Dynamics of age- and spatially-structured predator-prey interactions: individual based models and population level formulations

McCauley, E.; Wilson, W.G.; de Roos, A.M.

DOI
10.1086/285547

Publication date
1993

Published in
American Naturalist

DYNAMICS OF AGE-STRUCTURED AND SPATIALLY STRUCTURED PREDATOR-PREY INTERACTIONS: INDIVIDUAL-BASED MODELS AND POPULATION-LEVEL FORMULATIONS

EDWARD MCCAULEY,* WILLIAM G. WILSON,* AND ANDRE M. DE ROOS†

*Division of Ecology, Department of Biological Sciences, University of Calgary, Calgary, Alberta T2N 1N4 Canada; †Department of Pure and Applied Biology, University of Amsterdam, Kruislaan 318, 1098 SM Amsterdam, The Netherlands

Submitted October 31, 1991; Revised August 6, 1992; Accepted August 27, 1992

Abstract.—In this article, we investigate the spatial and temporal dynamics of predator and prey populations using an individual-based modeling approach. In our models, the individual is the fundamental unit, and the dynamics are governed by individual rules for growth, movement, reproduction, feeding, and mortality. We first establish the congruence between age-structured predator-prey population models and the corresponding individual-based population model under homogeneous spatial conditions. Given the agreement between the formalisms, we then use the individual-based model to investigate the dynamics of spatially structured predator-prey systems. In particular, we contrast the dynamics of predator-prey systems in which predators adopt either an "ambush" or a "cruising" strategy. We show that the stability of the spatially structured predator-prey system depends on the relative mobility of prey and predators and that prey mobility, in particular, has a strong effect on stability. Local density dependence in prey reproduction can quantitatively alter the asymmetrical influence of prey mobility on stability, but we show that the asymmetry exists when local density dependence is removed. We hypothesize that this asymmetrical response is due to prey "escape" in space caused by differences in rates of spread of prey and predator populations that arise because of fundamental differences between prey and predator reproduction.

Ecologists frequently reject population models because they are too simple. Populations are composed of highly complex individuals, whose traits such as age, stage, or genotype affect their behavioral responses and performance (i.e., probability of reproducing or dying). Since these complex individuals also live in environments that vary spatially and temporally, individuals can possess separate "histories," which makes their performance unique. Under these circumstances ecological interactions take on a "local" character. Sources of complexity such as these clearly limit our capacity to describe population processes by averaging the contributions of individuals.

Great strides have been made recently in developing models that can be used to understand how biological information from the individual level is translated into population-level processes (see, e.g., Gurney et al. 1983; Metz and Diekmann 1986; Caswell 1989). These formulations provide explicit links between physiological ecology and population dynamics by allowing the time evolution of vital rates to depend on an arbitrary number of individual state variables such as age,

Am. Nat. 1993. Vol. 142, pp. 412–442.

size, or energy reserves. The recognition that the population is not composed of a homogeneous collection of individuals with identical "average" vital rates and environmental responses imparts a structure to the population that must be modeled explicitly. However, all of the above formulations assume that the principle of mass action applies on either a global or a local scale to determine population-level responses (see, e.g., Metz and de Roos 1992). Thus, despite significant progress on model formulations for physiologically structured populations, several problems remain impervious, especially those dealing with the potentially local character of ecological interactions and the intractability of studying joint features of complexity (e.g., investigating the dynamical effects of age-structure *and* spatial variability).

One potential solution, advocated recently (see, e.g., Pacala and Silander 1985; Huston et al. 1988), is to abandon the constraints of a population-level description of demographic processes and focus instead on individual-based models. These models treat the population as a collection or ensemble of individuals, with explicit rules governing individual biology and interactions with the environment. Population-level phenomena (e.g., temporal or spatial dynamics) or vital rates can then be inferred directly from the contributions of individuals in the ensemble.

Individual-based models seem to offer some advantages: (1) Individual processes such as movement in space, growth, reproduction, or behavioral and ecological interactions can be represented explicitly, and they can depend on a number of individual state variables such as age, stage, genetic composition, or feeding history. (2) Time scales for processes such as movement and aging can be separated and varied independently. (3) Local interactions can occur, and the adherence to mass action on various spatial scales can be manipulated.

The added realism of individual-based models that is so attractive creates three major difficulties. First, modeling the complex features of individual biology and interactions requires considerable computational intensity, especially if dynamic rules for individual energetics are employed. Thus, the size of the "population" that can be simulated on any particular spatial scale may be limited, and this could introduce potential sources of instability such as demographic stochasticity that would distort the population dynamics. Second, the complexity of the interactions resulting from the added realism can render the identification of mechanisms responsible for the population-level dynamic behavior virtually impossible (i.e., the simulation becomes so complex that teasing apart the mechanisms or controlling parameters causing changes in stability becomes extremely difficult; see, e.g., Murdoch et al. 1992). The third difficulty concerns the implementation of rules governing interactions among discrete entities. Once the individual is adopted as the fundamental modeling unit, there is a broad spectrum of possible ways in which processes can be ordered in time or space. For example, individual interactions could occur sequentially (i.e., individuals could feed, then reproduce, then move) or concurrently. While this seems like mere computational detail, we show that it can profoundly alter dynamics by introducing new density-dependent relationships and by modifying parameter values.

In recent papers (de Roos et al. 1991; Wilson et al. 1993), we have described an individual-based modeling approach for studying population dynamics that we

believe directly confronts the three major difficulties outlined above. The first problem, regarding computational intensity, is addressed by employing an efficient algorithm for discrete-entity simulations that have been developed in other fields of science (e.g., chemical reaction kinetics; Kopelman 1988). The efficiency of the computations allows large numbers of interacting individuals to be investigated while incorporating a relatively complex state description and accommodating local interactions (de Roos et al. 1991; Wilson et al. 1993). In this article, we begin to address the second and third major problems, which deal with interpretation of mechanisms and sequences of events.

The second and third problems are very broad in scope. Biological species present an extremely diverse array of interactions among individuals, as well as diverse life-history traits. This diversity implies a wide range of possible ways of ordering rules for individual interactions that, to a large extent, has been ignored in population-level formalisms but must be considered once the individual is adopted as the fundamental modeling unit. It is beyond the realm of a single article to examine the entire range of possibilities; therefore, our approach toward the ultimate goal of understanding the dynamics of populations of highly complex individuals interacting in a spatial domain is to examine a general biological situation involving an interaction between an age-structured predator population and its prey. The complexities of this situation are simple enough that population-level analytical models can be constructed and analyzed for the spatially homogeneous case, yet complex enough that the further incorporation of spatial structure (i.e., limited mobilities of prey and predator or local density dependence in their interactions) renders the model analytically intractable.

We first present the biological complexities that we intend to add on the scale of the individual in the usual ordinary differential equation (ODE) framework. This framework provides the most concise "statement" of the biology; however, as we shall observe, it remains ambiguous concerning new questions that arise within the individual-based framework. After presenting a stability analysis of the age-structured predator-prey model, we then present a discussion of the individual-based behavior underlying the ODE model and the translation of that behavior into rules that can be incorporated into an individual-based simulation of the population dynamics. We show that it is possible to establish congruence between individual-based simulations and population-level analytical formulations under homogeneous spatial conditions. This congruence allows a direct and formal interpretation of mechanisms causing instability (or, conversely, stability) under situations in which mass action occurs. Interpreting mechanisms altering stability, as the assumption of mass action is relaxed in the discrete-entity simulations, then, simply relies on a comparison of changes on various spatial scales in vital rates and density dependence against the benchmark relationships in the homogeneous analytical and individual-based formulations.

The search for an equivalence between population-level models and individual-based models of a population should not be interpreted as an attempt to perform a "correct" numerical integration of a set of differential equations—the explicit goal is to understand how various features of individual-based behavior affect population dynamics. We believe that this understanding is enhanced by compar-

ing and contrasting the results from the population-level model and the individual-based simulation model. The comparison provides the foundation, whereas the contrast provides biological insight into population dynamics.

Finally, we use the discrete-entity simulations to study the spatial interaction between age-structured populations of predators and their prey (i.e., under conditions in which the assumption of mass action is relaxed). We investigate how foraging strategies by predators, such as sit-and-wait versus cruising predation, affect the stability of predator-prey population dynamics. Using the individual-based simulations, we manipulate the mobility of prey and predator individuals and assess the changes in dynamics at the population level.

BASELINE POPULATION-LEVEL PREDATOR-PREY MODEL

The predator-prey system that we wish to study possesses considerable biological realism and range of dynamics. In this system, the predator population is age structured with juvenile (with population J) and adult (with population A) predators sharing the same food supply (the prey population, F). Juveniles cannot reproduce, and they develop at a constant rate (i.e., there is a fixed developmental delay [τ]). Juveniles and adults are assumed to attack prey at the same rate (a); they possess a Type II functional response with handling time T_h and experience identical background mortality (d). Growth of F is density-dependent and follows the logistic equation with intrinsic growth rate r and carrying capacity K, and adult predators convert ingested prey into newborn juvenile predators with constant conversion efficiency (E_A).

The population balance equations (coupled differential equations) describing a population of individuals that encapsulate the assumptions described above for individuals are

$$dF/dt = rF(1 - F/K) - [aF/(1 + aT_hF)](J + A), \tag{1}$$

$$\begin{aligned} dJ/dt = {} & E_A[aF/(1 + aT_hF)]A \\ & - E_A[aF(t - \tau)/(1 + aT_hF(t - \tau))]A(t - \tau)e^{-d\tau} - dJ, \end{aligned} \tag{2}$$

and

$$dA/dt = E_A[aF(t - \tau)/(1 + aT_hF(t - \tau))]A(t - \tau)e^{-d\tau} - dA. \tag{3}$$

Models with similar structure have been used recently for both "strategic" and "tactical" purposes. For example, Cushing and Saleem (1982) and Hastings and Wollkind (1982) have used these formulations to determine the general effects of age structure or time delays on the stability of predator-prey interactions. Nisbet et al. (1989) have used such models of "lumped" age classes to investigate the detailed dynamics of one particular predator-prey system (the *Daphnia*-algal interaction in lakes and ponds). The biological complexity in these models, whether constructed for strategic or tactical purposes, pushes them close to (and often over) the "edge" of analytical tractability.

Equations for equilibria (prey, juvenile, and adult predators) and the stability analysis can be found in the Appendix. Figure 1 shows how stability changes

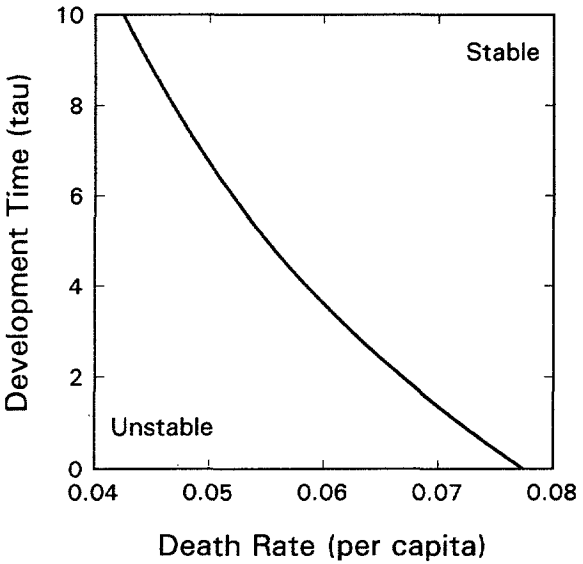


FIG. 1.—Stability portrait for linear stability analysis (see Appendix) of the baseline age-structured predator-prey model (eqq. [1]–[3]) for changes in two key parameters (predator development time and predator per capita death rate). The *solid line* indicates the stability boundary dividing parameter space into stable (above the line) and unstable (below the line) regions.

with two key parameters (τ and the per capita death rate of the predator): increasing the per capita death rate of predators stabilizes the dynamics, while decreasing the time delay associated with juvenile development is destabilizing. If the mechanisms causing predator-prey cycles or switches in stability are the same in the population-level analytical model and the individual-based simulation, then switches in stability in the discrete-entity simulations (produced by parameter variation) under homogeneous mixing conditions should follow the stability portrait for the analytical model (fig. 1).

