ELSEVIER

Ecological Modelling 117 (1999) 179–202

ECOLOGICAL MODELLING

From pattern to practice: a scaling-down strategy for spatially explicit modelling illustrated by the spread and control of rabies

H.-H. Thulke [a,*], V. Grimm [a], M.S. Müller [a], C. Staubach [b], L. Tischendorf [a], C. Wissel [a], F. Jeltsch [a]

[a] *Department of Ecological Modelling, UFZ Centre for Environmental Research Leipzig-Halle, Permoserstraße 15, D-04318 Leipzig, Germany*

[b] *Federal Research Centre for Virus Diseases in Animals, Institute for Epidemiology, Seestr. 55, D-16868 Wusterhausen/Dosse, Germany*

Received 1 July 1998; accepted 20 October 1998

Abstract

A major problem in ecological modelling is finding the appropriate level of resolution when describing the processes and structures of ecological systems. When modelling basic ecological questions, as a rule the best approach is to ignore as much detail as possible in order to obtain general insights. However, for applied problems focusing in particular on ecological systems, there are no clear guidelines for identifying the most appropriate resolution in space, time and the detail of description. Spatially explicit modelling thus has to mainly rely on trial and error in scaling-up from modelling at the local scale to exploration of the model at the global scale. We demonstrate here a modelling strategy that takes the opposite approach: starting at the global scale, with a strategic model of minimum resolution, we proceed step by step to a model addressing applied questions. The strategic model is designed to reproduce a certain pattern observed in nature. As an example, we use the wave-like spreading pattern of rabies. The applied model addresses the question of whether rabies might persist in areas with a high proportion of foxes immunized by oral vaccination. As a consequence of our scaling-down strategy, the resolution of the applied model is not chosen a priori, but emerges from the step by step modelling strategy. During each step of model refinement, one module of the preceding model is described with a slightly increased resolution. This stepwise approach allows both a backward reference to the pattern reproduced by the strategic model and a cross-reference between the coarser and finer version of the module refined. The main potential of the scaling-down strategy is that it leads to efficient models in an efficient way, but since scaling-down is a complement to scaling-up approaches, it might also help to bridge the gap between theoretical and applied ecological modelling.

Keywords: Ecological modelling; Spatially explicit; Scales; Pattern; Fox; Rabies; Vaccination

* Corresponding author. Tel.: +49-341-2352038; fax: +49-341-2353500.
E-mail address: hanst@oesa.ufz.de (H.-H. Thulke)

0304-3800/99/$ - see front matter
PII: S0304-3800(98)00198-7

1. Introduction

Holling (1966) introduced the distinction between 'strategic' models, which are designed to tackle general questions without referring to concrete ecological situations and 'tactical' models, which by contrast address concrete problems but are not at all general (May, 1973). The common feature of all models is thus to sacrifice something: strategic models "sacrifice detail for generality" (Levins, 1966), whereas tactical models do the opposite. Although this classification of models may be useful for demonstrating the principle that modelling always means sacrificing *something*, it provides no detailed hints as to what could and should be sacrificed (Grimm et al., 1996b). With strategic models, the motto 'sacrifice detail for generality' suggests that literally every detail should be sacrificed if only the model is general. This approach leads to models which produce probable explanations. However, these explanations are trend-setting hints but usually hard to test (Peters, 1991).

On the other hand, tactical models addressing applied problems follow no clear guideline concerning what aspects of the real system should be considered in the model (Nisbet and Gurney, 1982). Consequently, tactical models are often loaded with so much detail that (despite being otherwise well-designed) they are unwieldy and hard to investigate. Although the modules of tactical models often burrow their structure from strategic models, theoretical ecology, i.e. the conceptual framework which emerged from strategic models, seems not to be very helpful for designing tactical models.

In this paper we will show that this is not necessarily the case. We will demonstrate how in a certain research project on the spread and control of rabies we proceeded step-by-step from a strategic model to a tactical model. The theoretical question addressed was pattern formation in the spread of rabies, while the practical, applied problem was the control of rabies via oral vaccination.

Pattern formation in ecological systems is a current issue in ecological theory (Levin, 1992; Sato and Iwasa, 1993; Jeltsch and Wissel, 1994; Ratz, 1995). Both the process of pattern formation itself (Paine and Levin, 1981; Thiery et al., 1995; Jeltsch et al., 1996; Maron and Harrison, 1997) and the impact of given patterns on ecological processes (Silvertown, 1992; Glenn and Collins, 1993; Reichman, 1993; Cain et al., 1995; Kareiva and Wennergren, 1995; Tackmann et al., 1998) have a high potential for enabling a better understanding of ecological systems. Typical examples of pattern forming processes in ecological systems include invasions of populations and the spread of diseases (Hengeveld, 1989; Jeltsch et al., 1992). Reaction-diffusion models have proved to be a suitable method to describe and understand these pattern-forming processes on biogeographical scales (Skellam, 1951; Lubina and Levin, 1988; Okubo et al., 1989). However, the aggregation of the dispersal process in a diffusion coefficient inevitably ignores details of the spreading mechanism, such as the movement of individuals in a heterogeneous landscape (Lima and Zollner, 1996; Schippers et al., 1996; Tischendorf, 1997) or rare infection events in a spatially explicit context (Jeltsch et al., 1997a). However, these details are decisive in applied issues, for example in the evaluation of control mechanisms of invading species or spreading diseases. What we need in order to evaluate the efficiency of management or control measures (which are usually restricted to local scales) is a modelling strategy resulting in models that link regional patterns to local practice and *vice versa*.

A good example for which such a modelling strategy would be extremely useful is the spread of rabies where the interaction of processes on different scales causes a complex spatio-temporal pattern (Sayers et al., 1985; Jeltsch et al., 1997a). The typical wave-like spreading pattern of the disease on a regional scale (Toma and Andral, 1977; Sayers et al., 1977; see Fig. 1) is successfully described by a set of reaction-diffusion equations (Källen et al., 1985; Murray et al., 1986; Yachi et al., 1989; Mollison, 1991). The basic message of these models is that the interaction of local cycles consisting of infections, population reduction and recovery, with the spatial transmittance of the disease over a certain distance (described by a diffusion process) is sufficient to explain the typical epidemiological pattern of damped oscillations

