy is smooth away from the discontinuity (Fig. 4a), but is rather sharp when the discontinuity is crossed (Fig. 4b). Similar results can be obtained with other parameter gradients, such as the crowding rate χ, which might arise in the vicinity of a point source of a toxicant.

6. Discussion. The main conclusion from this paper is that abrupt changes in density of sedentary organisms may be a response to slow temporal or spatial variation in *intra-specific* interactions alone. The key model features leading to this conclusion are cooperative interactions influencing both recruitment and loss rates. While many abrupt changes in densities result from inter-specific interactions and/or spatial heterogeneity, for example, a simple change in predator density (Hughes, 1994) or physiological constraints (Paine and Levin, 1981), results presented here establish that intra-specific cooperative interactions produce similar effects. This observation may lead to a resolution in cases where no external causes for abrupt
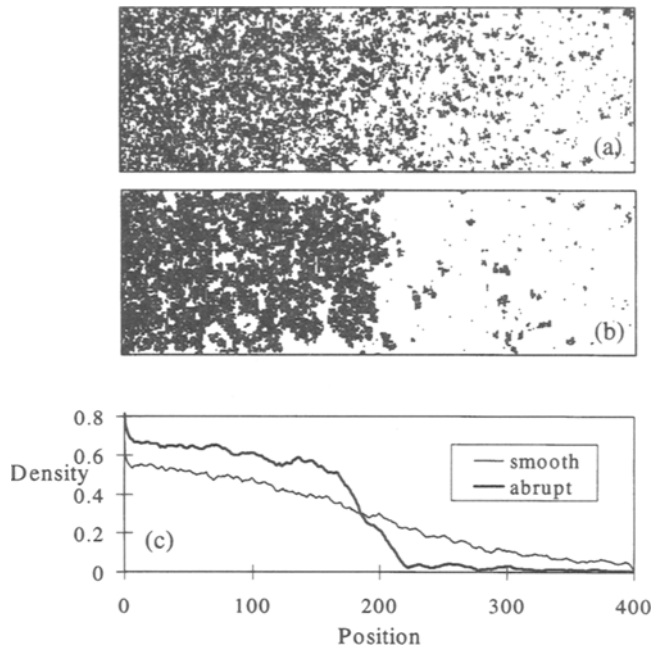
Figure 4. (a), (b) Spatial simulation snapshots of a 400 × 128 lattice. A black dot signifies occupancy. Both images use $\beta = 0.001$ and $\delta = 0.05$. (a) $\chi = 0.16$ and uniform wave intensity gradient with $\sigma = 0.05$ on the left edge and $\sigma = 0.15$ on the right edge. The population density varies smoothly. (b) $\chi = 0.3$ and uniform wave intensity gradient with $\sigma = 0.44$ on the left edge and $\sigma = 0.54$ on the right edge. The population density changes abruptly in the middle of the image. (c) Density profiles along the gradient reflect these variations.

density changes are apparent. On the transition boundary, but well away from the critical point, the system should spend most of its time in the dominant low- or high-density phase, but the occasional environmental fluctuation could take the system into the other phase for transient times (e.g. Nisbet and Onyiah, 1994). Long response times and patchiness on all spatial scales would characterize systems situated near the critical point in parameter space (Fig. 2).

There are many other causes of patchiness unrelated to cooperative interactions. Previous studies of other spatially explicit models demonstrate a correlation length ξ: Below this scale, populations behave much like a homogenous population, but above this scale, population fluctuations are uncorrelated (e.g. de Roos *et al.*, 1991; McCauley *et al.*, 1993). It is reasonable to interpret ξ, set by model interactions, as the characteristic patch size for such systems, but as yet there is no recipe for estimating its magnitude in any real system. While the mussel model exhibits "large patches" and "long transients" near the critical point, definitions of "large"

or "long" depend on system-specific details and are notoriously difficult to obtain quantitatively. There is also no prescription to distinguish critical fluctuations from correlated phenomena found in models with simpler dynamics.

Many different sets of biological assumptions lead to multiple equilibria models similar to the foregoing model (Noy-Meir, 1975; May, 1977; Harada and Iwasa 1994). Indeed, an equivalent dynamical model having a different biological interpretation arises from a quirk of occupancy models: The dynamics of occupied cells reflect the dynamics of *unoccupied* cells. The equilibrium n_L in Fig. 2b represents both a low density of occupied cells and a high density of unoccupied cells. In this spirit, the simulation pictures in Fig. 4 could instead show unoccupied cells as dark pixels, transforming the white areas into dark areas and vice versa. Defining a variable $m = 1 - n$, one avoids imagining the dynamics of nothing by letting $m = 1$ represent the presence of a "hole" (Wilson and Laidlaw, 1992). The newly transformed dark areas represent regions of high hole density ($m = 1$). A simple transformation of the density axes in Figs. 2–4 also provides the quantitative dynamics of the variable m. If the holes represent a new organism (and empty holes contain nothing), what would be that organism's biological features? The zero-correlation model for hole dynamics arises from equation (1) upon substitution of $n = 1 - m$, and this new model's interaction terms describe the biological features. The resulting dynamical model is