LATTICE MODEL OF INTERACTING INDIVIDUALS

In this section, we describe the general computational features of our discrete entity simulations and describe our implementation of rules for interacting predator and prey individuals according to the assumptions outlined above. We then compare the dynamics of the individual-based simulations with those from the population-level model presented in the previous section, under homogeneous mixing conditions (conditions in which the two formulations should yield comparable results).

Discrete-entity simulations have been used in other branches of science to study interactions of "species" (e.g., chemical reaction kinetics [Kopelman 1988] and fluid flow in a porous medium [Wilson and Laidlaw 1992]). The models have

been described as belonging to the class of stochastic cellular automata (Hogeweg 1988). These simulations are also relevant in condensed-matter physics involving the phase transitions of systems with discrete degrees of freedom, such as the Ising and Potts models (Stanley 1971; Wilson 1989), which are made up of many entities, each with only a few possible states. These problems naturally lead to efficient simulations that are encoded at the bit level of the computer. For example, the Ising model is a simple system in which the individual elements, or "spins," have only two states. Thus, to represent a single spin on the computer, only a single bit of a computer word is necessary. If the computer word is 32 bits long, then 32 spins can be represented within a single word. While this represents a tremendous saving of memory, its primary advantage comes in the automatic parallelization of computations. Through simple Boolean operations (AND, OR, XOR, and NOT), the entire calculation can be performed. These operations are performed between words, not just individual bits, which implies that 32 calculations can be done simultaneously. Thus, large lattices of discrete entities can be simulated in reasonably short lengths of computer time, and this feature is very important for avoiding difficulties associated with demographic stochasticity that can be encountered in modeling small populations of individuals. (Indeed, initial applications of these models to ecological systems [Nisbet and Gurney 1982] were limited by this problem; R. M. Nisbet and W. S. C. Gurney, personal communication.)

In our model, individual prey and predators live in a spatial domain described by a two-dimensional lattice of sites (three-dimensional lattices are feasible and we are currently investigating features of dimensionality). Single bits are assigned to each lattice site to keep track of the species involved. For example, if a lattice site is occupied by a prey individual, the array containing the bits associated with prey would have a 1 at the bit position associated with that lattice site. Otherwise the bit would be set to 0. A predator's position can be described similarly. It is then a simple matter of taking the conjunction of arrays for prey and predator species to determine which sites are occupied by one of each of the two species and therefore the locations of sites where an interaction is about to take place. Individual histories of both predators and prey can be described with additional arrays of bits.

In our model, individual predators are characterized by their age, stage (juvenile or adult), time since last feeding, and number of prey consumed. Despite the considerable amount of detail concerning individual state, simulations analyzed in this article study the dynamics of up to 32,768 individuals (16,384 prey and predators). These individuals live and interact on a lattice of 128 $\times$ 128 sites with periodic boundary conditions (i.e., with no repelling boundaries).

The population dynamics of juvenile plus adult predators ($C(t)$) and of prey ($F(t)$) are modeled as discrete time processes consisting of two major parts: movement and interactions. Because we wish to use the formulation to study the entire range of spatial dynamics presented by populations (e.g., from conditions of continuous mixing to environmental patches linked by migration), we have incorporated a variety of options for prey and predator movement. Prey and predators can independently be stationary, display diffusive movement, or mix homoge-

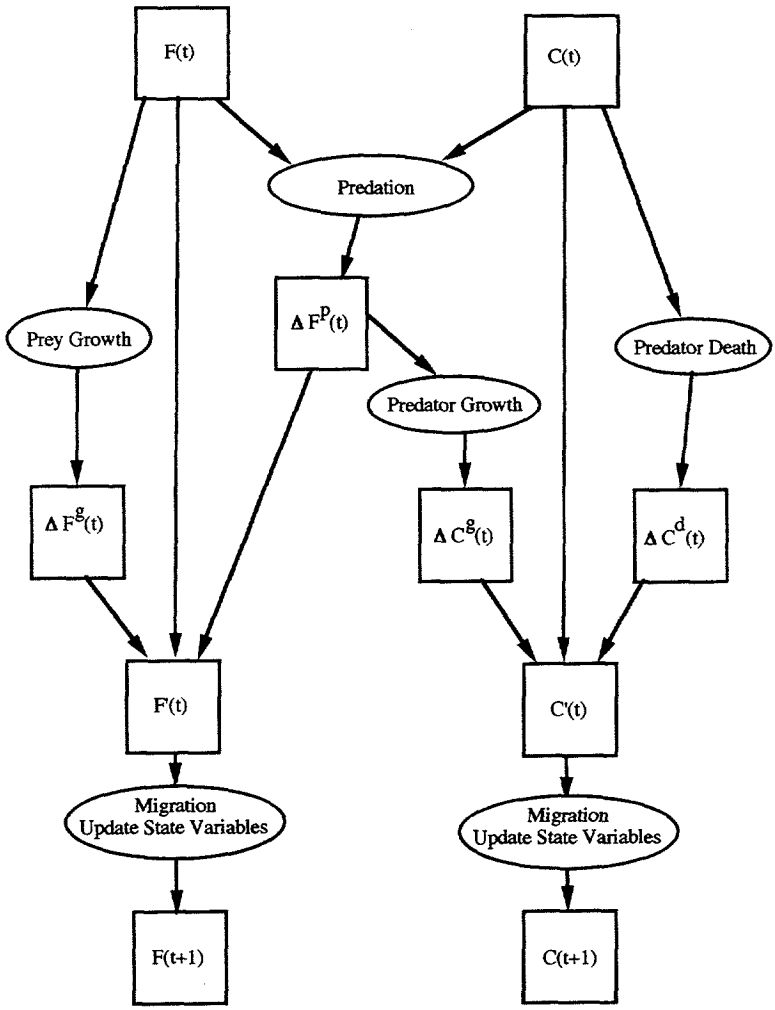


FIG. 2.—A description of the processes and their time sequence that occur during a single time step in the lattice simulation model. Interactions and demographic processes for prey (F) and predators (C) are denoted by *elliptical compartments*, and state variables and outputs are indicated by *square compartments*. Notice that the Δ − lattices are combined in a single step to calculate the resulting distribution of individuals, instead of the processes' acting sequentially (see fig. 6).

neously. Unlike other model formulations (e.g., Nicholson-Bailey-type models), processes of aging and mixing can proceed independently with different rates.

Figure 2 shows the order of individual movement, feeding, reproduction, and mortality in our model. We investigate, in this article, a variety of biological scenarios, including situations in which individual predators or prey mix homogeneously at each time step or move diffusively a variable distance throughout the lattice. Movement of prey and predators takes place simultaneously. Homoge-

neous mixing is achieved by taking all individuals alive at the beginning of the time step and scattering them randomly over the lattice. Diffusive movement is achieved by randomly walking all individuals through the lattice a variable number of steps. Once the migration or mixing portion of the time step is completed, operations on predators and prey are performed sequentially within the time step, which means that each operation is done within a single loop, or pass, through the lattice. Thus, a time step is actually made up of several passes through the lattice, each pass performing different operations on the predators and prey. (Note that executing a process at the end of the time step is equivalent to performing it at the beginning of the next time step because of the iterative nature of the calculations.)

For example, once migration is completed, updates are performed concerning the state of predators. First, each predator's age is incremented by one (five bits are used to represent a predator's age, yielding a maximum life span of 31 simulation steps, or $2^5 - 1$ [additional bits can be added to extend the life span]), and a test is performed that compares the new age with a developmental time or delay. Thus, an individual's interaction with prey could be both age- and stage-dependent (i.e., juvenile vs. adult). The time since the last prey was encountered is augmented, and this information can be used to incorporate starvation components of mortality and to determine feeding responses for an individual encountering a prey. Thus, we can incorporate a variety of functional responses for predators. In this article, once a predator has eaten a prey, it cannot eat for a fixed number of time steps, which represents a handling time and yields a Type II functional response for predators (see, e.g., de Roos et al. 1991).

Once the time-dependent states of individuals have been updated, encounters between predators and prey are performed. Whenever a predator and prey end up on the same lattice site, the predator attacks the prey unless it is preoccupied "handling" a previously attacked prey. At present, attack efficiency is unity, implying that the predator is always successful. This efficiency could be varied from zero to unity to study further details of the interaction. In this lattice pass, the attacked prey are labeled for later removal (see below) and the food count for juvenile or adult predators is augmented for individuals that have consumed prey. This adult food count is used to calculate offspring production. Predator reproduction (the number of offspring produced) can depend on both the total number of prey units required and the pattern of timing between feedings; however, in this article we assume immediate conversion with a probabilistic interpretation (E_A). Offspring are placed randomly in one of four neighboring sites or, if these are occupied, a nearby site.

Predator mortality can have three components: (1) an age-dependent component related to maximum life span (described above), (2) a starvation-dependent component (a predator dies if it does not eat a prey within x time steps), and (3) a random, density-independent component (at each time step, a fixed fraction of individuals, selected at random, die). These mortality factors could be stage-dependent (i.e., juvenile predators could have rules for starvation that are different from those of adult predators). For our purposes here, however, juvenile mortality and adult mortality are identical, and we consider only random compo-

nents of mortality (i.e., at every time step a constant fraction, P_D, of predators die) to facilitate comparison with the population-level analytical model (see below).

Prey growth on the lattice is density-dependent. Prey reproduce one offspring with a certain probability, P_{rg}, at one of four neighboring sites. If this site is occupied, the newborn prey is aborted, and this produces a density-dependent growth process. Alternatively, prey could have assured birth equivalent to the process of predator reproduction that yields density-independent growth with only the lattice size constituting a hard upper limit (a case not studied in this article, but examined in Wilson et al. 1993).

The Δ-lattices depicted in figure 2 are important components of the operations that occur within a time-step iteration. These lattices mark the positions of individuals that represent "outputs" of processes such as predation (ΔF^p), growth (ΔF^g and ΔC^g), or mortality (ΔC^d). Once all of the outputs are collected, the information is combined with the initial population distribution (e.g., $F(t)$) to determine the population size and distribution at the end of the time step. The use of these Δ-lattices ensures that interactions and demographic processes operate concurrently rather than sequentially. Later, we show that this independence is crucial for establishing congruence with population-level formulations.

With homogeneous mixing of prey and predators, the individual rules for growth, reproduction, interaction, and mortality are similar to those assumed for age-structured Lotka-Volterra-type models. In the next section, we compare dynamics from the lattice model with the population-level analytical model described above. As mentioned earlier, this comparison provides an essential foundation to ensure that under similar conditions the population-level model and individual-based simulation yield comparable results.