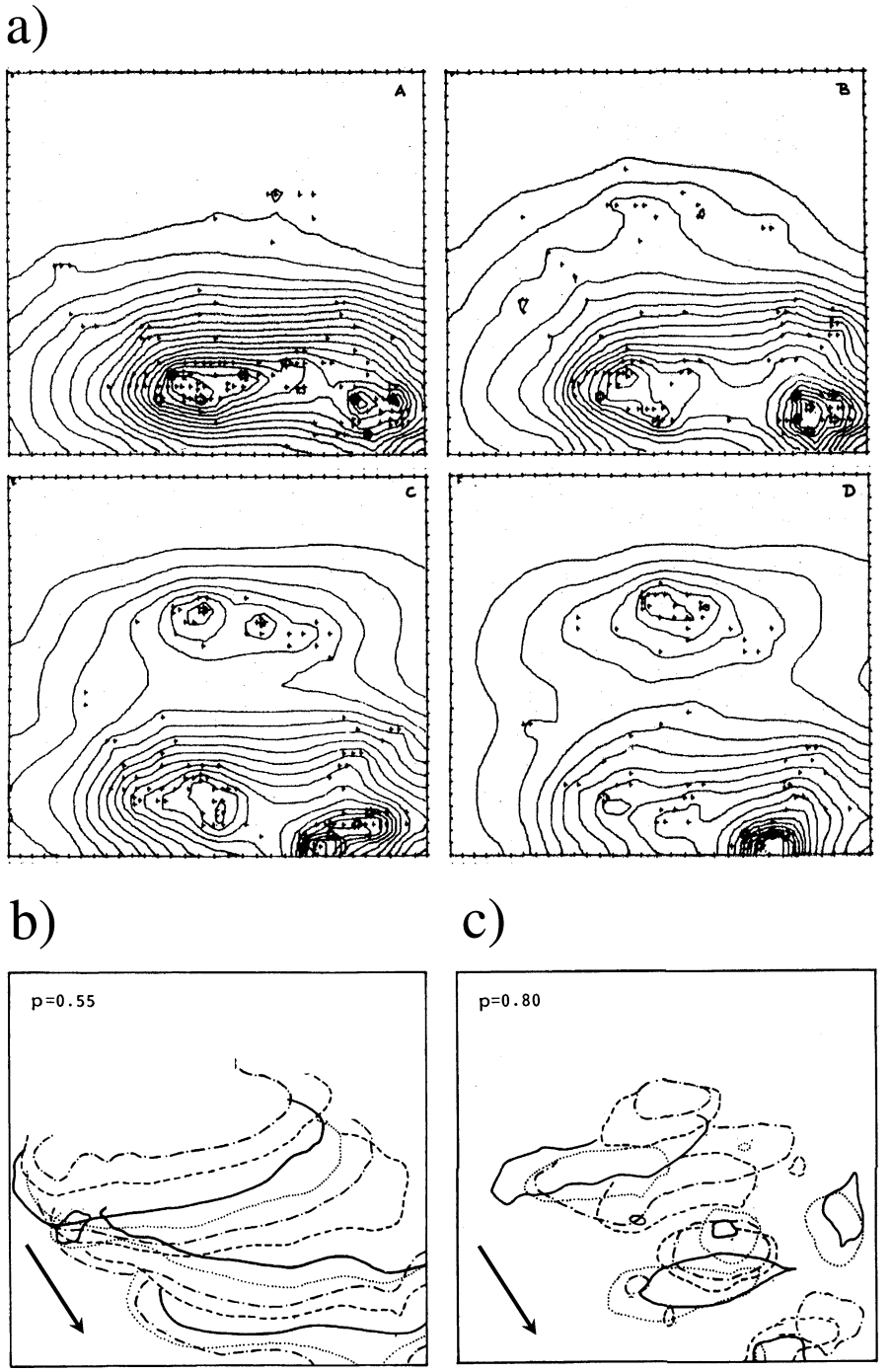

Fig. 1. Spatial rabies pattern based on approximately 3000 quarterly confirmed rabies cases from a 133 × 133 km study area in the Land Baden-Württemberg, Germany (after Sayers et al., 1977): (a), Smoothed density of case occurrence of rabies for four successive 3-month periods. The lowest contours indicate the global wave progression; the local, irregular high densities indicate individual disease foci; (b–c), Quarterly probability contour maps from autumn 1964–1967. Increasing the probability levels (P) the respective figures range from a more global to a more focal view of the epidemic. (b) Whereas at the broad scale the wave appears as a closed, uniform front; and (c) this front breaks up into isolated foci when viewed at the fine scale.

on regional scales and of recurrent fluctuations of rabies cases on local scales. However, the diffusion approach does not allow for a systematic analysis of the pattern-forming processes on small spatial and temporal scales, e.g. the spread of rabies by individual dispersing foxes. Yet it is precisely this level of detail which needs to be understood in order to develop models as an aid to management (Perrins et al., 1993; Jeltsch et al., 1997a). Consequently, individual-based models of detailed fox behavior on small spatial and temporal scales have been suggested as an alternative approach (Berger, 1976; David et al., 1982; Voigt et al., 1985; Smith and Harris, 1991). Such models have been successful in describing rabies outbreaks and control in small areas (Smith, 1995), but the number of individuals and the amount of data that can be processed restricts the spatial and temporal scale that can be modeled. Therefore, the model validation against any large-scale pattern (i.e. damped oscillation) would be possible only with an extrapolation in space and/or over time. However, such an attempt towards confidence is trapping if the necessary assumption of solely scale-invariant dependencies between pattern forming processes is violated as it is in the case of rabies spread (see Jeltsch et al., 1997a). Additionally, modelling the entire life history of each individual fox in time and space generates an enormous complexity of information that necessarily limits the interpretation of model results, i.e. identifying the details of the model essential for a particular result would be rather complicated as well as drawing more general conclusions about the holistic dynamics in the rabies–fox system (Wissel, 1992a; Mollison and Levin, 1995).

However, currently the most relevant problem concerning the vaccination of foxes requires both an assessment of management measures at the local scale and an assessment of the success of the whole control program at regional or even continental scales. There is an ongoing debate in Central Europe over whether the vaccination of foxes can be reduced or stopped in areas where rabies seems to have been eradicated (Stöhr and Meslin, 1996; Selhorst and Schlüter, 1997). The risk of an undetected endemic of rabies in these areas as well as suitable measures against the resurgence of rabies by dispersing individual foxes can only be evaluated by models that are both detailed enough to consider rare infection events and coarse enough to model large areas.

In the following we present a scaling-down strategy of spatially explicit modelling that consists of a sequence of models of different spatial and temporal resolution. Starting with the most basic features of a regional model, we successively integrate only the information that will be relevant to the next finer scale.

2. Biological background

The understanding of the scaling-down process described in this paper requires information on the biological facts associated with the red fox rabies system. The following sections provide this information and subsequently cover three elementary parts of the ecological system studied: the foxes; the disease and control measurement.

2.1. Red foxes

Red foxes (*Vulpes vulpes*) exhibit a distinct annual cycle: mating, reproduction, breeding, dispersal, resettlement, mating,… (Toma and Andral, 1977). During *mating* (December–February) male red foxes show high mobility and are involved in an increased number of encounters with conspecifics. In addition, receptive females attract males by barking and scenting. Two months later (February–April), the females give *birth* to on average five young cubs with up to 14 cubs per litter (Lloyd et al., 1976; Ansorge, 1990) which *grow up* by August. In autumn (September–October), the young foxes leave their birth family and *disperse*. Field data show that the distribution of effective dispersal distances varies for different habitat structures and population densities (Englund, 1980; Storm and Montgomery, 1975; Goretzki et al., 1997). However, there are common features: the dispersing fox follows its initial direction with sparse deviation (Storm and Montgomery, 1975). An empty habitat is preferred for settling (Storm and Montgomery, 1975), but high density does not cause significantly longer disper-

sal (Allen and Sargeant, 1993). After settling, the young fox occupies a home range and explores the mating resources. The next fox-year begins.

The home range represents the spatial context of fox activity and varies by individual, by season, by habitat quality and by local population density factors. Therefore wide ranges of home range size are reported, ranging from 0.5 to 9.6 sq. km (Scott, 1943; Trewhella et al., 1988; Meia and Weber, 1995). However, for foxes of small family groups the respective home ranges overlap widely (Storm and Montgomery, 1975; White et al., 1996). Therefore contacts between members of the social group occur much more frequently than between members of different groups (White et al., 1995) because the home ranges of the latter only have common boundary strips. Consequently, we subdivide the fox population by the social behavior of foxes (i.e. the frequency of contacts) and refer to the space potentially occupied by social groups as the smallest spatial unit of our models instead of referring to home ranges or territories (Thulke et al., 1997a). In the next section the social contact behavior within these groups is linked to the dynamics of the disease by the concept of an 'infection community' (IFC).

2.2. Rabies

Rabies is a virus infection of the central nervous system (Charlton, 1988) which in Europe mainly occurs in the red fox (Aubert, 1992; Schlüter et al., 1997). The virus is transmitted by injurious encounters of infected animals (Wandeler, 1980). The newly infected fox undergoes a variable incubation period with a mean duration of 21 days (Toma and Andral, 1977). It is significant that an incubating fox neither infects other animals nor can be proved to be infected. After the incubation period, the fox becomes infectious. This clinical phase ends after 7–10 days with the inevitable death of the individual (Sikes, 1970). During the last 4 days, 50% of all rabid foxes express the well-known 'frenzied' behavior causing unfounded attacks even on humans (Bacon, 1985).

Due to the frequent contacts between individuals within a social group (Sargeant, 1972; White et al., 1995), a diseased group-member eventually infects all other foxes of the group and consequently after a certain period of time the whole group dies from rabies. We define any fox group that will be completely affected by rabies after one disease cycle if any of its members becomes infected as an IFC. We see a chain of stages ('susceptible—infected—infectious—dead') which completes a cycle of local rabies dynamics if the space where the extinguished IFC has lived becomes re-colonized. In addition, a disease-related subdivision of the rabies–fox system in space and over time is evolved with the concept of IFCs, which emerged as a suitable model resolution (Thulke et al., 1997a).

