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Mobility versus density-limited predator–prey dynamics on different spatial scales

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SUMMARY

We investigate the dynamics of a predator–prey model that explicitly accounts for the spatial position and the movement behaviour of individual prey and predators, and does not assume the law of mass action. We show that limited individual mobility greatly reduces fluctuations in total density, although average densities and vital rates are virtually unaffected. We analyse the dynamics of patterns in the spatial distribution of prey and predator, which are generated by the model, and show that population dynamic observations at different spatial scales depend on the characteristic scale imposed by the individual biology.

1. INTRODUCTION

Most, if not all, models describing the interactions between biological populations are based on the principle or law of 'mass action' (Metz & Diekmann 1986; Metz & De Roos 1991), thus assuming that individuals mingle quickly and randomly and hence potentially interact with a representative part of their own or other populations. More specifically, if F and C denote the total number of prey and predators present within a certain area, respectively, and if at every time all prey individuals are vulnerable to predation by all predator individuals, the law of mass action assumption results in the number of prey eaten per unit time (predation rate) being proportional to the product of F and C:

$$\text{predation rate} \sim FC. \tag{1}$$

In mass action models, interaction rates are hence completely determined by the density of individuals.

Other branches of science, e.g. chemistry and astrophysics, have recognized that the dynamics exhibited by mass action models are one extreme of a spectrum (usually referred to as 'reaction-limited' kinetics), in which the diffusion rates of the reactants (and hence their mobility) are high, but the reaction probability on encounter is limited. At the other end of the spectrum, usually referred to as 'diffusion-limited' kinetics, the diffusion of reactants controls the overall interaction rates, as the reaction probability on encounter is very high (Noyes 1961; Rice 1987; Hoshen & Kopelman 1976; Kopelman 1988; Argyrakis & Kopelman 1990). The mass action law, as exemplified by (1), is always valid for reaction-limited processes, but extreme deviations from it have been found both theoretically and experimentally in the diffusion-limited case (Kopelman 1988; Argyrakis & Kopelman 1990).

Despite the fact that biological populations are composed of individuals with limited mobility, few studies (Kareiva & Odell 1987) have examined how such limitation, possibly leading to deviations from the mass action law, might influence the dynamics of interacting, biological populations. In this paper we present some preliminary results on the influence of limited mobility on the dynamics of interacting predator and prey populations. By using individual-based, stochastic simulations we show that limited mobility greatly reduces the fluctuations in predator and prey densities, but equilibrium densities and overall population rates are virtually unaffected. Our results also show that population dynamical features are tied to a characteristic spatial scale imposed by individual biology. We discuss how our results relate to existing theory on the dynamics of populations in non-homogeneous environments.

2. MODEL DESCRIPTION

To incorporate limited individual mobility, which automatically leads to only local individual interactions and possibly to the violation of the mass action law, into a model of an interacting prey and predator population, we have to use an individual-based model that keeps track of every single prey and predator and their positions within the arena in which they interact. In our model the spatial domain is represented by a square lattice of 128×128 possible individual positions or sites. Every site is either empty or occupied by at most one prey and one predator individual. Movement within the spatial domain and population dynamics are modelled as discrete time processes. We have

deliberately kept the rules governing the dynamic processes at the individual level as simple as possible to focus entirely on the effects of limited mobility.

Prey individuals are assumed stationary and do not move, whereas predator movement between sites is either homogeneous, that is, individuals are redistributed randomly over the entire lattice, or diffusive, entailing random movement to one of four neighbouring sites. In the latter case, if the destination site is already occupied by a predator the individual does not move at all, and if more individuals select the same destination site only one, randomly selected, candidate is allowed to move. Diffusive movement limits the mobility of the predator in contrast to homogeneous movement.

At every time step after the predators have moved, the following sequence of population dynamic processes takes place: First, all prey individuals reproduce with probability P_{rg} one offspring to one of the four neighbour sites. If this site is already occupied by prey, the newborn is aborted. This rule mimics a density-dependent growth process. As a result of predator movement and prey reproduction, a predator individual may end up in the same site as a prey individual. If the predator is not currently handling a previous meal, it will eat the prey. Upon eating, the predator cannot eat for the next T_h time steps, while it spends its time consuming. This rule implements a mechanism comparable with a predator handling time. After a predator has eaten F meals it reproduces R_{mh} (<4) offspring. Predator reproduction is assumed density independent: if a randomly chosen destination out of the four neighbour sites is occupied, a nearby empty site is found. Finally, every time step a random fraction P_d of the predators dies.