COMPARISON BETWEEN ANALYTICAL AND LATTICE SIMULATION MODELS

Before comparisons can be made of model dynamics, we have to interpret parameter estimates for population processes and interactions in both model formulations. Table 1 lists these parameter values. Only the handling time of the predator requires careful interpretation, because in the simulation model searching and handling of prey by predators takes place on the same time scale as predator population dynamics (in contrast to the ODE model, which assumes that behavioral and population-level processes are in a "pseudo" steady state). All newborn predators are hungry, whereas dying predators can be either hungry or satiated. This implies that the fraction of predators returning to the searching state in the subsequent time step is not $1/T_h$ as expected, but rather the overall handling time is $(1/T_h + P_D)^{-1}$ to account for this asymmetry.

Figure 3 shows how equilibria predicted by the analytical model compare to those observed from runs of our lattice model under homogeneous mixing conditions for changes in key parameters, such as the per capita growth rate of prey and the per capita death rates of predators. Overall, the discrete-entity simulations captured the classic features of equilibrium variation in Lotka-Volterra-type models: predator equilibrium density is positively related to prey growth rate (fig.

TABLE 1

A COMPARISON OF PARAMETERS FOR THE PREDATOR-PREY MODEL (ODE) DESCRIBED BY EQUATIONS (1)–(3) AND THE DISCRETE-ENTITY LATTICE SIMULATION MODEL

ODE	Lattice Simulation Model	Description
r	P_{rg}	Prey intrinsic growth rate
K	$128 \times 128 = 16{,}384$	Carrying capacity
a	$\frac{1}{16{,}384}$	Prey attack rate
T_h	$\frac{(T_h - 1)}{1 + P_D(T_h - 1)}$	Prey handling time
τ	$\tau - 1$	Juvenile development time
d	P_D	Predator per capita death rate
E_A	E_A	Predator conversion efficiency

NOTE.—The minimum time step in the lattice model is one, which corresponds to no delay. Symbols are as defined in sections Baseline Population-Level Predator-Prey Model and Lattice Model of Interacting Individuals.

3*A*), the prey equilibrium is positively related to predator per capita death rate (fig. 3*B*), and the predator equilibrium varies nonlinearly with predator per capita death rate (fig. 3*C*).

In classic Lotka-Volterra models, the predator equilibrium increases in direct proportion to the intrinsic growth rate of prey. Increased productivity of prey supports a larger predator population, which in turn suppresses prey to the same level observed in less productive environments. While our discrete-entity simulations captured the positive aspect of the relationship, it is not linear (fig. 3*A*). This probably results from a feature not present in classic models. Although predator birth is assured on the lattice, there is a maximum number of predators that can be supported (128^2), and as this maximum is approached the number of new "conceived" predators that are "aborted" increases.

Increasing the per capita death rates of juvenile and adult predators increases prey equilibrium density in classical predator-prey models. The prey equilibrium increases because a higher prey density is required to yield a higher predator birth rate that balances the higher predator death rate at equilibrium. As long as increases in prey productivity can compensate for increases in the per capita death rate, the predator equilibrium density does not change; but, as the equilibrium prey density approaches the carrying capacity, subsequent increases in predator birth rate cannot compensate and the predator equilibrium density declines. The lattice model captured this essential feature precisely (compare fig. 3*B* and *C*).

While we cannot claim perfect congruence between the lattice and analytical versions in terms of equilibria, there is certainly qualitative congruence and substantial quantitative agreement.

To examine the correspondence in stability between the lattice model and the analytical model, we compare dynamics observed on the lattice for parameter combinations that traverse the stability boundaries determined for the analytical model (fig. 1). Figure 4 shows how changes in the variability in the prey popula-

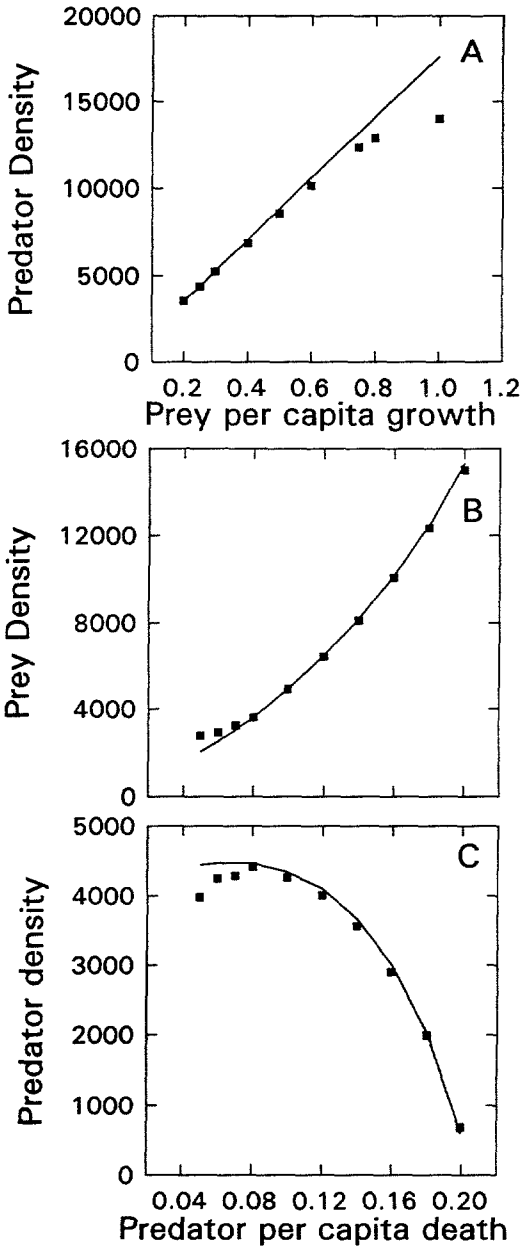


FIG. 3.—Predicted changes (*solid line*) in equilibria based on the analytical age-structured predator-prey model (eqq. [1]–[3]) and observed equilibria from the lattice simulation model (*square symbols*) for variation in two key parameters: prey per capita growth rate (*A*), and predator per capita death rate (*B*, *C*).

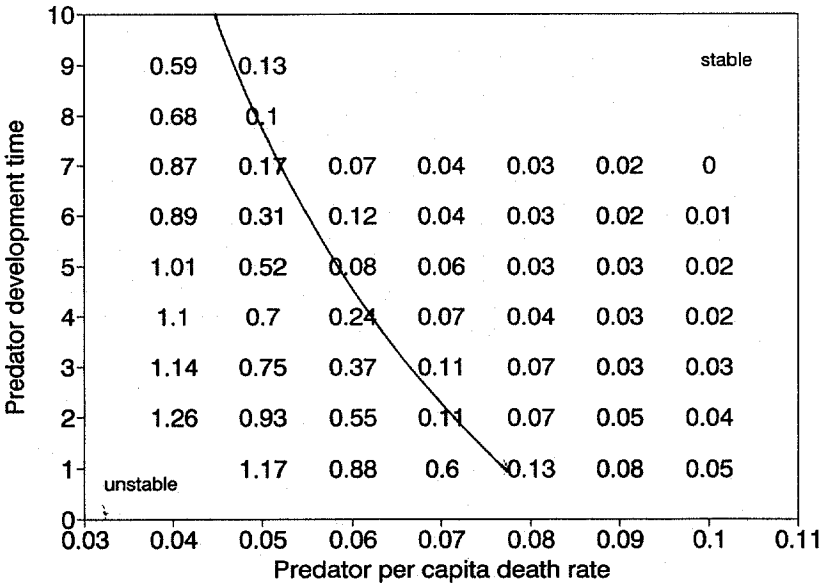


FIG. 4.—A comparison of changes in the observed coefficient of variation in the prey population from lattice simulation runs for the parameter plane in fig. 1, with corresponding parameter values. The *solid line* is the stability boundary shown in fig. 1. Note that a predator development time of one for the lattice simulation is equivalent to a zero development time in the analytical model (see table 1).

tion from the lattice model compare with expected changes based on the location of the stability boundary for the analytical model. In unstable regions of the stability plane, prey variability is high (fig. 4) and there are clear predator-prey cycles as predicted (fig. 5). Far away from the stability boundary the cycles have large amplitude, but, as it is approached by increasing maturation time, the amplitude of the cycles decreases. Simulations very near the stability boundary (but still in the unstable region) show small amplitude cycles that seem to damp out, only to appear again at other time intervals (fig. 5). Once the stability boundary is crossed (i.e., into the stable region), the dynamics on the lattice are "stable." There is no evidence of cyclic dynamics, and there are only small, stochastic fluctuations around the equilibrium density (fig. 5). Time-series analysis shows that these stochastic fluctuations damp out over time, and the return time to equilibrium decreases as parameter combinations move away from the stability boundary deeper into the stable region.

The congruence of the dynamics of the analytical model and the dynamics of the lattice model is most encouraging. Under homogeneous conditions, the population dynamics on the lattice mirror to a large extent the analytical results (i.e., the dynamics of a population of interacting individuals are directly comparable to a population-level description). Qualitative shifts in dynamics occur for corresponding changes in parameter values with appropriate changes in equilibria.

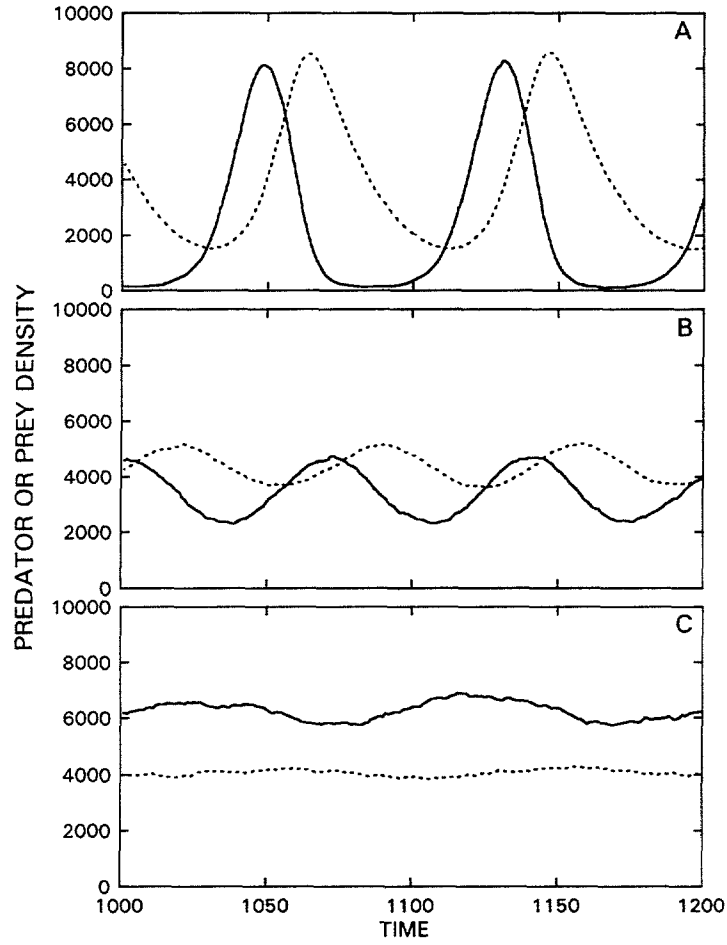


FIG. 5.—Examples of dynamics of predators (*dashed line*) and prey (*solid line*) populations for parameter combinations of predator development time and predator per capita death rate that cross the stability boundary. *A*, Predator development time = 1, predator per capita death rate = 0.06. *B*, Predator development time = 2, predator per capita death rate = 0.07. *C*, Predator development time = 3, predator per capita death rate = 0.08.