The cyclic dynamics of a rabies epidemic is also documented on larger scales. The current rabies epidemic in Europe originated in the eastern part and has spread throughout the continent over the last 50 years. The large-scale spreading pattern of the disease exhibits typical advancing waves (Sayers et al., 1977, Fig. 1; or see also Anderson et al., 1981, Fig. 1) which summarize separated foci of the disease on a finer scale that propagate independently (Sayers et al., 1977; Hengeveld, 1989). In the one-dimensional, the temporal description of the wavy spread of rabies the cyclic patterns of the epidemic (Curk, 1991) emerge from various regional case statistics (Sayers et al., 1977; Steck and Wandeler, 1980). Case reports demonstrate a spreading velocity of between 30 and 80 km per year (Ball, 1985) and 3–5 years between two consecutive waves of the epidemic (Jackson, 1982; Macdonald and Voigt, 1985).

The second characteristic of a rabies epidemic repeatedly seen in local case statistics is a periodic but spatially homogeneous increase in infection rates. This is caused by the more frequent contacts between foxes from different family groups during mating and dispersal. Time-series of rabies cases show the typical bimodal annual pattern with peaks indicating the periods of rut and dispersion (Toma and Andral, 1977; Curk, 1991).

2.3. Control measurements

The main goal of control measurements is the reduction of the effective number of susceptible

foxes either by killing or by immunizing during the critical annual phases with an increased infection rate. Vaccination has proven to be helpful but the strategies preferred differ from country to country (Stöhr and Meslin, 1996). In Germany, up to 25 vaccine-filled baits are distributed per square kilometer twice a year from aircraft in regions of rabies occurrence. Despite this enormous effort, not all foxes are immunized; the regular sampling (surveillance) accompanying the vaccination program has found about 70% of the fox population to be immune (Schlüter and Müller, 1995). As is known from theoretical models (Berger, 1976; Anderson et al., 1981; Harris and Smith, 1990; Schenzle, 1995) as well as from certain vaccination campaigns (MacInnes, 1987; Wandeler et al., 1988; Masson et al., 1996; Meslin, 1997), this rate of immunization is close to the threshold beyond which rabies can with high probability be eradicated. However, stopping regular vaccination would bear the risk of a new rabies outbreak due to the high uncertainty of detecting the last few rabid foxes (Bacon, 1981). Therefore a model is needed that serves to estimate the risk of the endemic persistence of rabies and the risk of a new outbreak after the end of a vaccination campaign, i.e. that takes into account local and regional processes of disease transmission and yet is also simple enough to be fully analyzed on a regional scale.

3. Modelling strategy

We start with a model designed to reproduce the regional wave-like pattern of the spread of rabies. It is one-dimensional and discrete and needs only three simple difference equations with in total three parameters that describe the local infection cycle and the spread of the disease. In a step-by-step approach, we then successively integrate new processes on finer spatial and temporal scales until we arrive at the temporal and, even more importantly, the spatial resolutions required to investigate the effects of management interventions with a model. Each single step towards finer scales relaxes some limitations of the previous description on a coarser scale and facilitates the critical evaluation of the impact and the relative importance of each modification. Therefore, one central element of our modeling strategy is a scaling-down from coarser to finer scales, as opposed to the scaling-up approach usually applied with grid-based simulation models which start on the local scale and then analyze the regional or global patterns emerging from the interaction of many local units (Green, 1989; Czárán and Bartha, 1992; Wissel, 1992b; Jeltsch and Wissel, 1994; Ratz, 1995; Jeltsch et al., 1996, 1997b). The second essential feature of our scaling-down strategy is that each interim model is tested against the large-scale pattern of rabies spread (cf. Fig. 1). This 'pattern-oriented' strategy (Grimm et al., 1996b) and the stepwise increase of model complexity make it easier to decide which factors have to be integrated into the model and which details are negligible.

3.1. Basic model

The basic model is discrete in time and space. One time step corresponds to 1 year. The local dynamics of the disease within an IFC consists of only three distinct states, namely 'S' (all foxes are susceptible), 'I' (at least one fox is infected, i.e. an undistinguished part of the IFC is infectious) and 'E' (the IFC is extinct, i.e. the foxes have died from rabies).

The chronological point of reference within a year is the start of the young foxes' migration period. Because of the enormous reproductive success of foxes and the high mobility of cubs we assume that in any empty spatial unit (state E = 'empty'), a new social group is deterministically established (state S = 'susceptible') in the following time step. A susceptible IFC remains susceptible if no infection occurs; otherwise the new state is either I (infected) after the next time step or it is E (extinct). The decision between these two possibilities depends on the point in time of infection. If infection occurs shortly after the chronological point of reference (i.e. the dispersal), the probability μ is high that next autumn the social group will already be extinct (state E). If the infection occurs some time after the point of reference (i.e. due to foxes from neighboring

IFCs), the state of the IFC next autumn is more likely to be *I*. In the latter case the new state of the cell in the time step after next is assumed to be *E* (extinct) due to the close contact within the social group.

To make the model spatial, a sufficiently large area is subdivided by imaginary strips arranged perpendicular to the direction of the spread of rabies. The horizontal width of the strips (i.e. the number of covered IFCs) is selected as being larger than one IFC but smaller than the typical wave-length—say ten IFCs. With this approach, we can use the percentage of IFCs in a certain stage (*S*, *I*, or *E*) as state variables in the model. $I(x, t)$ for example is the percentage of infected IFCs within a strip at location x at time t. The basic model is now:

$$I(x, t) = P_{\mathrm{SI,\,basic}}\,(x, t-1)S(x, t-1)\,(1-\mu) \quad (1)$$

$$E(x, t) = I(x, t-1) + P_{\mathrm{SI,\,basic}}\,(x, t-1)S(x, t-1)\mu \quad (2)$$

$$S(x, t) = E(x, t-1) + (1 - P_{\mathrm{SI,\,basic}}\,(x, t-1))S(x, t-1) \quad (3)$$

with

$$I(x, t) + E(x, t) + S(x, t) = 1 \quad (4)$$

The function $P_{\mathrm{SI,\,basic}}\,(x, t)$ describes the probability that an IFC in the strip at location x is infected in the time step t. This infection probability depends on the number of infected IFCs in the strip itself and in the neighboring strips. It can be quantified as follows (for a detailed derivation of Eq. (5), see Jeltsch et al., 1992):

$$P_{\mathrm{SI,\,basic}}\,(x, t) = 1 - \exp(-Z(x, t)) \quad (5)$$

with

$$Z(x, t) = a \cdot I(x, t) + a \cdot \Sigma_k\,[\exp(-bk^2)\,(I(x+k, t) + I(x-k, t))]$$

The function $Z(x, t)$ totals the influences of the infectious IFCs (state *I*) in neighboring strips depending on the distance k. The decrease in the influence with the increasing distance k of the neighboring strips is described by a simplified normal distribution $a \exp(-bk^2)$. Parameter a is a measure of the infection potential and b determines the decrease in the transmission probability with distance.

The model equations describing the state transitions do nothing more than simple bookkeeping. For example, in Eq. (2) the percentage $E(x, t)$ of empty IFCs in strip x at time t is determined by the percentage of infected IFCs, $I(x, t-1)$, in the last year and the percentage of susceptible IFCs, $S(x, t-1)$ that have been infected in the last year (infection probability $P_{\mathrm{SI,\,basic}}\,(x, t-1)$) and are already extinct in the actual year (probability μ).

The numerical iteration of these equations reveals the typical, wave-like distribution pattern of the disease for a wide range of variations of the parameters a, b and μ (Fig. 2a). The spread of rabies occurs via a shift in this pattern over the course of time (travelling wave). Comparison of the characteristics of the spreading pattern (e.g. spreading velocity, distance of successive peaks) with field measurements (Jackson, 1982; Sayers et al., 1985; Toma and Andral, 1977) reveals a fairly good correspondence on a regional scale. Thus, as anticipated, the simple mechanism of coupled local cycles of colonization, infection and extinction is sufficient to explain the regional pattern qualitatively and, to a fairly good degree, quantitatively.

However, the parameters a and b describing the spread of the disease are highly aggregated parameters. Consequently, it is not clear how the parameters of the basic model are actually related to the various infection mechanisms, operating on different spatial and temporal scales (infection by neighborhood contacts throughout the year, infections caused by itinerant or dispersing foxes in the mating or dispersal period and by foxes in the clinical phase of the disease). The basic model only gives an initial idea of the general probability distribution of infection ($P_{\mathrm{SI,\,basic}}\,(x, t)$) summarizing the various infection mechanisms. Since this general distribution $P_{\mathrm{SI,\,basic}}\,(x, t)$ leads to a regional pattern of rabies that corresponds well with known field data it can be used as a criterion for the quality of the modelling of the single processes on a finer scale. In these modified models, the

joined infection probabilities ought to correspond with the probability distribution used in the basic model.