Population densities and the number of births and deaths are then measured and used to calculate per capita vital rates, averaged over the total population.

The rules governing the processes at the individual level are reminiscent of a Lotka–Volterra type of predator–prey interaction with logistic prey growth, a type II predator functional response, and a constant predator conversion efficiency and death rate ('Rosenzweig–MacArthur' model). The following set of differential equations constitutes the simplest mathematical description of such a system:

$$\left.\begin{aligned} \mathrm{d}F/\mathrm{d}t &= r_m F\left(1-\frac{F}{K}\right)-\frac{aF}{1+a\tau_h F}C \\ \mathrm{d}C/\mathrm{d}t &= \epsilon\frac{aF}{1+a\tau_h F}C-\mu C, \end{aligned}\right\} \qquad (2)$$

where F and C are the densities of prey and predator, r_m and K represent the maximum average growth rate and carrying capacity of the prey population, and a, τ_h, ϵ and μ represent the attack rate, the handling time, the conversion efficiency and the random death rate of the predator, respectively.

The properties of the model, described by (2), are very well understood (see, for example, Freedman 1987). In a separate paper (McCauley *et al.* 1991), we compare in detail our stochastic simulation model, in which both prey and predator move homogeneously, with the analytical model (2), and show a general qualitative and quantitative agreement. More specifically, the analytical model (2) exhibits large amplitude oscillations for low values of μ and high values of K (the 'paradox of enrichment' (Rosenzweig 1971)), as does our simulation model for low values of P_d. In the following sections we will report the effects of limited predator mobility on the behaviour of the simulation model.

3. AVERAGE DENSITIES, VITAL RATES, DYNAMICS

Figure 1 illustrates the effect of limited predator mobility on average densities and average vital rates. The dynamics of the model were simulated over 500 time steps for a large number of different parameter combinations. Simulations were done with both homogeneous and diffusive predator movement. Total prey and predator densities and the number of births and deaths were subsequently averaged over the last 250 time steps to exclude transient behaviour. (Although in principle time averages cannot be equated with statistical averages over many different runs when

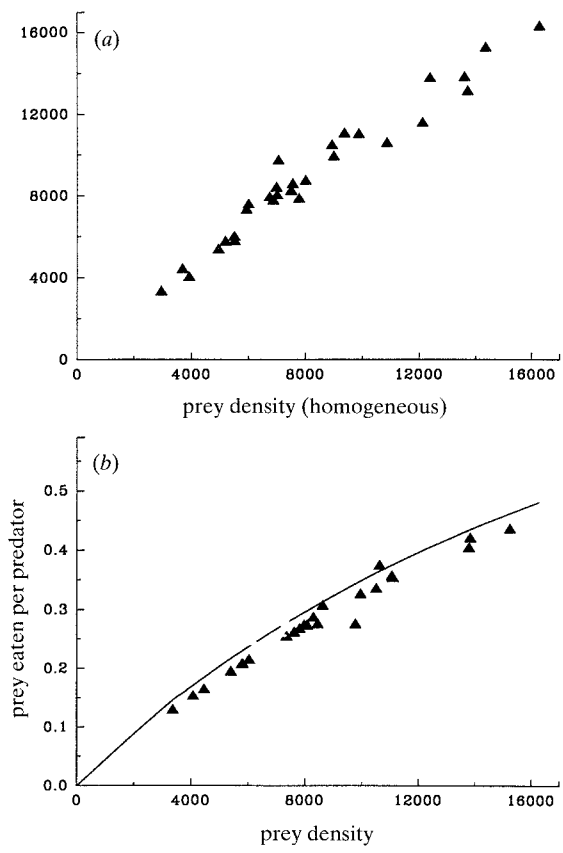

Figure 1. The effect of limited predator mobility on average densities and vital rates. (*a*) Average prey density with diffusive predator movement against the average density with homogeneous predator movement measured over the entire spatial domain. (*b*) Average predator functional response as a function of average prey density in the case of diffusive predator movement (symbols). The solid line represents the type II functional response curve ($F(x) = ax/(1+a\tau_h x)$), fitted to the equivalent data points in the case of homogeneous predator movement.