One of the major criticisms leveled at individual-based simulations is that the interpretation of mechanisms causing changes in stability is extremely difficult to infer from the model. This results from the fact that rules are formulated at the individual level, which in turn produces population-level dynamic responses via individual interactions. These individual rules and/or interactions can have subtle effects on density dependence at the population level that may produce unintentional effects on stability. The agreement that we have established between model formulations directly confronts this criticism.

However, we have found that, to establish this congruence, careful attention

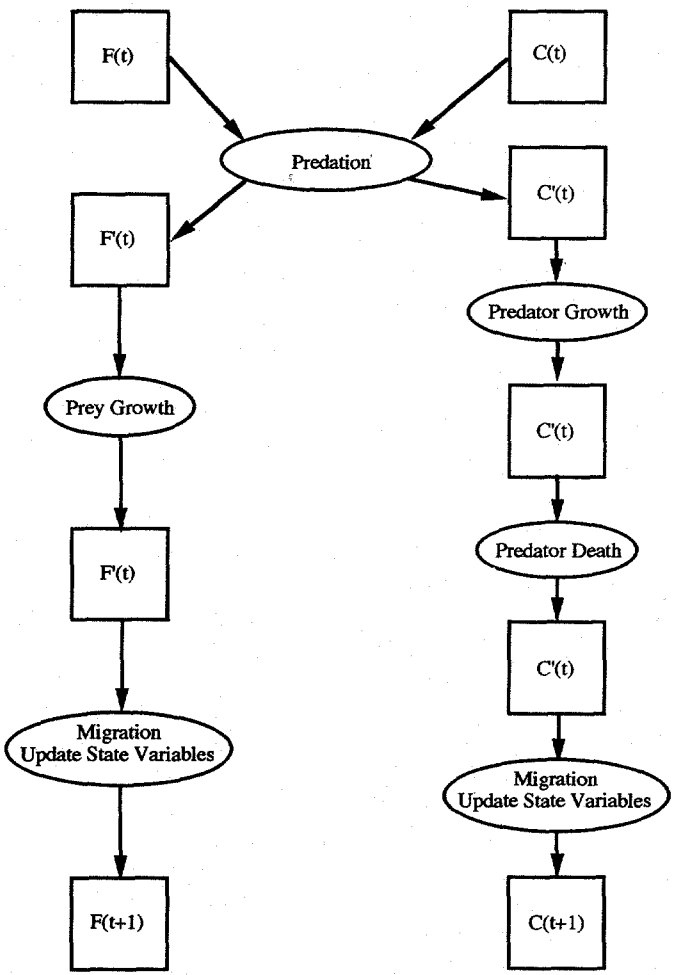


FIG. 6.—A description of processes that introduces complex interactions between prey and predator density and per capita growth rates due to sequencing of events. As in fig. 2, processes are represented by *elliptical compartments*, state and output variables as *square compartments*.

must be paid to the relationship between assumptions in the analytical model and sequencing of events (within a time step) when modeling interactions among discrete individuals.

For example, figure 6 portrays a slightly different sequence of events for the same processes and ecological interactions depicted in figure 2. In this scenario (fig. 6), predation, reproduction, and mortality occur sequentially within a time step (recall that this is possible because a time step actually contains several operational passes through the lattice). Consider the first sequence of events for prey: prey encountered by hungry predators at time t are consumed, then prey

surviving predation reproduce. At time t, a certain fraction of the prey population was "destined" to reproduce (i.e., a certain fraction of prey had conceived offspring but had not yet released them as newborn prey). Thus, predators consumed prey that would have reproduced, and this introduces an interaction between prey per capita growth rate and predator density. This sequence of events would lead to the following ODE describing prey growth:

$$dF/dt = G(F, C) - M(F, C), \tag{4}$$

where $G(\,)$ and $M(\,)$ represent growth and mortality functions, respectively. Equation (4) is structurally different from the ODE model for prey growth described previously (eq. [1]).

The effective prey growth rate at any given prey density is also altered by sequencing of events. Prey offspring compete locally for space. A newborn prey attempts to recruit to one of the four neighboring sites, and if all sites are occupied the prey is aborted. If prey reproduction occurs after prey have been consumed by predators (creating more open space on the lattice), then competition for space by recruiting offspring is reduced.

In general, we have found that sequencing of processes within a time step can have both qualitative and quantitative effects on dynamics. Qualitative effects include removing or introducing new stability boundaries for particular controlling parameters, and quantitative effects include shifting the stability boundaries in parameter space. Figure 7 shows an example in which merely changing the sequence of events dramatically alters the stability of the predator-prey interaction and removes the congruence between the lattice and analytical models. In an upcoming article (A. M. de Roos, E. McCauley, and W. G. Wilson, unpublished manuscript), we investigate problems posed by process sequencing in simpler unstructured models of predator-prey interactions.

These examples underscore the importance of establishing agreement between individual-based simulations and analytical formulations under homogeneous conditions. Once this has been established, the lattice model can then be used to investigate spatial problems that are analytically intractable (i.e., for situations in which it is not possible to even write down an ODE model for population dynamics). The congruence provides us with an understanding of how the classes of dynamics are produced under homogeneous mixing conditions on the lattice; subsequent changes in vital rates or density-dependent responses that arise as interactions become local (a property that cannot be achieved at the individual level in analytical population models) can then be contrasted with the homogeneous case. This feature greatly facilitates the discovery of mechanisms causing changes in dynamics when individual predators and prey can move diffusively and interact on the lattice (i.e., relaxing the assumption of mass action). In the next section, we implement the biological model discussed in this section but replace the conditions of homogeneous mixing with limited prey and predator mobility to examine how the foraging strategy of ambush versus cruising predators, coupled with prey mobility, might influence stability of the predator-prey interaction—a problem that is seemingly impervious to treatment by analytical population models, given the level of biological complexity that we are discussing.

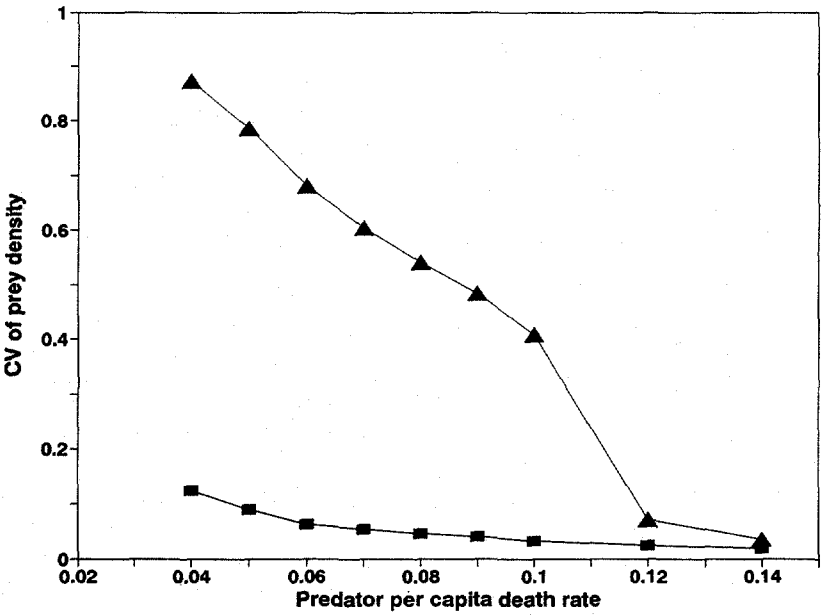


FIG. 7.—An example illustrating how the sequence of events can alter predator-prey dynamics in the lattice simulation model. Parameter values were chosen for the analytical model (eqq. [1]–[3]) that yield stable dynamics along with varying predator per capita death rate. The lattice simulation model following the concurrent rules outlined in fig. 2 captured this result (*square symbols*). The output from a lattice simulation model following the sequential rules described in fig. 6 (*triangles*) produces a result that is dramatically different from that of the analytical model, indicating a significant lack of congruence between the sequential rules and the analytical predator-prey model.

DYNAMICS OF PREDATOR-PREY SYSTEMS: AMBUSH VERSUS CRUISING PREDATORS

In this section, we use our discrete-entity simulations to examine how contrasting foraging behaviors (sit-and-wait/ambush or cruising tactics) affect the stability of predator-prey systems. Predator and prey individuals can move randomly on the lattice at each time step, and the distance moved by predators and prey can be varied independently. To investigate ambush versus cruising situations, we change the mobility of predators and observe changes in dynamics as the mobility of the prey varies.

The foraging behavior of predators is sometimes classified according to the relation between their mobility and that of their prey. Sit-and-wait, or ambush, predators remain stationary, and prey are attacked once they wander into an encounter area or volume. At the other extreme, cruising predators move throughout the habitat and attack prey that are either mobile or stationary. Much effort has been devoted to studying the behavioral aspects of these contrasting strategies, and each strategy is found in a wide variety of taxa, such as birds (e.g., swallows vs. bluebirds [Power 1980]), insects (e.g., water bugs [Cloarec 1990], *Notonecta* vs. *Chaoborus* [Neill 1981], centipedes [Formanowicz and

Bradley 1987], and beetles [Gilbert 1989]), crustaceans (e.g., *Leptodora* [Browman et al. 1989]), snakes (see, e.g., Jones and Whitford 1989), turtles (see, e.g., Formanowicz and Brodie, Jr. 1989), and fish (pike vs. bluegill [see, e.g., Savino and Stein 1989]). In addition, some species display both sit-and-wait and cruising behavior, depending on the prey density or encounter rate (e.g., the tropical water bug, matamata turtles). While foraging behavior has been investigated in some detail (see, e.g., Caraco and Gillespie 1986; Harper and Blake 1988), the population-level consequences of these different strategies have not been studied.

A Fixed Foraging Strategy

To examine the effects of ambush and cruising tactics, we chose a canonical set of predator and prey parameters that yield large amplitude fluctuations in abundance under complete mixing conditions (see, e.g., fig. 5*A*) and then examined how equilibrium densities and dynamics change with variation in mobility of individual predators and prey.

Changing the foraging strategy from cruising to ambush predation in environments in which prey mix continuously does not alter the variability in predator-prey dynamics (fig. 8*A*) or change the equilibrium levels of the populations (fig. 9). In this situation, prey mix randomly and the probability that a prey individual lands on a lattice site occupied by a predator is identical to the case in which predators cruise the habitat. This implies no change in the attack rate of the predator between cruising and ambush predation. In addition, since predator birth is assured (see Lattice Model of Interacting Individuals), there is no density-dependent interaction among stationary predator individuals (i.e., predator offspring produced do not compete locally for a site), and thus no change in the numerical response is observed for stationary predators.

The predator-prey dynamics do change, however, once the realistic feature is added that prey are mobile rather than randomly mixed throughout the environment. Figure 10 illustrates how the coefficient of variation of the prey population is altered with changes in predator and prey mobility. Reducing the cruising speed of predators for a given prey mobility, or reducing the mobility of prey for a given cruising predator speed, decreases the variability of the populations.