3.2. Cellular automaton (or grid-based) approaches

3.2.1. Cellular automaton 1 (CA1)

First, we refine the model in a spatially explicit manner. To this end, the modeled area is subdivided into an imaginary grid of cells with each cell representing an IFC. In combination with the 'state and transition' description of the local dynamics as described in the basic model, this results in a typical cellular automaton approach (Wolfram, 1986; Ermentrout and Edelstein-Keshet, 1993; Jeltsch and Wissel, 1994). Each grid cell which is in the state S can be infected with a certain probability by any of the infected IFCs in the neighborhood (up to a maximum distance). The decrease in the infection probability $P_{\mathrm{SI,\,CA1}}(x, t)$ with increasing distance between the IFCs is again described by a normal distribution. However, in this spatially explicit version it is no longer necessary to consider aggregated strips—the spatial pattern can be simulated explicitly (Fig. 3a). The one-dimensional projection of this pattern closely matches the pattern produced by the basic model (Fig. 2a and b). Although, the spatial pattern of the CA1-model displays consecutive waves (Fig. 3a) both the stereotypic regular shape of the wave fronts and the absence of local foci are dissatisfactory when compared even to the large-scale pattern of 'natural' rabies (Fig. 1).

In this version the model still implicitly assumes an unlimited infection and colonization potential, i.e. the model does not account for the limited number of young foxes that leave their parental IFC in autumn and search for a new one. In the following modification of the model towards finer scales, we distinguish between the transmission of rabies among foxes of neighboring IFCs throughout the year and the spread of the disease during the dispersal period with subadult foxes moving over large distances. In this way, we relinquish the unrealistic assumption (previously implicit in the model; Table 1) of an unlimited number of potential colonization and infections that can originate from a single IFC.

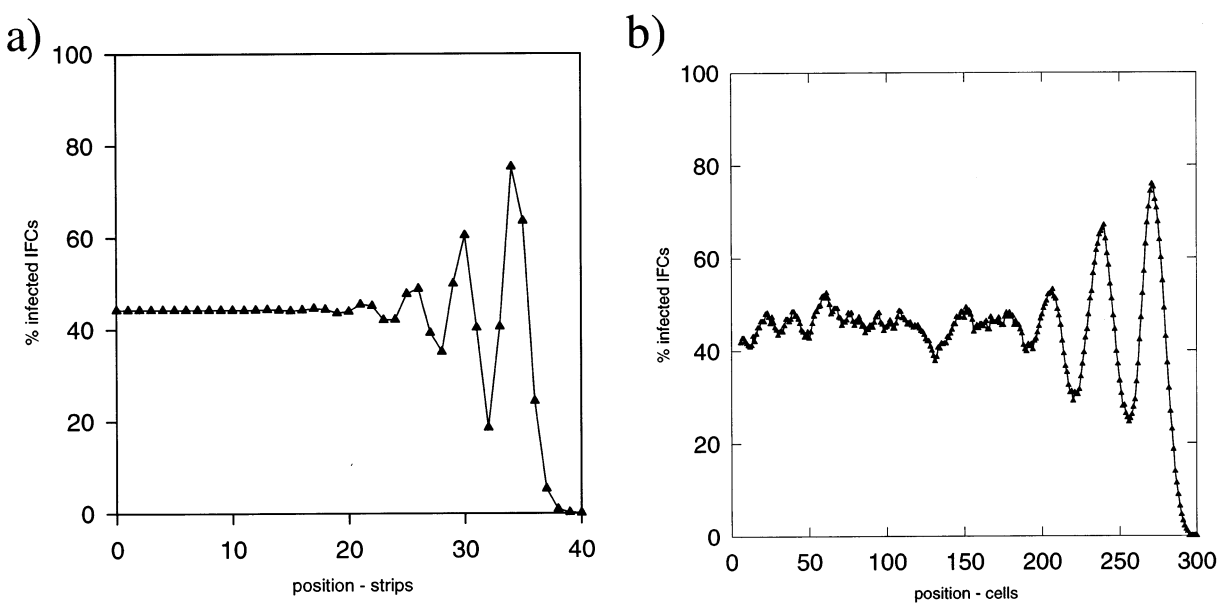

Fig. 2. Oscillating patterns of rabies spread: (a) Basic model ($a = 17$; $b = 0.5$; $\mu = 0.75$); (b) Cellular automata I, time-step 1 year ($a = 0.2$; $b = 0.01$; $\mu = 0.75$).

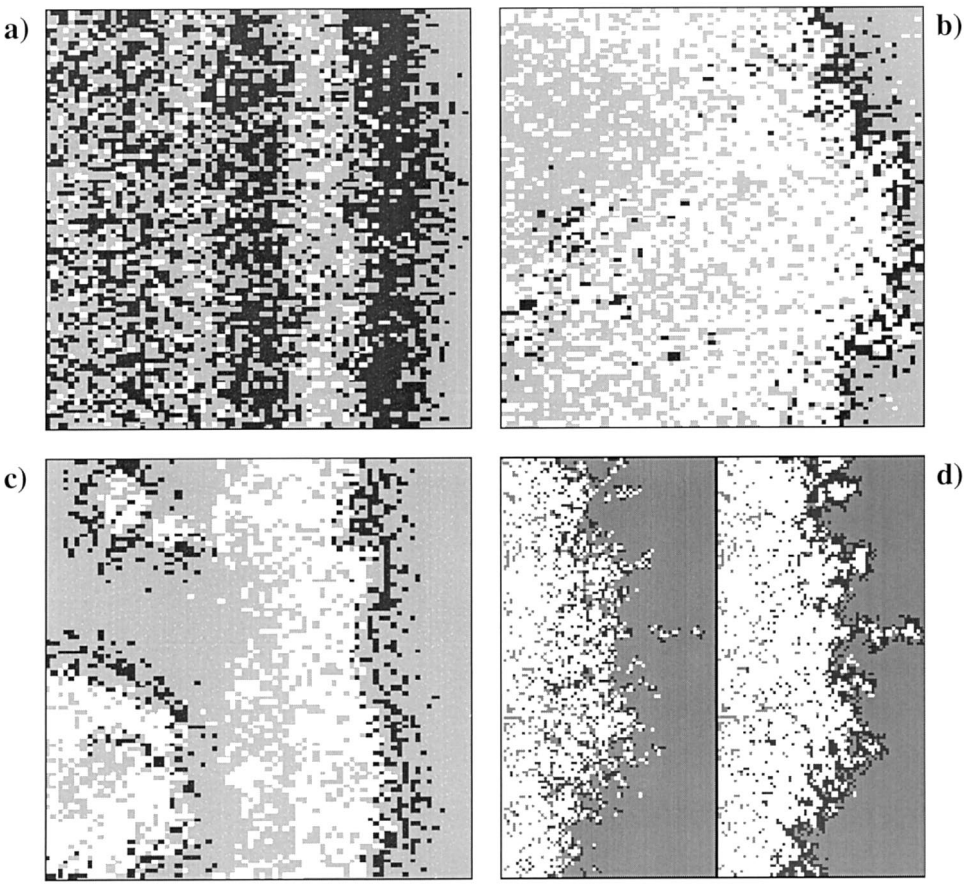

Fig. 3. The spatial epidemic patterns of different model versions (grey, susceptible; black, infected; white, empty): (a) Cellular automata I, time-step 1 year, area 100 × 100 IFCs, ($a = 0.2$; $b = 0.01$; $\mu = 0.75$), stereotype regular pattern; (b) Cellular automata II, time-step 2 months, area 100 × 100 IFCs, ($P_{\mathrm{SIneigh}} = 0.5$; $b_{\mathrm{disp}} = 0.013$), heterogeneous structure of the wave front; (c) Hybrid model, 2 months, 100 × 100, ($P_{\mathrm{SIneigh}} = 0.4$; disp = 4; maxdist = 0.6; $P_{\mathrm{SImig}} = 0.1$), the second wave originates in scattered foci induced by individually dispersing foxes; and (d) Final model, 2 months, 70 × 140, left one time-step before 'mating' and right after 'mating' ($P_{\mathrm{SIneigh}} = 0.4$; disp = 4; maxdist = 0.6; $P_{\mathrm{SImig}} = 0.1$).