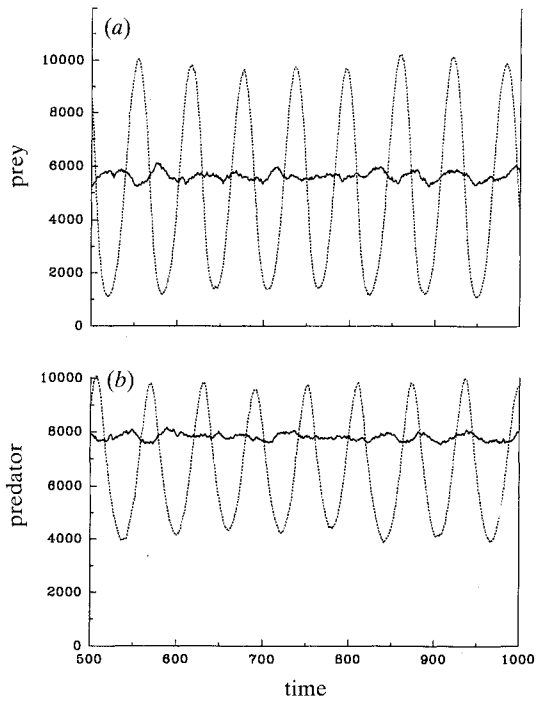

Figure 2. Dynamic behaviour of the number of (*a*) prey and (*b*) predators in the entire spatial domain in case of homogeneous (dotted line) and diffusive (solid line) predator movement, respectively. Parameter values: $P_{rg} = 0.75$, $T_h = 2$, $R_{mh} = 2$, $F = 4$ and $P_d = 0.06$.

dynamics are strongly oscillatory, the difference does not seem to influence our results.)

The transition from homogeneous to diffusive predator movement (i.e. the introduction of mobility limitation) does not seem to produce significant differences in average prey (figure 1*a*) or predator densities (not shown) in the entire spatial domain. Perhaps more surprisingly, there are also no major changes detected in the predators' functional response (i.e. number of prey eaten per predator) (figure 1*b*) when individual predators move diffusively, nor in prey average growth rate (not shown). Arguably the number of prey eaten per predator is slightly lower in the case of diffusive predator movement, but in general we conclude that the observed interaction rates do not significantly violate the law of mass action when calculated as averages over the total population.

The dynamics of the predator–prey model with limited predator mobility are, however, very different from its density-limited counterpart, as is illustrated in figure 2. With homogeneous predator movement and a high predator death rate, P_d, the simulated prey and predator densities are more or less constant over time. The coefficients of variation (cvs: standard deviation/average value) of these time series are usually in the order of 0–0.05, as a result of stochastic influences. Decreasing the predator death rate leads to a sudden and large increase (usually more than one order of magnitude) in the cvs of the prey and predator densities over time. Both populations then display large amplitude cycles, similar to oscillations found in the predator–prey model described by (2) (i.e. limit cycles (McCauley *et al.* 1991)). When predators move diffusively, the sudden increase in cv and the concomitant cycles in total density do not occur. For all parameter combinations we have used, the dynamics resemble a noisy signal (with relatively small deviations) around a roughly constant number of prey and predators (figure 2). The limitation of mobility inherent in predator biology has obviously reduced the predator–prey fluctuations.

4. SPATIAL HETEROGENEITY AND ITS DYNAMICS

The explanation of the apparent stabilization should be found in the spatial interaction of prey and predators in our model. In the case of diffusive predator movement, the spatial distribution of both prey and predator can be shown to be clustered via a variety of methods, e.g. visual inspection, spatial correlation functions (Cliff & Ord 1981) and hierarchical analysis of variance (Moellering & Tobler 1972). However, as we have shown in the previous section, this heterogeneity in spatial distribution does not lead to significant changes in total densities and population level average rates, nor to the introduction of obvious density dependence in these rates (for instance, 'pseudo-interference' (Free *et al.* 1977)). Such mechanisms are invoked by classical theories to explain reduced population fluctuations in spatially heterogeneous environments (Hassell & May 1973; Free *et al.* 1977).