The reduction in the coefficient of variation reflects a decrease in the amplitude of the predator-prey cycles (fig. 8). As prey and/or predator mobility is reduced, the amplitude of the cycles decreases, and at very low mobilities of prey and predator (see, e.g., fig. 8*C*), the dynamics appear to be stable (i.e., there are small stochastic fluctuations and no sustained oscillations—time-series analysis shows that these stochastic fluctuations quickly damp out and are aperiodic). The change in dynamics with modifications of prey and predator mobility occurs without systematic changes in equilibria (fig. 9). We also find that decreasing prey and predator mobility introduces spatial heterogeneity in the distribution of individuals on the lattice (fig. 11). Figure 11 illustrates the temporal evolution of the spatial pattern of prey and predator individuals for cases in which predators are relatively immobile and prey mobility is altered. When prey or predators are mixed randomly, there is no spatial heterogeneity (not shown in figures). When prey are highly mobile, there is spatial heterogeneity but large regions of the

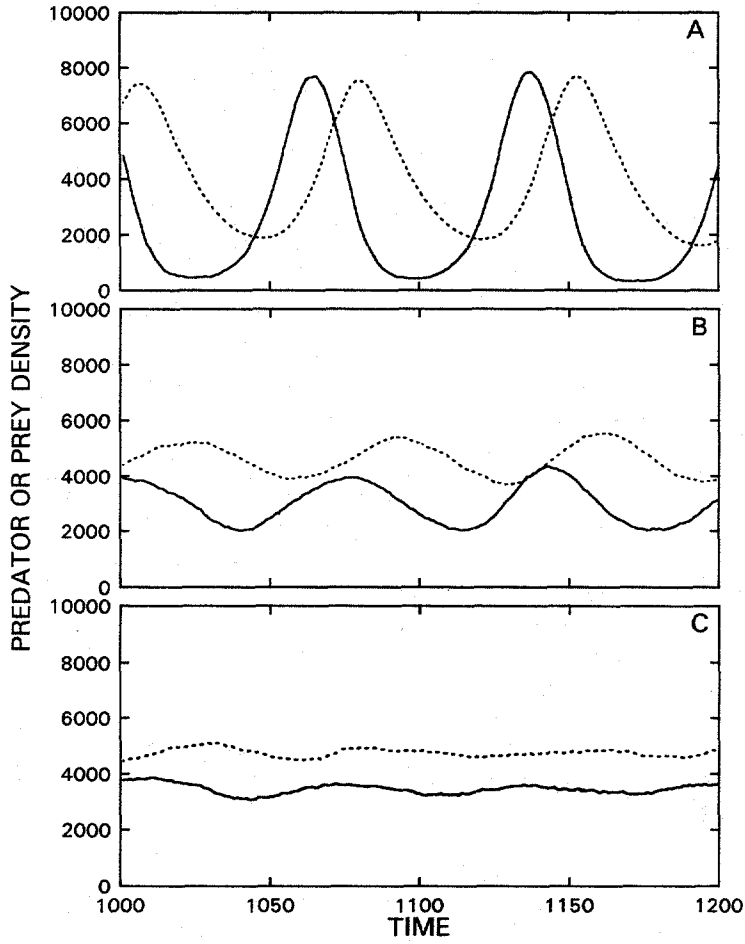


FIG. 8.—Examples of predator-prey dynamics for stationary predators and diffusive cases. In *A*, prey mix randomly and predators are stationary. In *B*, prey "diffuse" randomly eight lattice sites at each time step. In *C*, prey move randomly one lattice site at each time step. All other parameters are set for dynamics as in fig. 5*A*.

lattice remain correlated and display temporal fluctuations. As prey mobility is reduced, the distribution of individuals becomes more patchy and the synchrony among regions of the lattice decreases. This is readily observed by contrasting the formation and destruction of prey "patches" over time in different regions of the lattice in figure 11*B* or *C*. It is important to remember that the spatial heterogeneity arises from the limited mobility of individuals and the predator-prey interaction (see, e.g., de Roos et al. 1991).

Reducing the cruising speed of predators and prey also reveals that there is a significant asymmetry in the reduction of fluctuations (fig. 10): changes in prey mobility alter the fluctuations much more dramatically than corresponding changes in predator mobility.

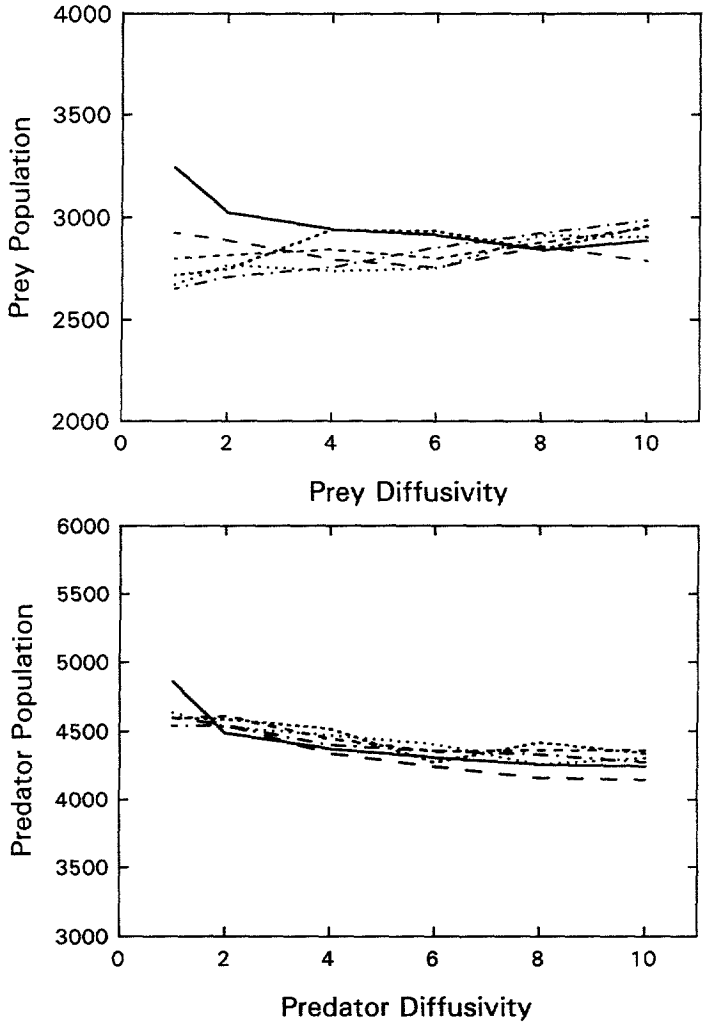


FIG. 9.—Variation in prey and predator equilibria measured on the lattice with changes in prey and predator diffusivities. Diffusivities of one (*solid line*), two (*long dash*), four (*medium dash*), six (*short dash*), eight (*dotted*), and ten (*dash-dot*) lattice sites per time step are shown.

How does the limited mobility of predators and prey reduce the amplitude of fluctuations in predator-prey dynamics, and how can the asymmetrical effect on variability brought about by changes in prey versus predator mobility be explained? One potential explanation for the reduced amplitude of the predator-prey cycles is that reducing prey and predator mobilities decreases the encounter rate, thereby reducing the attack rate in the predator functional response. Figure 12 contrasts the changes in predator attack rate for prey mixing and prey limited-mobility cases (note: the temporal dynamics for these exact cases are shown in

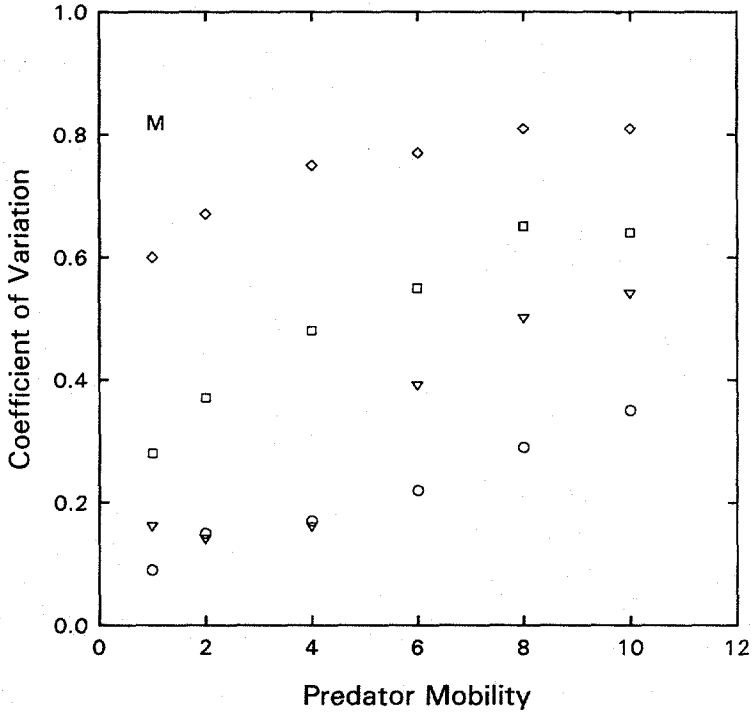


FIG. 10.—Coefficient of variation for prey dynamics in relation to mobility of individual prey and predators. Prey mobilities are represented as symbols: *circles* (one), *triangles* (two), *squares* (four), *diamonds* (eight), and *M* (random mixing at each time step).

fig. 8). As predicted, the attack rates decrease, but the magnitude of the effect is quite small. Reducing prey movement from randomly mixed to virtually stationary decreases the attack rate by only approximately 15%–20%. Reductions in the attack rate of this magnitude in the corresponding homogeneous ODE model yield cycles with reduced amplitude; but the effect is small compared with the dramatic reduction observed in the lattice model (see, e.g., fig. 8).

An alternative explanation considers how changes in mobility alter the spatial scale of interactions rather than modify density-dependent responses. Reducing mobility of prey and predators appears to set a spatial scale for the size of the ephemeral patches of prey and predators (see, e.g., fig. 11 and de Roos et al. 1991). Since the lattice size is fixed, the number of these patches will increase as the mobility of prey and predators declines. The patches are asynchronous and it is well known, at least in systems with externally imposed environmental patches (see recent reviews by Kareiva [1990] and Reeve [1990]), that asynchrony leads to reduced temporal fluctuations in prey and predator population density. We believe that the spatial pattern that arises under homogeneous environmental conditions on the lattice is probably not caused by diffusive instabilities (sensu Turing 1952; Murray 1989), since we have found (Wilson et al. 1993) spatial