$$\frac{dm}{dt} = -\beta'm + \delta'(1 - m) - \chi'm(1 - m) + \sigma'(1 - m)m^3(4 - 3m), \quad (4)$$

with primes attached to this new model's parameters. The important point is that model (4) is mathematically equivalent to model (1), but describes a different biological scenario. The first two terms represent patch mortality and open recruitment, as in the mussel model (1), but with the two parameters' roles reversed. The third term represents a mortality function in which organisms risk death for each empty neighbor site. This is somewhat similar to the safety-in-numbers idea, except here the "safety" is directly proportional to the number of neighbors present. The last term represents cooperative recruitment to empty sites surrounded by three or four occupied neighbor sites. Such a term could represent the protection of offspring by adults in high-density regions. Hence, occupancy models both explore the implications of specific biological processes and provide connections between biological systems having distinct processes.

These results also have implications concerning the role of competition in determining community structure, since, in natural communities, interspecific competition is likely to occur alongside intra-specific interactions.

Much insight, particularly on competitive hierarchies and species zonation, has come through experimental manipulations involving controlled exclusion of other factors such as predation (e.g. Dayton, 1971; Lubchenko and Menge, 1978; Robles, 1994). If enhanced cooperation reduces predation, this paper demonstrates that population changes in such manipulations may be indirectly related to intra-specific cooperative interactions. The presence of vulnerable prey at particular locations within a community may not indicate the absence of predator foraging, but rather the effectiveness of cooperation at those locations. Also, competitors might serve as unwitting cooperators, for example, when young mussels indiscriminately recruit to surface irregularities, perhaps the roots of macroalgae, the bysal threads on an existing mussel patch or substrate heterogeneities, seeking protection from predators or hydrodynamic drag. All these aspects indicate that sharp transitions in dominant species along environmental gradients may result from subtly combined effects of inter-specific competition and intra-specific cooperation (Wilson and Nisbet, 1996).

Predictive modeling may target the location of discontinuities on environmental gradients as a particularly sensitive indicator of environmental change. The preceding work began after finding an unexpectedly high sensitivity to environmental change in a very detailed model of mussel population dynamics (Wilson, Nisbet and Ross, unpublished research), and suggests that a high priority for future research should be relating phase transition behavior to interactions among individuals. Useful predictions regarding environmental change in specific systems may arise by linking spatial features to individual-based population models in toxicology (e.g. Kooijman, 1993 and references therein; Nisbet et al., 1995; Kooijman and Bedeaux, 1995).

APPENDIX

This appendix provides details regarding the simulation used in Figs. 3b and 4. The simulation uses discrete time steps and discrete cells to approximate continuous time and space. Each cell contains an occupancy variable having states 0 or 1, denoting unoccupied or occupied. respectively. Figure 1 depicts the rules defining the transitions between cell states that occur during a time step. Each rule displays the initial configuration on the left, the final configuration on the right, and the transition probability above the arrow.

The arrangement of cells is an $L \times M$ square lattice partitioned, like a checkerboard, into two sublattices A and B. Cell transitions are either site-specific or dependent on nearest neighbor cells' states (see section 2) which allows simultaneous updating of all A cells during a time step, and subsequently all B cells, without introducing spurious update order effects.

The C code representing a cell state update precisely defines the simulation rules. Letting "origin" be a pointer to a lattice site, "state" be its occupancy variable and "nabes" be an array of pointers to origin's neighbor cells, the cell updating code is a single C statement:

```
if(origin → state = = 0)
{
    if(drand48( ) < beta) origin → state = 1;              /* uniform recruitment */
    else if(drand48( ) < chi)                              /* crowding */
    {
        dir = (int)4*drand48( );
        origin → state = origin → nabes[dir] → state;
    }
}
else
{
    if(drand48( ) > delta) origin → state = 0;             /* patch mortality */
    else                                                  /* safety-in-numbers */
    {
        nncnt = 0;
        for(j = 0; j < 4; j++ ) if(origin → nabes[j] → state = = 1) nncnt++ ;
        if(nncnt <= 1 && drand48( ) < sigma) origin → state = 0;
    }
}
```

where "drand48( )" is a system supplied random number generator. The time step defines unit time $\Delta t = 1$, so does not enter explicitly. The values of β and δ are small enough so that interaction ordering within a time step poses no problems. Although χ and σ may be relatively large, their interactions occur for mutually exclusive cell states, again avoiding updating problems. There are two coding options for crowding events to match the zero-correlation model: (1) Examine all four neighbors, each "crowding" with probability $\chi/4$, or (2) examine one randomly chosen neighbor crowding with probability χ. The latter case examines each neighbor, on average, every fourth time step. Neither option exactly matches the zero-correlation model since both avoid multiple crowding events in which two or more neighbors crowd into the same cell. The above code uses the latter option to reduce random number generation.