Visual inspection of the simulation results also shows that the heterogeneous distributions of prey and predator change continuously over time. To gain more insight into the dynamics of the spatial pattern we have studied the fluctuations in (local) prey and predator densities at different spatial scales. cvs and autocorrelation functions were computed for the time series of prey and predator density within a randomly positioned, square section (a window) of the total lattice. By using differently sized windows, a relative measure of variability in prey and predator density was obtained as a function of the spatial scale of observation. Figure 3 shows the results of this procedure for the time series of prey density obtained from the simulation presented in figure 2.

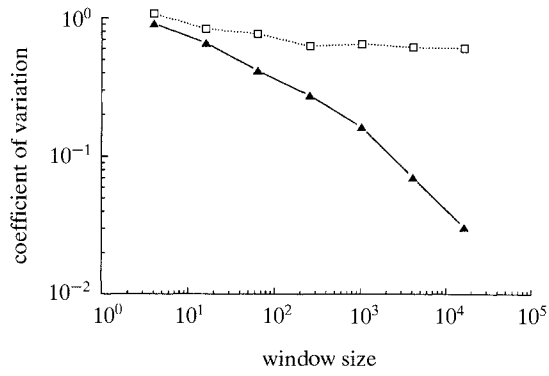

Figure 3. Variability in prey density, expressed in terms of the coefficient of variation of the simulated time series, as a function of the spatial scale of observation. Homogeneous predator movement (open squares), diffusive predator movement (solid triangles). Parameter values as in figure 2.

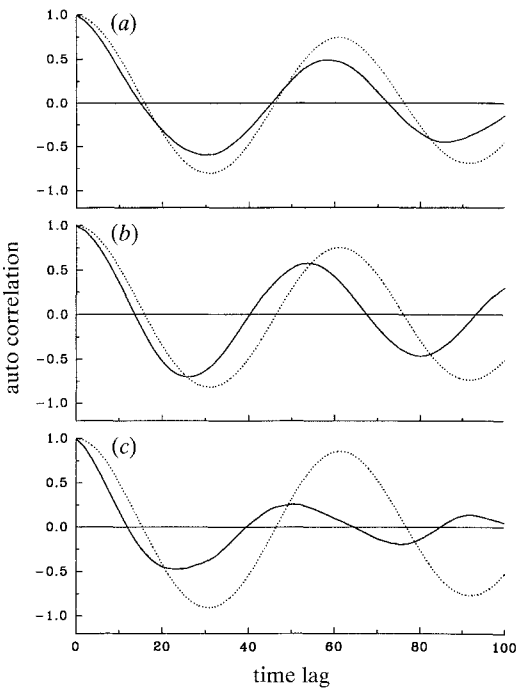

Figure 4. Temporal autocorrelation functions on different spatial scales of observation for the same time series of prey density as used in figure 3. Homogeneous predator movement (dotted line), diffusive predator movement (solid line). (*a*) 16×16 sites; (*b*) 32×32 sites; (*c*) 128×128 sites.

On small spatial scales, the variability in prey density over time under homogeneous and diffusive predator movement is roughly similar (figure 3). On larger spatial scales the two deviate more and more, with the variability under diffusive predator movement decreasing approximately in proportion to the square root of the area of the observation window (figure 3). The transition between these two regimes seems to be rather abrupt.

Figure 4 shows examples of the autocorrelation functions, which were constructed from the time series of prey and predator density within the observation windows. The periodicity in the density fluctuations under homogeneous and diffusive predator movement are also similar on the smaller spatial scales (figure 4). However, an increase in window size leads to a dampening of the autocorrelation function, implying that, in addition to exhibiting smaller fluctuations, the dynamics on larger spatial scales also show less temporal correlation.

The limitation of predator mobility therefore seems to stabilize the dynamics 'statistically' (as opposed to stabilization by a biological mechanism in the interaction), as the reduction in fluctuations is a scale-related phenomenon, and fluctuations on small spatial scales are invariably large. The transition point, where the cv in the case of limited mobility starts to deviate from the cv in the case of density-limited dynamics (figure 3), can be adopted as a definition of the 'natural' or 'characteristic' spatial scale of the system (cf. the correlation length (Huang 1987; Grimmett 1989)). This characteristic spatial scale is imposed on the spatial domain by the limited predator mobility. On smaller scales the dynamics of prey and predators are reminiscent of a homogeneously mixing, density-limited predator–prey interaction. The limited movement, however, leads to only weak coupling between parts of the spatial domain that are far apart in terms of the characteristic scale. Their dynamics are hence out of phase. The dynamics in total population sizes result as a superposition of the dynamic signals from all these weakly correlated parts, with the phase differences obviously counteracting fluctuations.