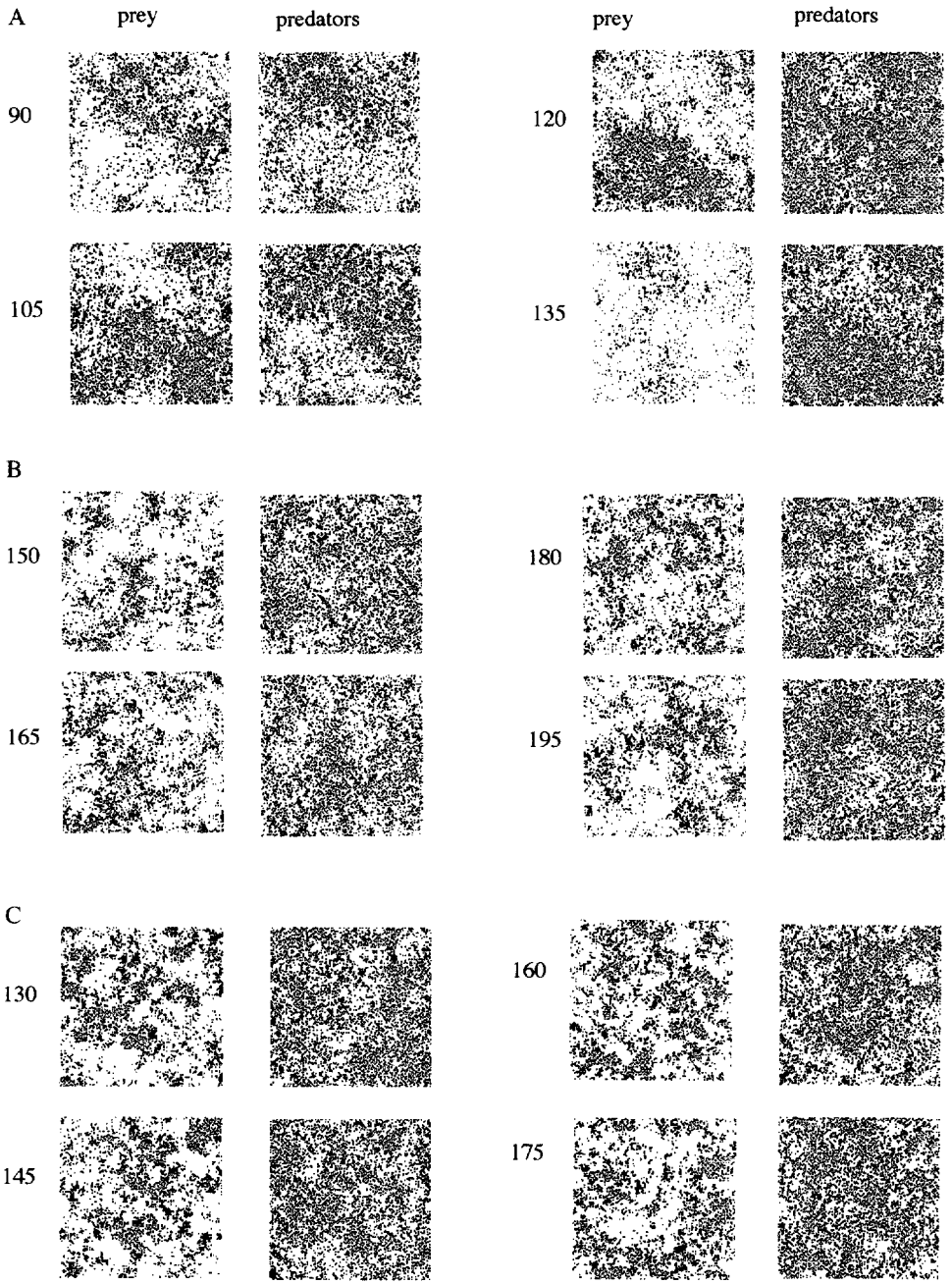


FIG. 11.—Pictures of the spatial distribution of prey and predators on the 128 × 128 lattice at different times. Each dot represents an individual, and the "snapshots" of the spatial distribution are taken at four instances in time separated by 15 time steps. *A*, Prey diffusivity = 8, predator diffusivity = 2. *B*, Prey diffusivity = 2, predator diffusivity = 2. *C*, Prey diffusivity = 1, predator diffusivity = 1.

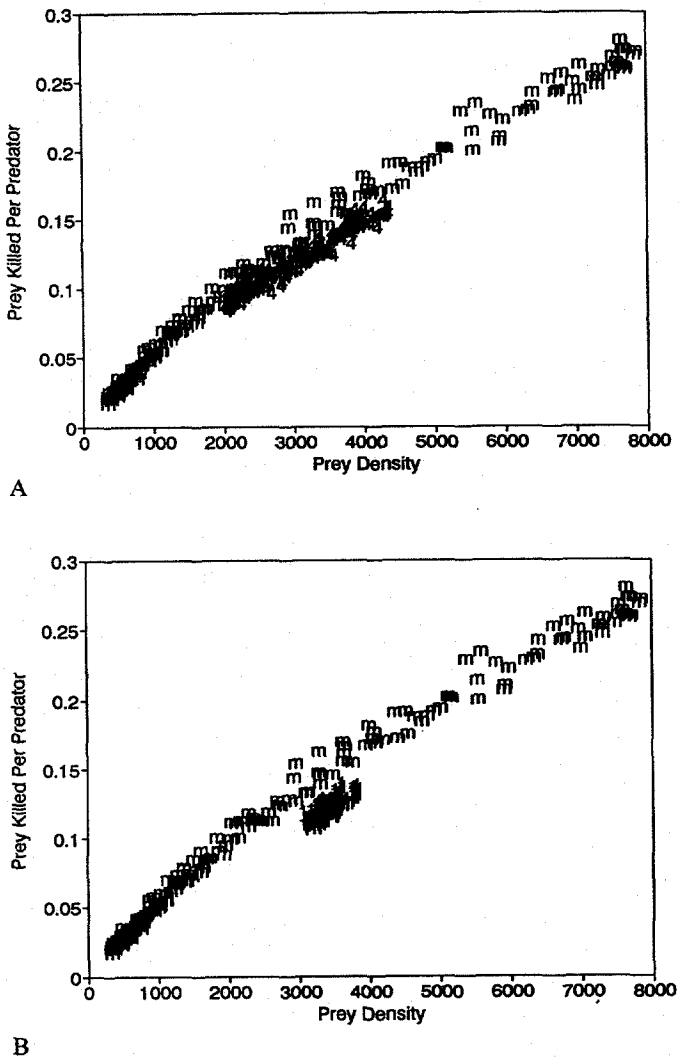


FIG. 12.—Number of prey killed per predator vs. prey density as measured on the lattice at different times. *A* compares attack rates of stationary predators for randomly mixing prey (*m*) and prey with diffusivities of four lattice sites per time step. *B* compares attack rates for mixing prey and prey with a mobility of one lattice site per time step.

inhomogeneities in Lotka-Volterra predator-prey systems in which diffusive instabilities are not possible (Okubo 1980).

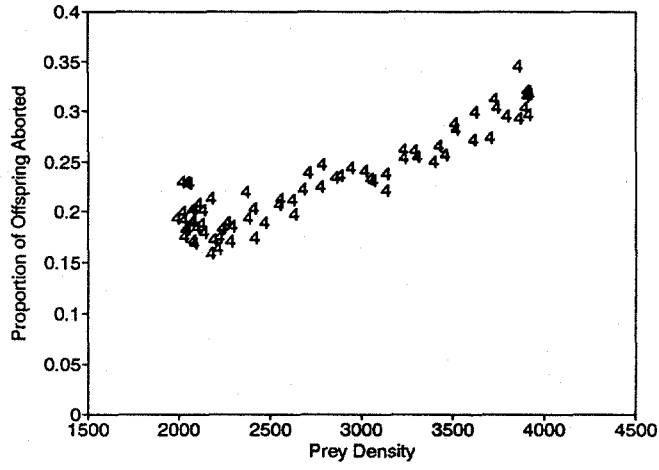
In our model, the patches develop from the finite mobility of prey and predators and the predator-prey interaction among individuals, without the imposition of environmental patches. We believe that there are at least two requirements for the development of these patches and the maintenance of their asynchrony. First, there must be some degree of localization of interactions among individuals. The

interactions include those between prey individuals (e.g., two individuals attempting to recruit to a particular spatial site) and between prey and predator individuals. The limited mobility of individuals promotes local effects and prevents them from being quickly propagated over the lattice. Second, there must be individual stochasticity or variability in the interactions among individuals, which assures asynchrony among spatial regions (Chesson 1981).

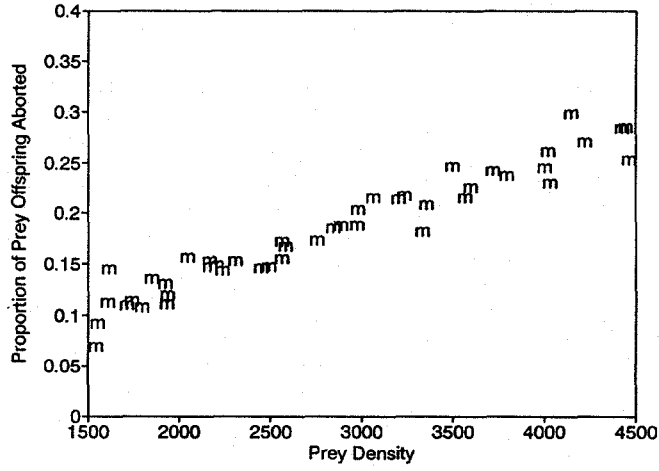
Key areas for future research, which are beyond the scope of this article, include understanding how the spatial heterogeneity arises from the limited mobility of prey and predators and how prey and predator mobility interact with their biological features to determine patch characteristics, such as average size or longevity. This understanding will allow us to determine, for realisitic prey and predator individuals, the spatial scale of the habitat below which the prey and predators are behaving as a homogeneous system essentially following the law of mass action and above which there is a potential for asynchrony to develop among the ephemeral patches.

Figure 10 shows that prey mobility has a stronger influence on dynamics than does predator mobility. A possible explanation for this asymmetry is that prey density dependence changes as prey mobility decreases. Prey reproduction is conditional on the occupancy of neighboring sites by other prey. A prey offspring attempts to recruit to one of four neighboring lattice sites, and if this site is occupied the offspring is aborted. In the homogeneous case (i.e., when prey mix continuously), this rule leads to a linear increase in the abortion rate of prey offspring (fig. 13) and subsequently to a linear density dependence between prey per capita growth rates and prey density (not shown), since the probability of a prey's producing an offspring is fixed. In systems where local patches of prey can develop, however, increasing prey density on the lattice produces both a nonlinear increase in the abortion rate (fig. 13) and an overall increase in the abortion rate, which, in turn, modifies the shape and slope of the relationship between prey per capita growth rate and prey density.

To test whether local density dependence in prey reproduction explains the asymmetrical response, we modified prey reproduction by assuring that prey offspring recruit to the lattice and repeated the simulations for limited prey and predator mobility. For this experiment, the rules for prey and predator reproduction are identical, producing density-independent reproduction. Figure 14 shows that when prey reproduction is assured (i.e., local density dependence is removed) the asymmetrical response is maintained. Increasing predator mobility while prey mobility is low leads to a modest increase in fluctuations, whereas increasing prey mobility with relatively immobile predators produces a much steeper increase in the amplitude of fluctuations. Removing prey density dependence increased the population fluctuations, which can be seen by comparing the coefficient of variation for corresponding prey and predator mobilities in figures 10 and 14. Indeed, at high prey and predator mobilities (i.e., mobilities greater than 12), the fluctuations in density were so large that extinction was commonly observed. This increased instability is consistent with general results from corresponding ODE models in which prey growth is density-independent and predators possess a Type II functional response (i.e., inverse density dependence). Thus,



A



B

FIG. 13.—Proportion of prey offspring that are aborted at a given prey density at different times measured on the lattice. Examples are shown for mixing prey (*m*) and prey with a diffusivity of four lattice sites per time step (*4*).

while local density dependence quantitatively modifies the asymmetrical response, it does not produce the asymmetry.