In Fig. 3b, the lattice used periodic boundary conditions where cells on the left edge neighbor cells on the right edge, and similarly for top and bottom edges. Simulation results were insensitive to initial conditions (initial cell occupancy probability). Determination of the surface of Fig. 3b used two sets of simulation runs. An initial examination measured each mussel density over 1000 time steps after discarding the first 1500 time steps to allow relaxation of transients. Transients decayed very slowly in the region of parameter space surrounding the phase transition line, so longer runs in this region used 20,000 relaxation steps and 5000 measurement steps. Results at the boundaries between the short and long runs agree.

In Fig. 4, the lattice used boundary conditions where cells on the left edge connected to a fully occupied boundary and cells on the right, top and bottom edges connected to empty boundaries. These boundaries mimic low and high wave intensities on the left and right edges, respectively, and physiological constraints of the intertidal zone at the top and bottom edges. Simulations began with each cell having an 80% occupancy probability. Measurements in Fig. 4c used 20,000 relaxation steps and 5000 measurement steps.

REFERENCES

Bertness, M. D. and E. Grosholz. 1985. Population dynamics of the ribbed mussel, *Geukensia demissa*: the costs and benefits of an aggregated distribution. *Oecologia* **67**, 192–204.

Connell, J. H. 1975. Some mechanisms producing structure in natural communities: a model and evidence from field experiments. In *Ecology and Evolution of Communities*, M. L. Cody and J. Diamond (Eds), pp. 460–490. Cambridge MA: Belknap Press.

Dayton, P. K. 1971. Competition, disturbance, and community organisation: the provision and subsequent utilisation of space in a rocky intertidal community. *Ecol. Monographs* **41**, 351–389.

Denny, M. W. 1987. Lift as a mechanism of patch initiation in mussel beds. *Exp. Marine Biol. Ecol.* **113**, 231–245.

Denny, M. W., T. L. Daniel, and M. A. R. Koehl. 1985. Mechanical limits to size in wave-swept organisms. *Ecol. Monographs* **55**, 69–102.

de Roos, A. M., E. McCauley, and W. G. Wilson. 1991. Mobility versus density-limited predator–prey dynamics on different spatial scales. *Proc. Roy. Soc. London Ser. B* **246**, 117–122.

Durrett, R. 1988. *Lecture Notes on Particle Systems and Percolation.* Pacific Grove, CA: Wadsworth & Brooks/Cole Advanced Book & Software.

Efford, I. E. 1970. Recruitment to sedentary marine populations as exemplified by the sand crab, *Emerita analoga* (Decapoda, Hippidae). *Crustaceana* **18**, 293–308.

Gurney, W. S. C. and R. M. Nisbet. 1978a. Predator–prey fluctuations in patchy environments. *Amer. Naturalist* **112**, 1075–1090.

Gurney, W. S. C. and R. M. Nisbet. 1978b. Single species population fluctuations in patchy environments. *J. Animal Ecol.* **47**, 85–102.

Haken, H. 1983. *Synergetics: An Introduction. Nonequilibrium Phase Transitions and Self-Organization in Physics, Chemistry, and Biology*, 3rd rev. ed. Berlin: Springer-Verlag.

Harada, Y. and Y. Iwasa. 1994. Lattice population dynamics for plants with dispersing seeds and vegetative propagation. *Res. Population Ecol.* **36**, 237–249.

Hastings, A. and K. Higgins. 1994. Persistence of transients in spatially structured ecological models. *Science* **263**, 1133–1136.

Huang, K. 1987. *Statistical Mechanics.* New York: Wiley.

Hughes, T. P. 1994. Catastrophes, phase shifts, and large-scale degradation of a Caribbean coral reef. *Science* **265**, 1547–1551.

Keddy, P. A. 1985. Wave disturbances in lakeshores and the within-lake distribution of Ontario's Atlantic coastal plain flora. *Canadian J. Botany* **63**, 656–660.

Klein, M. J. 1974. Historical origins of the van der Waals equation. In *Van der Waals Centennial Conference on Statistical Mechanics*, C. Prins (Ed). Amsterdam: North-Holland.