Given this stabilization hypothesis, scaling arguments (Huang 1987; Grimmett 1989) can be exploited to predict that, above the characteristic scale, the observed cv under diffusive predator movement decreases with the square root of the area of the observation window. (Briefly, scaling theory conjectures that a quantity of dimension $(\text{length})^D$ is proportional to ξ^D, with ξ the characteristic length scale of the system, or, equivalently, that this quantity only changes with changes in the ratio between the unit of length and the characteristic length scale.) As we have hypothesized that the time series of the total number of individuals within a given observation window results as a superposition of the dynamic signals from regions that are far apart in terms of the characteristic spatial scale, it follows that the variance of this time series should be inversely proportional to the number of these, almost independent, regions within the window, i.e. to the quotient of window size and the characteristic spatial scale. (Note also that we have consistently used characteristic spatial scale to refer to an area measure.) We therefore predict that the cv under diffusive predator movement varies as:

$$(\text{window size}/\text{characteristic spatial scale})^{-\frac{1}{2}}.$$

Linear regression of cv against window size using the last five observations from figure 3 in case of diffusive movement yields a slope of -0.47 (standard error, 0.04), consistent with this prediction.

If we furthermore assume that the characteristic scale under homogeneous predator movement is proportional to the total lattice size (16384 sites), the same arguments imply that a relative estimate of the characteristic scale under diffusive movement can be obtained from:

$$16384 \times (\text{cv}_{\text{diffusive}}/\text{cv}_{\text{homogeneous}})^2, \qquad (3)$$

in which $\text{cv}_{\text{homogeneous}}$ and $\text{cv}_{\text{diffusive}}$ are the coefficients of variation of the time series of total population size in the entire spatial domain in the case of homogeneous and diffusive predator migration, respectively.

Equation (3) allows us to study how the characteristic scale changes with increasing predator mobility. An increase in predator mobility can be modelled by increasing the number of diffusive steps taken by the predators per unit time, whereas all population dynamic processes and interactions only take place once every time step. The results show a roughly linear increase of the characteristic scale with predator mobility (see figure 5).

To test our idea that the observed characteristic scale is set by the restricted movement behaviour of the predator, we derived theoretically the relation between the number of diffusive steps taken per unit time and the average area influenced by the predator over the

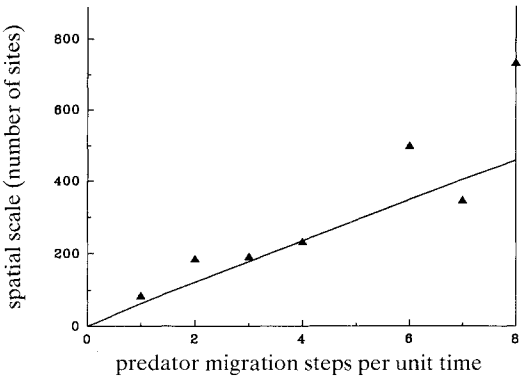

Figure 5. Estimated, relative measure of the characteristic spatial scale (symbols) and average lifetime range of the predator (solid line) as a function of the number of diffusive migration steps taken by the predator per unit time. See text for details.

course of its lifetime (referred to below as 'average lifetime range'). If unhampered by conspecifics, an individual predator performs a two-dimensional random walk from its position at birth. Given this situation, we calculated the probability distribution for an individual predator taking M diffusive steps per unit time, to die after N time steps at a distance D from its place of birth. This distribution was subsequently used to calculate the total number of sites within the average distance travelled between birth and death, as a measure of the 'average lifetime range' of the predator (the probability distribution for the position (x, y) after S steps taken during a two-dimensional random walk can simply be expressed as the product of the binomial distributions, characterizing two independent, one-dimensional random walks, by choosing the lines $y = x$ and $y = -x$ as a new coordinate system). Figure 5 shows that both the characteristic spatial scale and the average lifetime range exhibit the same, linear relation with the number of diffusive steps taken per unit time. (Note that equation (3) yields a relative measure of the characteristic spatial scale and that the agreement between the absolute values of the two quantities should not be taken as significant.) The agreement lends support to our claim that the characteristic spatial scale is determined by the individual behaviour.