We hypothesize that the asymmetry results from the effect of prey escape on the lattice. When predator mobility is low, increasing prey mobility will allow prey to colonize "empty" regions of the lattice more quickly and grow unrestrained for a period of time. Prey can escape in space and increase their density locally prior to being "discovered" by the relatively slow-moving preda-

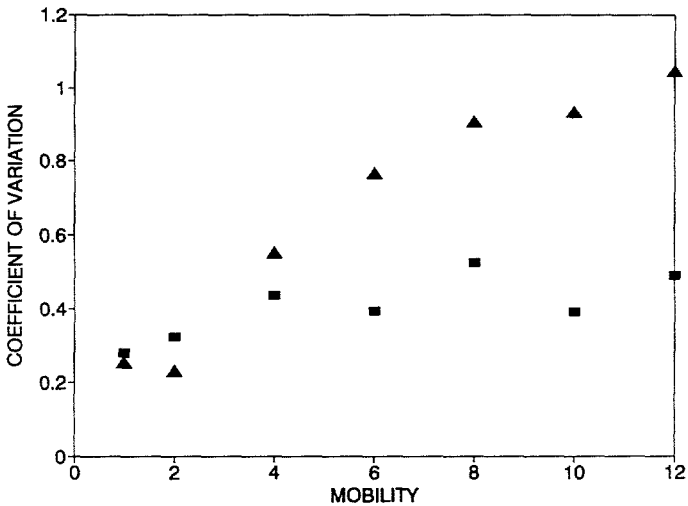


FIG. 14.—Changes in the coefficient of variation in relation to changes in mobility of prey and predator, assuming that prey reproduction is assured. *Squares* indicate the response for prey mobility fixed at one while predator mobility is manipulated. *Triangles* show the response for predator mobility of one with prey mobility varying.

tors. This mechanism may be the spatial "analogue" of the prey escape mechanism found in spatially homogeneous ODE models (see de Roos et al. 1990).

If only diffusive movement of predator and prey individuals is considered, the average territory covered by individuals with time passes through three scaling regimes, finally settling down to an area proportional to $t/\ln(t)$ for long times (t) in two dimensions (Larralde et al. 1992). This long-time scaling regime is valid for a constant number, N, of independently diffusing individuals. Unlike mere diffusing particles, however, individual predators and prey are interacting, reproducing, and dying, and there are other components that influence the spread of individuals, such as recruitment of offspring to the lattice. At very short times, when individuals are crowded, the scaling is much faster, with the area covered being proportional to t^2 in two dimensions (Larralde et al. 1992). We anticipate that a population that is both diffusing and reproducing propagates within the faster scaling regime: as individuals diffuse they become separated from their spatial cohort but surround themselves with offspring. Clearly, the rates of reproduction and mortality will determine the scaling regime for long times.

Prey and predators will differ in their scaling, since prey reproduction occurs at a constant rate, while predator reproduction is conditional on ingesting and converting these ingested prey into predator offspring with a conversion efficiency of less than one. Predator propagation must have a more complicated temporal scaling, approaching that of prey for high conversion efficiencies and low predator mortality and being significantly slower for low conversion efficiencies and high mortality. This difference allows the prey escape mechanism to operate asymmetrically, since it assures that the spread of prey individuals in a local area will be faster than that of predator individuals.

CONCLUSION

There are several possible modeling approaches that can be used to investigate how features of individuals and their interactions affect population dynamics. A common goal of these approaches is to understand how complexity at one level of biological organization influences phenomena at higher levels. One approach is to construct quantitative descriptions of individual biology and then either simulate ensembles of individuals or abstract population-level formalisms (see, e.g., Nisbet et al. 1989; de Roos et al. 1990; Gurney et al. 1990; McCauley et al. 1990; Metz and de Roos 1992; Murdoch et al. 1992). In this article, we have proposed that lattice simulation models may be particularly appropriate for studying cases in which local interactions are central to the problem. In other recent papers, we examine how limitations imposed by individual mobility affect dynamics in predator-prey systems (de Roos et al. 1991) and the relationship between stochasticity and stability in spatially diffusive predator-prey models (Wilson et al. 1993).

The use of lattice simulation models to study spatial dynamics of interacting populations supplements, without attempting to supplant, existing theoretical approaches involving metapopulation models. In the metapopulation approach (see recent reviews by Hastings [1990], Kareiva [1990], Reeve [1990], Taylor [1990], and Hanski and Gilpin [1991]), the population is typically divided into a number of explicit patches, which imposes a spatial scale on the system. However, there are many biological systems in which readily identifiable patches may not be a central feature of the environment. For example, in the open water of lakes, ponds, or oceans, one would be hard-pressed to define a patch, except by the circular route of using the spatial heterogeneity in the distribution of the organisms themselves. These situations provide a compelling argument for using a model formalism, such as the lattice modeling framework, in which patches are not defined externally but are generated by the interactions and movements of the organisms. Since the rules are implemented at the level of the individual, the relative importance of biological mechanisms causing spatial heterogeneity and formation of patches can be investigated (see, e.g., Addicott et al. 1987) along with the consequences for population dynamics.

The lattice simulation models are not restricted, however, to systems in which spatial heterogeneity or patchiness is "internally" generated. Patches can be imposed externally on the lattice, and questions concerning the role of asynchrony in affecting stability can be investigated as in metapopulation approaches (e.g., Nisbet et al. 1993), and the effects of local rules for migration or diffusion between or among patches on population dynamics can be assessed (see, e.g., Hassell et al. 1991). An important area of research must address the relation between system size and stability. The finite size of the lattice, combined with the characteristic spatial scale of the patches, dictates the number of feasible patches on the lattice and therefore the potential for asynchrony among patches. We are currently investigating how this scale dependence in stability relates to individual rules for mobility and density-dependent interactions.

ACKNOWLEDGMENTS

We would like to thank W. Gurney, W. Laidlaw, H. Metz, W. W. Murdoch, and R. Nisbet for many inspiring discussions and comments. A.deR. and W.G.W. were supported by postdoctoral fellowships from the University of Calgary. The research was supported by a Natural Sciences and Engineering Research Council operating grant to E.M.

APPENDIX

Equilibria and Stability of the Analytical Population Model

In this appendix we investigate the equilibrium and stability properties of the baseline population-level predator-prey model, described by the balance equations (1)–(3) in the main text. As a first step in the analysis we select r^{-1}, K, and r/a as our natural units in which to express the time and prey and predator densities, respectively, and we therefore define the scaled, dimensionless variables:

$$t' = rt; \quad F' = F/K; \quad J' = aJ/r; \quad A' = aA/r. \tag{A1}$$

In addition, we also define the following dimensionless parameter groups:

$$\delta = r\tau; \quad \mu = \frac{d}{r}; \quad \epsilon = \frac{aE_A K}{r}; \quad \beta = aT_h K. \tag{A2}$$

Using the scaled, dimensionless quantities of equations (A1) and (A2), we can reexpress the original system of balance equations (1)–(3) in the dimensionless form:

$$\frac{dF'}{dt} = F'(t')[1 - F'(t')] - \frac{F'(t')}{1 + \beta F'(t')}[J'(t') + A'(t')]; \tag{A3}$$

$$\frac{dJ'}{dt} = \epsilon \frac{F'(t')}{1 + \beta F'(t')} A'(t') - \epsilon \frac{F'(t' - \delta)}{1 + \beta F'(t' - \delta)} A'(t' - \delta) e^{-\mu\delta} - \mu J'(t'); \tag{A4}$$

$$\frac{dA'}{dt} = \epsilon \frac{F'(t' - \delta)}{1 + \beta F'(t' - \delta)} A'(t' - \delta) e^{-\mu\delta} - \mu A'(t'). \tag{A5}$$

The equivalences (A2) between the compound parameters in the above scaled model and the parameters of the original model (eqq. [1]–[3]) are also shown in table A1. In addition, this table identifies the relations between the compound parameters of the scaled model and the parameters that occur in the individual-based simulation model.

Below, we will derive expressions for the equilibrium densities of prey and juvenile and adult predator densities in the scaled population model described by equations (A3)–(A5). Subsequently, we will linearize the system of equations (A3)–(A5) to obtain the characteristic equation, which determines the stability of the population model for any given parameter combination. For simplicity of notation we will, in the following sections, drop the prime superscripts of the quantities t', F', J', and A', assuming that henceforward the quantities, t, F, J, and A will always refer to the scaled variables occurring in equations (A3)–(A5).

EQUILIBRIUM DENSITIES OF PREY AND PREDATORS

Equilibrium densities can be derived by equating the right-hand side of the equations (A3)–(A5) with zero and solving for the quantities F, J, and A, which are now assumed to be time-independent. We will restrict our analysis to the equilibrium situation in which both prey and predator individuals are present, that is, the internal equilibrium. Let the equilibrium densities of prey and juvenile and adult predators be denoted by F^*, J^*, and

TABLE A1

EQUIVALENCES BETWEEN THE COMPOUND PARAMETERS OF THE SCALED POPULATION-LEVEL MODEL, THE ORIGINAL PARAMETERS OF THE UNSCALED MODEL, AND THE PARAMETERS OF THE INDIVIDUAL-BASED SIMULATION MODEL

Scaled Model	Unscaled Model	Simulation Model
δ	$r\tau$	$R_{mg}P_{rg}\tau$
μ	$\frac{d}{r}$	$\frac{P_D}{R_{mg}P_{rg}}$
ϵ	$\frac{aE_AK}{r}$	$\frac{E_A}{R_{mg}P_{rg}}$
β	aT_hK	$\frac{T_h}{1 + P_DT_h}$

NOTE.—It should be noted that, in the individual-based simulation model, the product aK of the predator attack rate and the prey carrying capacity equals one and is hence omitted from the equivalence relations wherever it would occur. The number of prey offspring produced per reproductive event is represented by R_{mg}.

A^*, respectively. By setting the right-hand side of equation (A5) to zero we can immediately solve for F^*, which yields the following equation for the prey density in equilibrium:

$$F^* = \frac{\mu}{\epsilon e^{-\mu\delta} - \beta\mu}. \tag{A6}$$

From this expression we infer that the prey density is entirely determined by parameters that pertain to the predator biology, that is, the predator imposes the equilibrium density of prey.

We next set the right-hand side of equation (A4) to zero and substitute the prey density F in this equation with the expression (A6) for F^*. Straightforward algebraic manipulations then lead to the following relation between the juvenile and adult predator densities in equilibrium:

$$J^* = (e^{\mu\delta} - 1)A^*. \tag{A7}$$

Finally, we set the right-hand side of equation (A3) to zero and use the relations (A6) and (A7) to replace the quantities F and J. We can then solve for A^*, which leads to the following expressions for the equilibrium densities of juvenile and adult predators:

$$J^* = \frac{(e^{\mu\delta} - 1)[\epsilon^2 e^{-\mu\delta} - \epsilon\mu(\beta + 1)]}{(\epsilon - \beta\mu e^{\mu\delta})^2}; \tag{A8}$$

$$A^* = \frac{\epsilon^2 e^{-\mu\delta} - \epsilon\mu(\beta + 1)}{(\epsilon - \beta\mu e^{\mu\delta})^2}. \tag{A9}$$

The unscaled analogues of these expressions for the original model described by the equations (1)–(3) are given in table A1.