3.2.2. Cellular automaton 2 (CA2)

To distinguish between the transmission of rabies during the dispersal period (2 months) and the remainder of the year (10 months), a finer temporal resolution is introduced: a time step represents now 2 months. During each time step, an IFC in state *S* can be infected by any neighboring IFC (Moore neighborhood) in state *I*. This neighborhood infection takes place with a probability depending on the parameter P_{SIneigh} (Table 2). However, during one time step of each year (i.e. in autumn) the large-scale movement of young foxes and thus also of rabies takes place in addition to neighborhood infection: a number *disp* (Table 2) of young foxes leaves each IFC in search of a new homerange. Young foxes that emigrate from IFCs in the state *I* may thus spread rabies over longer distances. The direction of the dispersal of each individual fox is chosen randomly (cf. Storm and Montgomery, 1975) and the distance of each dispersal event is again drawn from a normal distribution. Thus we again only describe the result of the dispersal. The process of dispersal is not yet modeled in detail: migration is neither influenced by the states of IFCs that are passed nor is rabies transmitted during dispersion.

Table 1
Characteristics of and adjustments within the model cascade

	Basic model	CA I	CA II	Hybrid model	Final model	Vaccination
a) Characteristics of the model						
Local dynamics in a IFC	S → S or I or E I → E E → S	S → S or I or E I → E E → E or S	S → S or I I → I or E or S E → E and (E or S)[2] [1] due to vaccination; [2] during dispersal; [3] due to birth			S → S or I and SM[1] I → E E → E and (E or S)[2] SM → SM or IM IM → EM EM → EM and SM[3] and (EM or SM)[2]
Dimensions	one	two				
Spatial resolution	intervals representing 10 IFCs	grid cells representing one IFC				
Time step	1 year		2 months			
Modelling the rabies spread	summarised (normal distributed); automatic re-colonisation	summarised (normal distributed); random re-colonisation			transmission by itinerant adults	
			transmission trough neighborhood infection			
			transmission by dispersing cubs			
			migration summarised (normal distribution)	migration explicit		

Table 1 (*Continued*)
Characteristics of and adjustments within the model cascade

b) Characteristics of modelling the spread					
Number of possible infections induced by one infected IFC within one time-step	unlimited	limited			
		8 neighbouring IFCs			
		4 IFCs related to the number of dispersing cubs per IFC (*disp*)	multiple IFCs per cub during dispersal		
		no additional IFCs during mating		40 additional IFCs during mating	

c) Realism of the models					
Does the model display the regional spatial patterns?	yes, its one dimensional projection, for large parameter subspaces, no spatial structure	yes, for large parameter subspaces, no spatial structure	yes, with spatial fine structure		
			looming local rabies foci	local rabies foci pronounced, later forming the wave front	spatially heterogeneous vaccination success due to the local state of the epidemic)
Does the model display the temporal patterns?	no	yes, long period (3-5 years)	yes, long period (3-5 years) and the dispersal peak	yes, long period (3-5 years) and the bimodal annual cycle	yes, time course and dynamics of cessation in fine adjustment to observed case statistics

Table 1 (*Continued*)
Characteristics of and adjustments within the model cascade

<table>
<tr><td colspan="6">d) Scaling-down the description of the host population</td></tr>
<tr><td rowspan="4">Foxes</td><td rowspan="4">summarised in time and space</td><td colspan="4">explicit in space on the level of IFCs</td></tr>
<tr><td rowspan="3">summarised in time</td><td colspan="3">time-span with long-range transmission (dispersal)
(summarised) | (explicit)</td></tr>
<tr><td rowspan="2">time-span with short-range transmission</td><td colspan="2">time-span with increased mobility (mating)</td></tr>
<tr><td>time-span with standard mobility</td><td>birth
no special event from fox biology</td></tr>
<tr><td colspan="6">Scaling-down the rabies infection cycle (S-I-E)</td></tr>
<tr><td>Smallest unit for rabies cycle</td><td>rabies effects aggregated sub populations (vertical strips)</td><td>rabies effects aggregated local groups (IFCs)</td><td colspan="2">rabies effects aggregated local groups (IFCs) + temporarily individuals</td><td>rabies effects partial local groups + temporarily individuals</td></tr>
</table>

The impact of the dispersing fox, i.e. the infection of a susceptible IFC or the foundation of a new IFC, is restricted to the target area. Table 2 lists the model parameters of the current model version.

The spatial pattern resulting from the second cellular automaton model already looks much more realistic than the stereotypical pattern of the first CA model (Fig. 3a and b). This is mainly due to the increased similarity of the shapes of observed and simulated epizootic waves (Fig. 1a and Fig. 3b). They differ from a simple straight line as the infection potential of the disease is restricted to a local neighborhood resulting in locally different spreading velocity.

Even though the resulting spatial pattern appears more realistic, the second cellular automaton model does not allow the detailed investigation of the impact of short and long-range fox movements on the pattern formation process because of its coarse modelling of the young foxes dispersion. Thus in the next step a submodel for the explicit movement of foxes is included.

Table 2
Parameters with meanings for the successive models

Model	Parameters	Meaning	Investigated range of values
Basic and CA1	a	Infection potential of an infected IFC, used in $P_{SI,..}(x, t)$	
	b	Decrease of the transmission probability by distance, determines $P_{SI,..}(x, t)$	wide ranges
	μ	Distinguish whether an infected IFC will remain infected at following point in time of reference	
CA2	$P_{SIneigh}$	Transition probability $S \rightarrow I$ by one adjacent IFC within one time-step	0–1.0
	b_{disp}	Decrease by distance of the probability to continue the dispersal for young foxes in autumn	wide ranges
Hybrid	$P_{SIneigh}$	Transition probability $S \rightarrow I$ by one adjacent IFC within one time-step	0–1.0
	disp	Number of dispersing cubs leaving the birth IFC in autumn	0–6
	maxdist	Maximal dispersal distance for young foxes in IFCs	0–120
	P_{SImig}	Probability $S \rightarrow I$ by foxes crossing an IFCs in state S	0–1.0
Final	$P_{SIneigh}$	Transition probability $S \rightarrow I$ by one adjacent IFC within one time-step	0–1.0
	disp	Number of dispersing cubs leaving the birth IFC in autumn	0–6
	maxdist	Maximal dispersal distance for young foxes	0–120
	P_{SImig}	Probability $S \rightarrow I$ by foxes crossing an IFCs in state S	0–1.0
Vaccination	$P_{SIneigh}$	Transition probability $S \rightarrow I$ by one adjacent IFC within one time-step	0–1.0
	IR	Immunisation rate in percent	50–90

3.3. Hybrid model: cellular automaton and individual-based model

The hybrid model consists of two components: the local infection dynamics between the IFCs that corresponds to the previous version and an individual-based module which explicitly describes the dispersal of the young foxes through the landscape of IFCs in different states.

In the migration module we model the single sub-adult fox to move explicitly from IFC to IFC, reacting to the state of the grid cell they encounter according to a set of rules. The whole dispersion process takes place within one model time step of 2 months. As in the previous version we assume that each dispersing fox (males and females are not distinguished for reasons of simplicity) has a preference of direction which is randomly chosen. In each move (i.e. step to the next IFC) the probability of entering a neighboring IFC in the preferred direction is 0.5. The probability of entering one of the two adjacent IFCs is 0.25. In each IFC entered by the fox there is a certain settling probability P_{end}. Consequently, if the IFC is in state E and the invading fox is susceptible, colonization takes place (i.e. the state E changes to S) with the probability P_{end}. Since dispersing foxes often tend to follow a 'dispersion instinct' that prevents them from settling in the nearest suitable empty home range (Storm and Montgomery, 1975), we assume the settling probability P_{end} to increase with the distance the dispersing fox has covered:

$$P_{end} = 0.2 + 0.8 \cdot step/maxdist, \text{ if the IFC is empty,}$$

$$P_{end} = 0.05 + 0.35 \cdot step/maxdist, \text{ else.} \quad (6)$$

step is the number of IFCs the fox has already passed; *maxdist* is the maximal dispersal distance (for parameters and their values see Table 2). The probability of settling in a non-empty IFC (i.e. state S or I) is modeled as being lower but also increases linearly with increasing distance (Eq. (6)). If the IFC from which a dispersing fox originates is 'infected', we assume it to be a potential carrier of rabies. This is modeled by a constant infection probability P_{SImig} for IFCs in the state S that are crossed by the disperser (Table