Kooijman, S. A. L. M. 1993. *Dynamic Energy Budgets in Biological Systems: Theory and Applications in Ecotoxicology.* Cambridge: Cambridge University Press.

Kooijman, S. A. L. M. and J. J. M. Bedaux. 1995. Analysis of toxicity tests on *Daphnia* survival and reproduction. Preprint.

Levin, S. A. and R. T. Paine. 1974. Disturbance, patch formation and community structure. *Proc. Natl. Acad. Sci. U.S.A.* **71**, 2744–2747.

Lewis, J. R. 1964. *The Ecology of Rocky Shores.* London: English Universities Press.

Lubchencko, J. and B. A. Menge. 1978. Community development and persistence in a low rocky intertidal community. *Ecol. Monographs* **59**, 67–94.

Ludwig, D., D. D. Jones, and C. S. Holling. 1978. Qualitative analysis of insect outbreak systems: the spruce budworm and forest. *J. Animal Ecol.* **47**, 315–332.

Marsh, C. P. 1986. Rocky intertidal community organisation: the impact of avian predators on mussel recruitment. *Ecology* **67**, 771–786.

Matsuda, H., N. Ogita, A. Sasaki, and K. Sato. 1992. Statistical mechanics of population. *Prog. Theor. Phys.* **88**, 1035–1049.

Maxwell, J. C. 1875. On the dynamic evidence of the molecular composition of bodies. *Nature* **11**, 357–374.

May, R. M. 1977. Thresholds and breakpoints in ecosystems with a multiplicity of stable states. *Nature* **269**, 471–477.

McCauley, E., W. G. Wilson, and A. M. de Roos. 1993. Dynamics of age-structured and spatially structured predator–prey interactions: individual-based models and population-level formulations. *Amer. Naturalist* **142**, 412–442.

Menge, B. A. 1976. Organization of the New England rocky intertidal community: role of predation competition, and environmental heterogeneity. *Ecol. Monographs* **46**, 355–393.

Murray, J. D. 1989. *Mathematical Biology*. Heidelberg: Springer-Verlag.

Nielsen, C. 1987. Distribution of stream-edge vegetation along a gradient of current velocity. *J. Ecol.* **75**, 513–522.

Nisbet, R. M. and W. S. C. Gurney. 1982. *Modeling Fluctuating Populations*. Chichester: Wiley.

Nisbet, R. M. and L. Onyiah. 1994. Population dynamic consequences of competition within and between age-classes. *J. Math. Biol.* **32**, 329–344.

Nisbet, R. M., A. H. Ross, and A. J. Brooks. 1995. Empirically based dynamic energy budget models: theory and an application to ecotoxicology. *Nonlinear World*, in press.

Noble, I. 1993. A model of the responses of ecotones to climate change. *Ecol. Appl.* **3**, 396–403.

Noy-Meir, I. 1975. Stability of grazing systems: an application of predator–prey graphs. *J. Ecol.* **63**, 459–481.

Paine, R. T. and S. A. Levin. 1981. Intertidal landscapes: disturbance and the dynamics of pattern. *Ecol. Monographs* **51**, 145–178.

Robles, C. 1987. Predator foraging characteristics and prey population structure on a sheltered shore. *Ecology* **68**, 1502–1514.

Robles, C. 1994. Changing recruitment rates in constant species assemblages: implications for predation theory in intertidal communities. Unpublished.

Roughgarden, J., S. D. Gaines, and H. P. Possingham. 1988. Recruitment dynamics in complex life cycles. *Science* **241**, 1460–1466.

Sato, K. and Y. Iwasa. 1993. Modeling of wave regeneration in subalpine Abies forests: population dynamics with spatial structure. *Ecology* **74**, 1538–1550.

Schlögl, F. 1972. Chemical reaction models for non-equilibrium phase transitions. *Z. Phys.* **253**, 147–161.

Slatyer, R. O. and I. Noble. 1992. In *Landscape Boundaries: Consequences for Biotic Diversity and Ecological Flows*, A. J. Hansen and F. di Castri (Eds), pp. 346–359. New York: Springer-Verlag.

Wilson, W. G. and W. G. Laidlaw. 1992. Microscopic-based fluid flow invasion simulations. *J. Statist. Phys.* **66**, 1165–1176.

Wilson, W. G. and R. M. Nisbet. 1996. Cooperation and competition along smooth environmental gradients. Unpublished.

Wilson, W. G. and C. A. Vause. 1989. Ferromagnetic $q = 4,5$ Potts models on the two-dimensional Penrose and square lattices. *Phys. Rev. B* **39**, 4651.

Wolfram, S. 1983. Statistical mechanics of cellular automata. *Rev. Mod. Phys.* **55**, 601–644.