LOCAL STABILITY ANALYSIS

To study the local stability of the equilibrium state, derived above, we imagine a system that has remained at this equilibrium state for all values of t less than or equal to zero, and that is perturbed from this equilibrium by small inoculations at values of t equal to

zero. Let $f(t)$, $j(t)$, and $a(t)$ denote these small perturbations from the equilibrium prey, juvenile predator, and adult predator densities, respectively. That is, $F(t)$, $J(t)$, and $A(t)$ are assumed to be described by the equalities

$$F(t) = F^* - f(t); \quad J(t) = J^* - j(t); \quad A(t) = A^* - a(t). \tag{A10}$$

Using these equalities, we can substitute the variables F, J, and A in the system of equations (A3)–(A5) and derive a system of differential equations governing the dynamics of $f(t)$, $j(t)$, and $a(t)$. As these perturbations are assumed to be small, we may neglect all terms of second and higher order, an assumption that leads to the following linear system of equations:

$$\frac{df}{dt} = (1 - 2F^*)f(t) - \frac{(J^* + A^*)}{(1 + \beta F^*)^2}f(t) - \frac{F^*}{(1 + \beta F^*)}[j(t) + a(t)], \tag{A11}$$

$$\begin{aligned}\frac{dj}{dt} = {} & \frac{\epsilon A^*}{(1 + \beta F^*)^2}f(t) - \frac{\epsilon A^*}{(1 + \beta F^*)^2}f(t - \delta)e^{-\mu\delta} \\ & + \frac{\epsilon F^*}{(1 + \beta F^*)}a(t) - \frac{\epsilon F^*}{(1 + \beta F^*)}a(t - \delta)e^{-\mu\delta} - \mu j(t),\end{aligned} \tag{A12}$$

and

$$\frac{da}{dt} = \frac{\epsilon A^*}{(1 + \beta F^*)^2}f(t - \delta)e^{-\mu\delta} + \frac{\epsilon F^*}{(1 + \beta F^*)}a(t - \delta)e^{-\mu\delta} - \mu a(t). \tag{A13}$$

We next assume that the quantities $f(t)$, $j(t)$, and $a(t)$ have solutions of the form

$$f(t) = f_0 e^{\lambda t}; \quad j(t) = j_0 e^{\lambda t}; \quad a(t) = a_0 e^{\lambda t}, \tag{A14}$$

with factors λ (defined below) and initial perturbations at $t = 0$ (f_0, j_0, and a_0). Substitution of these equalities and the expressions (A6)–(A8) for the equilibrium densities F^*, J^*, and A^* into the equations (A11)–(A13) leads, after lengthy manipulations, to a system of algebraic equations that can be expressed in matrix form as:

$$\begin{pmatrix} -\left(2\dfrac{\mu}{\epsilon e^{-\mu\delta} - \beta\mu} - \dfrac{\mu(\beta + 1)}{\epsilon e^{-\mu\delta}} + \lambda\right) & -\dfrac{\mu}{\epsilon e^{-\mu\delta}} & -\dfrac{\mu}{\epsilon e^{-\mu\delta}} \\ (\epsilon e^{-\mu\delta} - \mu(\beta + 1))(1 - e^{-\lambda\delta}e^{-\mu\delta}) & -(\mu + \lambda) & -(\mu e^{-\lambda\delta} - \mu e^{-\lambda\delta} - \mu e^{\mu\delta}) \\ (\epsilon e^{-\mu\delta} - \mu(\beta + 1))e^{-\lambda\delta}e^{-\mu\delta} & 0 & -(\mu + \lambda - \mu e^{-\lambda\delta}) \end{pmatrix} \begin{pmatrix} f_0 \\ j_0 \\ a_0 \end{pmatrix} = 0. \tag{A15}$$

By equating the determinant of the matrix occurring in this equation with zero, we obtain the following characteristic equation, which governs the stability of the scaled population model:

$$\lambda^2 + (2C_1 - C_2 + \mu)\lambda + (2C_1 - 2C_2 + 1)\mu = (\lambda + 2C_1 - C_2)\mu e^{-\lambda\delta}, \tag{A16}$$

in which

$$C_1 = \frac{\mu}{\epsilon e^{-\mu\delta} - \beta\mu}$$

and

$$C_2 = \frac{\mu(\beta + 1)}{\epsilon e^{-\mu\delta}}.$$

If all the roots λ of the characteristic equation (A16) have a negative real part, any combination of small perturbations f_0, j_0, and a_0 at time $t = 0$ will only become smaller over time and the internal equilibrium will be locally stable.

Stability diagrams of the scaled population model were obtained by numerical investigations of the characteristic equation (A16). Figure 1 shows one example of such a stability diagram in which we have used the relations between the compound parameters of the scaled model and the parameters of the individual-based simulation model (see table A1) to translate the resulting diagram back in terms of the parameters of our individual-based simulation model.

LITERATURE CITED

Addicott, J., J. M. Aho, M. F. Antolin, D. K. Padilla, J. S. Richardson, and D. A. Soluk. 1987. Ecological neighborhoods: scaling environmental patterns. Oikos 49:340–346.

Browman, H., S. Kruse, and W. J. O'Brien. 1989. Foraging behavior of the predacious cladoceran, *Leptodora kindtii,* and escape responses of prey. Journal of Plankton Research 11:1075–1088.

Caraco, T., and R. G. Gillespie. 1986. Risk-sensitivity: foraging mode in an ambush predator. Ecology 67:1180–1185.

Caswell, H. 1989. Matrix population models. Sinauer, Sunderland, Mass.

Chesson, P. L. 1981. Models for spatially distributed populations: the effect of within-patch variability. Theoretical Population Biology 19:288–325.

Cloarec, A. 1990. Factors influencing the choice of predatory tactics in a water bug, *Diplonychus indicus* Venk. and *Rao* (Heteroptera: Belostomatidae). Animal Behaviour 40:262–271.

Cushing, J. M., and M. Saleem. 1982. A predator-prey model with age-structure. Journal of Mathematical Biology 14:231–250.

de Roos, A. M., J. A. J. Metz, E. Evers, and A. Leipoldt. 1990. A size-dependent predator-prey interaction: who pursues whom? Journal of Mathematical Biology 28:609–643.

de Roos, A. M., E. McCauley, and W. G. Wilson. 1991. Mobility versus density limited predator-prey dynamics on different spatial scales. Proceedings of the Royal Society of London B, Biological Sciences 246:117–122.

Formanowicz, D. R., and P. J. Bradley. 1987. Fluctuations in prey density: effects on the foraging tactics of solopendrid centipedes. Animal Behaviour 35:453–461.

———. 1989. Foraging behavior of matamata turtles: the effects of prey density and conspecifics. Herpetologica 45:61–67.

Gilbert, C. 1989. Visual determinants of escape in tiger beetle larvae (Cicindelidae). Journal of Insect Behavior 2:557–574.

Gurney, W. S. C., R. M. Nisbet, and J. Lawton. 1983. The systematic formulation of stage-structured models. Journal of Animal Ecology 52:479–495.

Gurney, W. S. C., E. McCauley, R. M. Nisbet, and W. W. Murdoch. 1990. The physiological ecology of *Daphnia:* a dynamic model of growth and reproduction. Ecology 71:716–732.

Hanski, I., and M. Gilpin. 1991. Metapopulation dynamics: brief history and conceptual domain. Biological Journal of the Linnean Society 42:3–16.

Harper, D. G., and R. Blake. 1988. Energetics of piscivorous predator-prey interactions. Journal of Theoretical Biology 134:59–76.

Hassell, M. P., H. Comins, and R. M. May. 1991. Spatial structure and chaos in insect population dynamics. Nature (London) 353:255–258.

Hastings, A. 1990. Spatial heterogeneity and ecological models. Ecology 71:426–428.

Hastings, A., and D. Wollkind. 1982. Age structure in predator-prey systems. I. A general model and a specific example. Theoretical Population Biology 21:44–56.

Hogeweg, P. 1988. Cellular automata as a paradigm for ecological modeling. Applied Mathematics and Computation 27:81–100.

Huston, M., D. DeAngeles, and W. Post. 1988. New computer models unify ecological theory. BioScience 38:682–691.

Jones, K. B., and W. G. Whitford. 1989. Feeding behavior of free roaming *Mastcophis flagellum:* an efficient ambush predator. Southwestern Naturalist 34:460–467.

Kareiva, P. 1990. Population dynamics in spatially complex environments: theory and data. Philosophical Transactions of the Royal Society of London B, Biological Sciences 330:175–190.

Kopelman, R. 1988. Fractal reaction kinetics. Science (Washington, D.C.) 241:1620–1626.

Larralde, H., P. Trunfio, S. Havlin, H. E. Stanley, and G. H. Weiss. 1992. Territory covered by N diffusing particles. Nature (London) 355:423–426.

McCauley, E., W. W. Murdoch, R. M. Nisbet, and W. S. C. Gurney. 1990. The physiological ecology of *Daphnia:* development of a model of growth and reproduction. Ecology 71:703–715.

Metz, J. A. J., and A. M. de Roos. 1992. The role of physiologically structured population models within a general individual-based modeling perspective. Pages 88–111 *in* D. L. DeAngeles, L. A. Gross, and T. G. Hallam, eds. Populations, communities, and ecosystems: a perspective from modeling at the level of individual organisms. Chapman & Hall, London.

Metz, J. A. J., and O. Diekmann, eds. 1986. The dynamics of physiologically structured populations. Springer, Berlin.

Murdoch, W. W., E. McCauley, R. M. Nisbet, W. S. C. Gurney, and A. M. de Roos. 1992. Individual based models: combining testability and generality. Pages 18–35 *in* D. L. DeAngeles, L. A. Gross, and T. G. Hallam, eds. Populations, communities, and ecosystems: a perspective from modeling at the level of individual organisms. Chapman & Hall, London.

Murray, J. D. 1989. Mathematical biology. Biomathematics. Vol. 19. Springer, Berlin.

Neill, W. E. 1981. Impact of *Chaoborus* predation upon the structure and dynamics of a crustacean zooplankton community. Oecologia (Berlin) 48:164–177.

Nisbet, R. M., and W. S. C. Gurney. 1982. Modeling population fluctuations. Wiley, New York.

Nisbet, R. M., W. S. C. Gurney, W. W. Murdoch, and E. McCauley. 1989. Structured population models: a tool for linking effects at individual and population level. Biological Journal of the Linnean Society 37:79–99.

Nisbet, R. M., C. J. Briggs, W. S. C. Gurney, W. W. Murdoch, and A. Stewart-Oaten. 1993. Two patch metapopulation dynamics. *In* S. A. Levin, T. Powell, and J. H. Steele, eds. Patch dynamics in terrestrial, freshwater, and marine ecosystems. Lecture notes in biomathematics. Springer, Berlin (in press).

Okubo, A. 1980. Diffusion and ecological problems: mathematical models. Biomathematics. Vol. 10. Springer, Berlin.

Pacala, S. W., and J. A. Silander. 1985. Neighborhood models of plant population dynamics. I. Single species models of annuals. American Naturalist 125:385–411.

Power, H. W. 1980. Foraging behavior of mountain bluebirds with emphasis on sexual foraging differences. Ornithological Monographs 28:1–72.

Reeve, J. D. 1990. Stability, variability, and persistence in host-parasitoid systems. Ecology 71:422–426.

Savino, J. F., and R. A. Stein. 1989. Behavior of fish predators and their prey: habitat choice between open water and dense vegetation. Environmental Biology of Fishes 24:287–294.

Stanley, H. E. 1971. Introduction to phase transitions and critical phenomena. International series of monographs of physics. Oxford University Press, Oxford.

Taylor, A. D. 1990. Metapopulations, dispersal, and predator-prey dynamics: an overview. Ecology 71:429–433.

Turing, A. 1952. The chemical basis of morphogenesis. Philosophical Transactions of the Royal Society of London B, Biological Sciences 237:37–72.

Wilson, W. G. 1989. Quenched dilution tri-critical point in the Q = 3, D = 3 Potts model. Physics Letters Section A: General Atomic and Solid State Physics 137:398–402.

Wilson, W. G., and W. Laidlaw. 1992. Microscopic based fluid-flow invasion simulations. Journal of Statistical Physics 66:1165–1176.

Wilson, W. G., A. de Roos, and E. McCauley. 1993. Spatial instabilities within the diffusive Lotka-Volterra system: individual based simulation results. Theoretical Population Biology 43:91–127.

Associate Editor: Peter Chesson