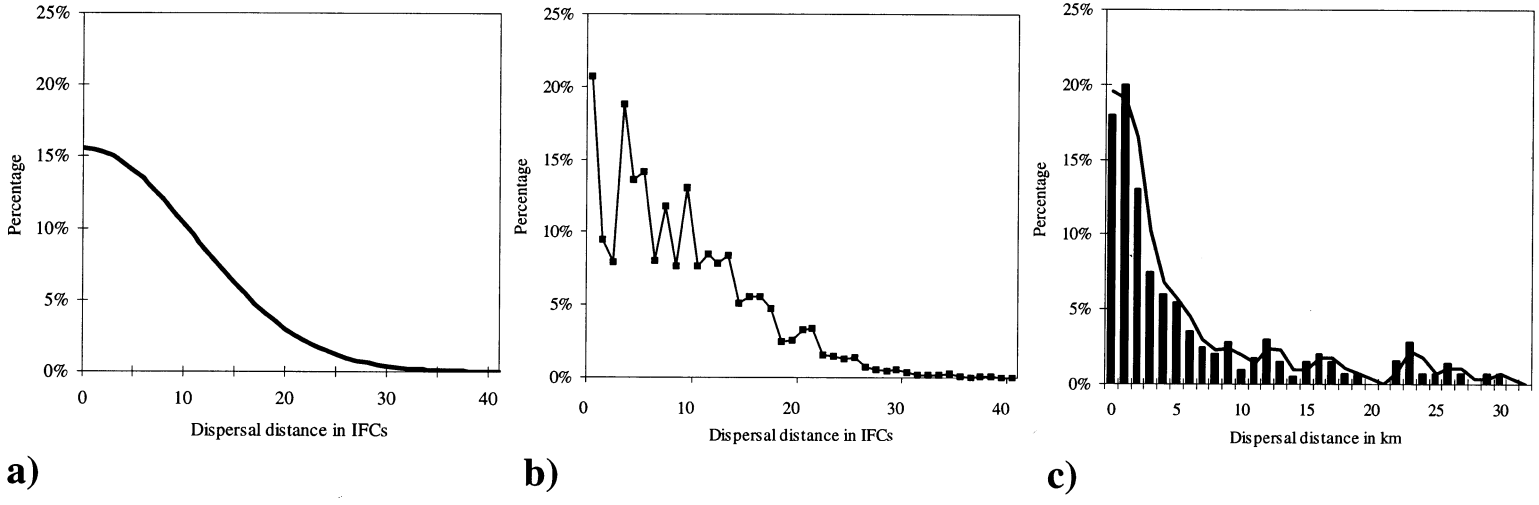

Fig. 4. The distribution of dispersal distances in the respective model: (a) Cellular automata II, summarized dispersal, distances are normally distributed with variance $b_{\text{disp}} = 13$; (b) Hybrid model, individual-based dispersal, distances are governed by probabilistic rules which use the distance already moved and the assumed maximum for dispersing foxes (see Eq. (6)), i.e. maxdist = 60; and (c) Final model in application context, individual-based dispersal, distances are distributed in accordance with field data from capture-recapture studies, i.e. Goretzki et al. (1997).

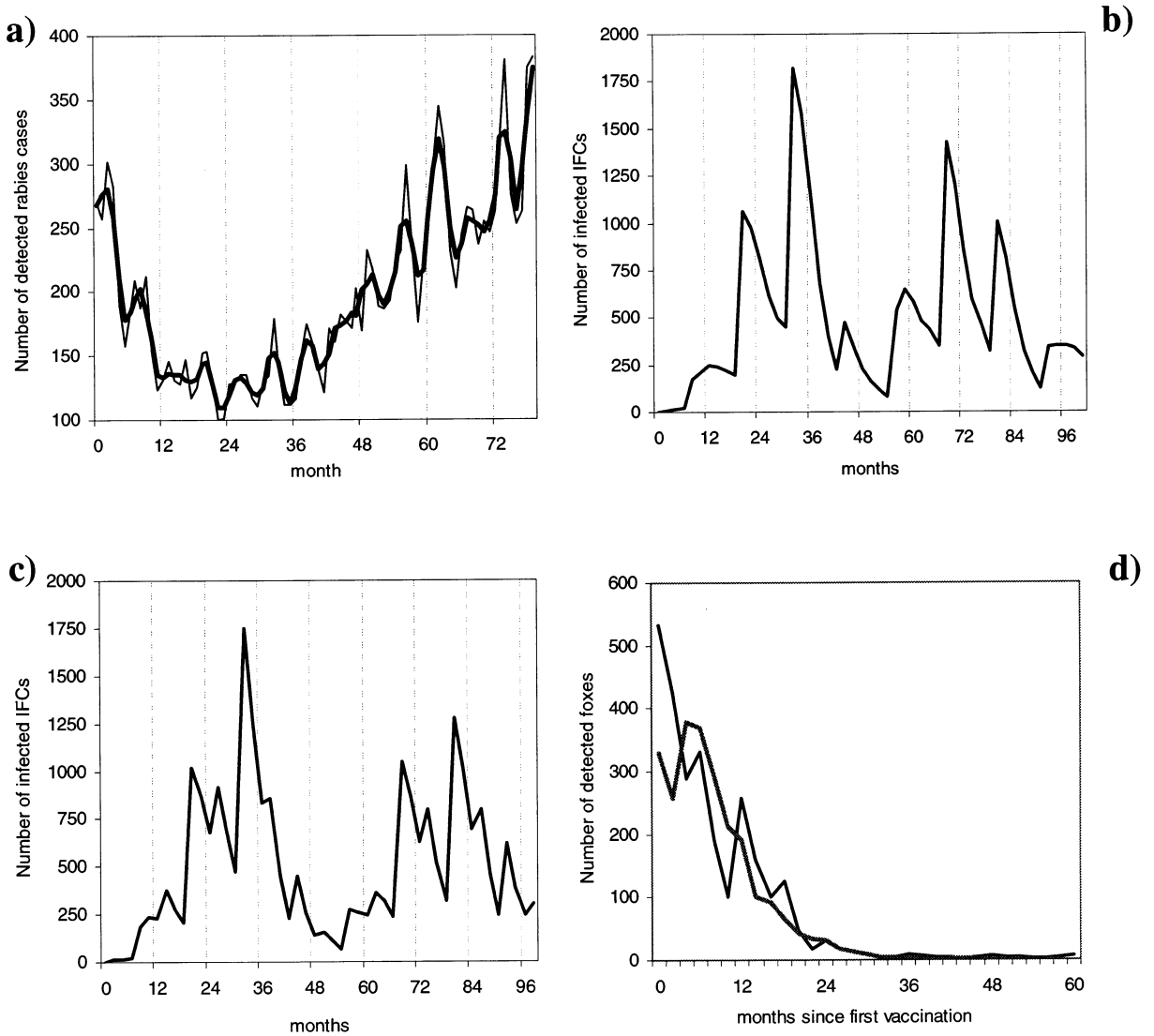

Fig. 5. The temporal epidemic patterns of rabies: (a) Field data, detected rabies cases from eastern Germany 1984–1990 (normal line monthly statistics from ICP-database WHO Collaborating Center Wusterhausen, bold line moving average with three periods). The graph depicts the characteristic bimodal annual cycle of rabies with mating and dispersal peak (cf. Toma and Andral, 1977); (b) Hybrid model, ($P_{\mathrm{SIneigh}} = 0.4$; disp = 4; maxdist = 60; $P_{\mathrm{SImig}} = 0.1$; 100×100). The model reproduces the long-term cyclic (3 years) from rabies dynamics as well as the annual dispersal peak (months 22, 34, 46 cont.), but no peak due to the rut is displayed; (c) Final model, ($P_{\mathrm{SIneigh}} = 0.4$; disp = 4; maxdist = 60; $P_{\mathrm{SImig}} = 0.1$, 100×100). The locally increased infection probability during the time-step of mating adds the second peak to the time-series of infected IFCs in the final model version; and (d) Field data (from ICP-database; gray line) + final model with vaccination ($P_{\mathrm{SIneigh}} = 0.4$; IR = 70, 140×140; black line). To validate the simulated effect of vaccination in the applied model by field data, the simulated time-series of infected IFCs are converted in accordance with the estimated lack of detection (90%, Schlüter and Müller, 1995; see text) and the estimated population density in the field study area (1.4 per sq. km without cubs, Goretzki et al., 1997; Stiebling, 1998, personal communication).

2). The movement itself is modeled for the infected fox in the same way as for susceptible foxes. The description of the dispersal movement contains the mortality of the disperser.