5. DISCUSSION AND CONCLUSIONS

The model results reveal a very important aspect of the individual behaviour that strongly influences the population dynamics. The behaviour of individuals (e.g. movement and interactions) automatically imposes a characteristic scale on the spatial domain. Observations on scales smaller or larger than this characteristic scale are entirely different: the dynamics on small spatial scales are largely determined by the biological aspects and characteristics of the interaction between the individuals, and resemble the behaviour of density-limited models. With increasing spatial scale, the dynamic signals of smaller-scale regions, which are only weakly correlated with each other, are superimposed to yield dynamic phenomena that bear less and less resemblance to the smaller scale dynamics, as a result of the averaging influence of lumping weakly correlated subdomains. In mass-action based models with homogeneous mixing of individuals, the range of individuals is 'infinite', and all observations hence pertain to a spatial scale much smaller than the characteristic scale. Interpretation of population dynamic phenomena therefore requires insight into the relation between the spatial scale of observation and the spatial scale set by the individual biology.

The absence of mass action implies that there is significant variability in local prey and predator density as perceived by the individual members of both populations. In other words, there is spatial heterogeneity at the individual level. It is very remarkable that this fact does not significantly influence the static properties of the interaction rates, when assessed at the level of the total population, while having such a profound effect on dynamics.

The relation between spatial heterogeneity and stability has been a central issue in predator–prey theory for a long time. The combination of limited dispersal, as a synchronizing mechanism, and asynchrony in dynamics between different regions is a well-known stabilizing mechanism of global dynamics, when local dynamics are unstable (see, among others, Crowley 1981; Hassell & May 1988; Reeve 1988; Taylor 1990). Broadly speaking, three classes of models have been used to study and corroborate the mentioned relation: (i) Metapopulation models (Murdoch & Oaten 1975; Crowley 1981; Diekmann *et al.* 1988, 1989; Reeve 1988, 1990; Freedman & Takeuchi 1989*a*, *b*; Hastings 1990; Ives 1991) model a collection of subpopulations in distinct environmental patches, linked by individual dispersal; (ii) Diffusion equation models (Segel & Jackson 1972; Levin 1974; Okubo 1980; Hastings 1990) use diffusion terms to describe dispersal of individuals throughout a continuous spatial domain, in conjunction with terms describing the population dynamics locally; (iii) A class of models that describes phenomenologically the dynamics within a continuous spatial domain of a single, non-randomly distributed prey and predator population (Hassell & May 1973; Murdoch & Stewart-Oaten 1989). A necessity for asynchrony between different regions and hence for global stability is some form of variability on the smallest spatial scale. In all these models, such variability is introduced by assuming *a priori*, for instance, environmental heterogeneity, a (fixed) aggregated distribution of prey or predator or the occurrence of stochastic, catastrophic events. In sharp contrast, the variability on the individual level in our model is very much a model result itself and is generated by the limited mobility of individuals.

Maybe more important than the apparent stabilization of global dynamics within the model is the generation of distinct spatial patterns that change over time. We hereby chose the population as our level of interest. Metapopulation and diffusion equation models have invariably assumed mass action type dynamics at the smallest spatial scale, either an individual patch (metapopulations) or a single location in space (diffusion equation models). Essentially, these models thus distinguish two separate types of individual

movement, taking place at very different spatial scales: (i) the individual mixing, necessary for the interactions within the local population; and (ii) long range, individual dispersal between various subpopulations. It is the stabilizing role of the latter, and the resulting patterns at the level of the metapopulation, that are investigated. Obviously, in these models there is no direct relation between the dynamics of a local (sub)population and its spatial distribution.

In the present study we investigated the generation of spatial patterns within a single population living in a continuous spatial domain, the dynamics of these patterns and their relation with the various population dynamical and movement processes, taking place at the level of the individual. Without assuming an *a priori* subdivision of the environment, or aggregative behaviour of prey or predator individuals, a patchy spatial distribution of both populations results. The temporal dynamics of this distribution were studied, using time series analysis at different spatial scales. New theoretical concepts, such as the characteristic spatial scale described here, and new methods seem necessary to gain more insight in this field. An in-depth analysis of the spatial patterns generated by the model is outside the scope of the current article and will be given in a future publication (Wilson *et al.* 1991).

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