Introducing in the hybrid model the interaction of rather simple processes on local and regional scales in a homogenous space, the resulting spatial pattern (Fig. 3c) is further adjusted to the spatial rabies pattern (Fig. 1). The pattern of the simulated epidemic is marked by distinct local foci. These 'hot spots' are the centers of new micro-epidemics that in the course of time combine to form a new wave. However, the increased likeliness of the simulated spatial pattern is based on changed dispersal rules. But since the dispersal distribution resulting from this dispersion module closely matches the previously used normal distribution (cf. Fig. 4a and b), the consistent 'scaling-down' of the previous models is guaranteed.

The hybrid model combines the potential of the cellular automaton model to explore complex spatial patterns with the flexibility of the dispersion module in order to consider the consequences of individual behavior. The resulting components are marked in Table 1 and a typical spatial pattern as produced by the model is plotted in Fig. 3c. The detailed sensitivity analysis of the hybrid model and an analysis of the process of pattern formation by dispersing and frenzied foxes is given elsewhere (Jeltsch et al., 1997a). Here we focus on the transition towards the next modelling step.

3.4. Final model: including temporal patterns at the local scale

With the next step we overcome the restriction towards spatial patterns being the only criterion to assess the model quality. Now the regional temporal pattern of seasonal variation in the disease is taken into account. Comparing the simulated total number of infected IFCs over time as produced by the hybrid model with field data (Fig. 5a,b), both time series display the 'dispersal peak' (Toma and Andral, 1977). Admittedly, the second peak in the empirical time series related to the increased mobility during the rut (Storm and Montgomery, 1975) is not reproduced by the hybrid model. Therefore the description of the annual cycle of the fox population is refined (Table 1d), i.e. the feature 'mating' is now added to the hybrid model. During the time span of mating, an IFC in state S can be infected by any IFC in state I situated within a neighborhood of three consecutive rings of IFCs around the IFC in question. Consequently, the probability of infection increases for the susceptible IFCs. The probability that state S will change into state I during mating is calculated from the numbers k, m and n of infected IFCs (state I) within the first, second and third ring of the neighborhood:

$$P_{\mathrm{SImat,\,final}} = 1 - [(1 - P_{\mathrm{SIneigh}})^k * (1 - P^2_{\mathrm{SIneigh}})^m * (1 - P^3_{\mathrm{SIneigh}})^n] \quad (7)$$

With this modification, the model (Fig. 5c) reproduces both observed peaks within the time series of rabies cases (Toma and Andral, 1977; Curk, 1991). Once again, the advantage of the scaling-down approach is demonstrated: To model the regional wave-pattern of the disease, it is sufficient to distinguish between the phase of high-velocity spreading (i.e. long-distance transmission during dispersal) and the phase of low-velocity spreading (i.e. short-distance transmission by neighboring IFCs). If focus is on the regional temporal pattern, we have to refine the description of the low-velocity phase with respect to the frequency of transmission events. Turn-around in time mating and dispersal are evenly related to increased frequency of transmission (cf. the bimodal time-series). However, combining the temporal point of view with the spatial aspect allows the impact of rut and dispersal to be distinguished: during dispersal the increased number of infections is correlated to a propagation of the disease on a global scale, but during the mating period forced transmission results in a spatially restricted local accumulation of infections within the vicinity of infected IFCs, i.e. the qualitative properties of the large-scale spatial rabies pattern are not affected by the local consequences of the mating period. This finding illustrates why the biologically important details of mating could be omitted as long as the posed question is not tangent to smaller temporal scales, i.e. up to the hybrid model. However, rabies management potentially acts on short-

term scales and thus mating has to be included when preparing the final model to the application as management tool.

3.5. Management tool: vaccination

The model is adjusted to both the spatial and temporal patterns of the dynamics of the disease. Consequently, the model resolution in space and time or the resolution under which local processes and dispersal behavior are taken into account, are now appropriate to use the model in the exploration of applied questions. Particularly, combining vaccination and culling in spatially different patterns, we investigated the impact of such control strategies on the spread of the epidemic (for the details how to include culling into the model see Brandl et al., 1994). This investigation restricts to the combination of both control measures because pure culling was suggested as insufficient for rabies eradication (Bacon, 1985) especially in regions like Europe that have high population density and good habitat conditions for the foxes (Goretzki, 1995). Currently, however, temporally different vaccination regimes are being investigated to find the most efficient eradication strategy. Management implications for the continuation of the vaccination program in Germany were derived from these studies. As another example we address the question of lasting sporadic rabies cases occurring after several years of repeated vaccination campaigns common in some parts of Germany. The remaining cases call into question the huge effort for disease eradication because no final proof of eradication seems possible. Consequently, insights into possible reasons for disease persistence found with the model assisted decision making in rabies control policy (Tischendorf et al., 1998).

Vaccination, i.e. the distribution of baits twice a year, can easily be incorporated into the model. A new parameter, IR (Table 2) is introduced to describe the proportion of the fox population in the modelled area immune to the disease. Modelling the vaccination procedure is then equal to a further refinement of state variables I, S and E which describe the state of an IFC. Every IFC in state S is now distinguished between the susceptible and the immune foxes. The new state variable is SM (Susceptible + iMmune). If we assume a homogeneous immunization rate in the entire area, the ratio of immune to susceptible foxes within each IFC is equal to IR. Within the following time step an infection may occur and would result in a second and third mixed cell state, IM and EM, respectively. In other words, introducing vaccination into the model means simply restricting the disease-related transitions (S–I–E) in the former models to the non-immunized proportion of the respective IFCs (Table 1).

It is important to note that the simulation of vaccination causes a further scaling-down in time (Table 1). Two events must be scheduled in addition: firstly, the transition S–SM caused by the vaccination event and secondly, the possible transition EM–SM induced by the birth of cubs in IFCs which no longer die out completely after an infection because of partial immunity. However, it is not necessary to model adult mortality in detail at the actual scale of resolution because along with the repeated vaccination campaigns immunized and non-immunized foxes die in rather equal proportions. Consequently, the immunity level changes simply due to the event birth restricted to one time-step.

If the infection probabilities used for the different spreading mechanisms represent aggregated frequencies of infectious contacts between animals, 'vaccination' demands a cogent modification of these probabilities because only $(100 - IR)\%$ of all transmission events are still infectious. Consequently, if the involved IFCs contain immune animals, the actual infection probabilities are reduced according to the parameter IR:

$$P_{\mathrm{SI}<\mathrm{trans}>;\mathrm{Vacc}} = (100\% - \mathrm{IR}) * P_{\mathrm{SI}<\mathrm{trans}>;\mathrm{Final}}, \quad \mathrm{trans} \in \{\mathrm{neigh}, \mathrm{mat}, \mathrm{disp}\}. \tag{8}$$

The rules described for vaccination may seem coarse but are nevertheless sufficient in view of the available resolution in field data. The behavior of the immunized fox–rabies system simulated by the model coincides with general empirical knowledge from field data (Stöhr and Meslin, 1996;

Masson et al., 1996) and from theoretical considerations (Anderson et al., 1981; Murray and Seward, 1992; Schenzle, 1995), i.e. the onset of vaccination results in a drastic decrease of disease prevalence within 2 years (cf. Fig. 5d for observed and simulated data). Furthermore, the 'threshold value' for the necessary effectiveness of the vaccination in order to eradicate a disease is predicted by various models (Anderson et al., 1981; Murray and Seward, 1992; Rhodes and Anderson, 1997). Indeed, our model correspondingly produces thresholds for the parameter *IR* above 70% (Tischendorf et al., 1998). The thresholds define a range of *IR* beyond which the simulated epidemic either recovers or breaks down. However, within this range it is not possible to decide a priori how the epidemic will behave in the long run. These results and the systematic exploration of the parameter space with the relevant conclusions are presented in detail elsewhere (Tischendorf et al., 1998). Here we apply the management tool in a more particular fashion and provide an explanation for the lasting rabies cases observed despite vaccination in Germany.

4. Application

4.1. Adapting to a particular ecological set-up

So far the models aim at being general (Durrett, 1995) instead of describing particular situations. This generality enables a demonstration of the 'realism' (Barlow, 1995) of our model by comparison with existing knowledge about fox rabies epidemics. Addressing the particular question of lasting rabies cases following a long-term vaccination program in eastern Germany, more subtle modifications are necessary to adapt our model to the respective ecological set-up.

Consequently, further adjustments were made in accordance with field data concerning red foxes documented in eastern Germany (Goretzki and Paustian, 1982; Goretzki et al., 1997). An empirically obtained distribution of dispersal distances (Fig. 4c; Goretzki et al., 1997) now governs the migration module. It is interesting that the previous assumption on increasing settling probability by distance agrees with the field data. In addition, the number of dispersing foxes per IFC is no longer a constant, i.e. parameter *disp*. Instead, for each IFC *disp* is randomly chosen from an empirical distribution of litter sizes (Ansorge, 1990; Goretzki et al., 1997). Taking observed postnatal mortality into account, on average 3–4 cubs leave every IFC (Lloyd et al., 1976; Allen, 1983; Ansorge, 1990), which corresponds with the assumed range of *disp* used in the former model versions.

4.2. Low-level persistence of rabies in immunized fox populations

With the model adjusted to the particular region, we simulate 10 years of biannual area-wide vaccination as was applied in the study area. The aim of the simulation study is to decide whether the lasting rabies cases as observed in the field even after 6 years of repeated vaccination are necessarily due to immigrating infected foxes from Eastern Europe (i.e. non-controlled areas) or if they might reflect an inherent spatio-temporal pattern of the epidemic under control by vaccination. Obviously, while the assumption of immigrating infected individuals favors some form of borderline control (Aubert, 1992; Murray and Seward, 1992; Brandl et al., 1994), the endemic state of the disease in the latter would require quite different measures for further management.

We found low-level persistence of rabies to be a possible inherent disease dynamic within partially immunized populations (Tischendorf et al., 1998). In fact, totally ignoring immigrating infected foxes, simply the controlled fox rabies system itself can develop a spatio-temporal pattern of case-occurrence suitable to maintain the disease for 10 years despite vaccination but only with a few lasting IFCs simultaneously infected (Tischendorf et al., 1998). Fig. 6 illustrates an example of the phenomenon for a parameter configuration that can produce low-level persistence of the disease ($P_{\mathrm{SIneigh}} = 0.34$; $IR = 70$). After the onset of vaccination (Fig. 6a) the disease is suppressed dramatically and the fox population recovers (Fig. 6b). However, small local seeds of infected IFCs are maintained and in turn final global extinction occurs by chance. Moreover the residual foci of

infection move in space over time due to the mechanisms of transmission and the lethality of the disease (Fig. 6c–d). To sum up, low-level persistence of the disease clusters a very few number of cases from an otherwise largely rabies-free area in foci which are small but not spatially fixed over time. With these circumstances in mind we recall the detection rate to be less then 10%, even for rabies epidemics without vaccination (Braunschweig, 1980; Jackson, 1982; Schlüter and Müller, 1995). But if the disease persists at the low level under discussion, the situation becomes worse again, for detecting one rabid fox supposes that the random sampling in the whole area meets at least once a foci of the disease and kills an infected fox there; more over, this fox must be in the clinical phase. Consequently, the low-level persistence of the disease induces a majority of unreported cases and a few sporadic proofs (Thulke et al., 1997b).

5. Discussion

Ecological modelling is a process. Any description of a particular model is thus only a snapshot showing a certain stage in this process. A model which is, for example, presented in a paper is

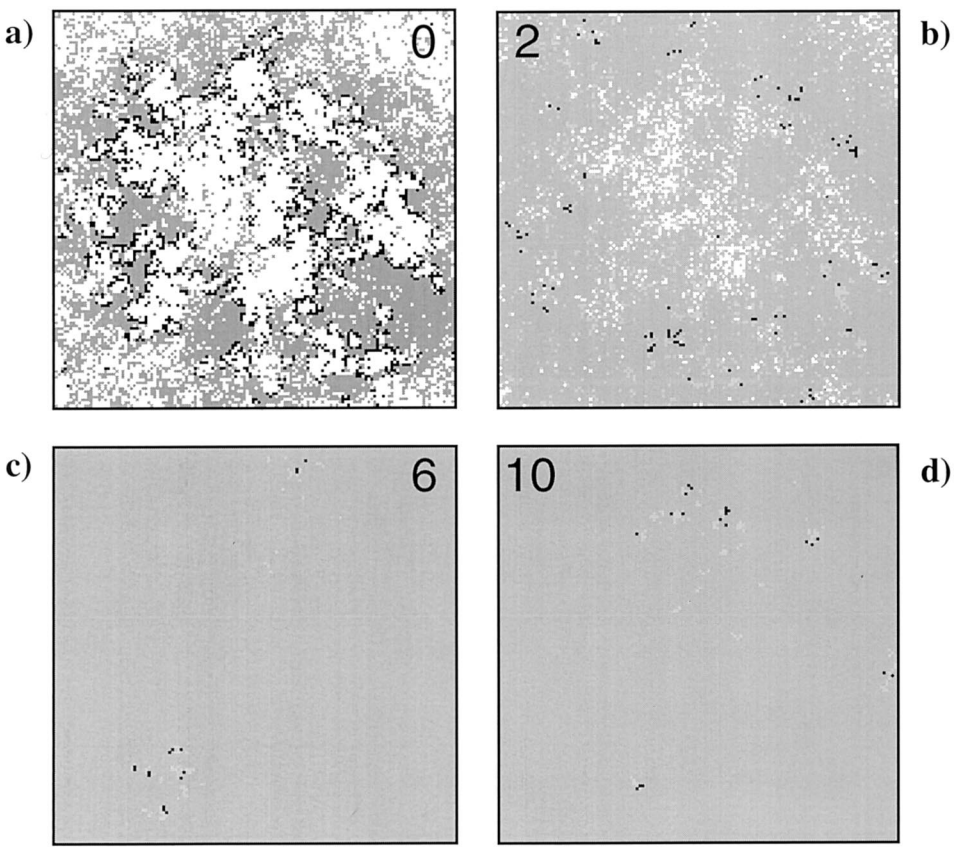

Fig. 6. Spatial snapshot of the simulated low-level persistence of rabies in immunized fox populations ($P_{\mathrm{SIneigh}} = 0.34$; IR = 70; 140×140): The spatial snapshots plot the initial setting (a) and consecutive time-steps 12 (b), 36 (c) and 60 (d) (i.e. years 2, 6 and 10 of repeated biannual vaccination). After the first vaccination at time-step two (spring of year 1), the number of infected IFCs decreases strongly within the following 2 years (compare (a) and (b)). Furthermore, the actual level of prevalence neither recovers nor reaches the zero-level. In time rabies 'persists' for 8 years at a 'low-level' of prevalence (less than 0.2%) and in space low-level persistence of the disease is linked to small spatio-temporal moving infection clusters.

never exactly the same as the first model from which the investigation started, nor is the presented model ever complete such that it cannot be improved. There are, of course, good reasons to present only snapshots. The main reasons are the limited space in the journals and the fact that interim models usually do not do what they are supposed to do and therefore have become modified. But there is a problem if we never publish the process of modelling itself: we do not learn from each other how to use systematic guidelines to modify a model for a certain need. This is a waste of resources: the wheel of ecological modelling is being repeatedly reinvented. Moreover, the question of *how* ecological models for applied problems could be efficiently constructed is not really acknowledged as an important question per se (but see Starfield and Bleloch, 1991).

Classification into 'strategic' and 'tactical' models gives the impression that general, theoretical models give no guidelines of how to design applied models, i.e. how to find the appropriate level of resolution (cf. Grimm, 1998b). In this paper we demonstrate that this is not necessarily the case. We started our investigations with a purely theoretical problem: How can we explain in detail the emergence of the wave-like spread of rabies (Jeltsch et al., 1997a)? But almost at the same time, once we had finished our theoretical studies, we came into contact with people who had literally no interest in explaining any fancy wave pattern, but had the problem of interpreting the sporadic incidence of rabies in areas that have been continuously vaccinated for many years. Could this be an intrinsic property of the vaccinated system, or is the invasion of individual infected foxes the only possible explanation? To tailor our theoretical model, we continued the modelling strategy of scaling-down the model (Brandl et al., 1994; Grimm et al., 1996a). The main stages of this strategy are as follows: (1) Start with a coarse model that aims at producing some overall distinctive pattern of the system; (2) modify the model step by step. This allows each interim model to be linked to all its predecessors and in particular to the pattern at the beginning; and (3) try to link the different models by overlapping modules, i.e. a coarse description of a process or component in a module is replaced in the next version of the model by a more detailed and, if possible, empirically motivated description of the same process. This enables the question of whether the parameters used in the coarse description were correct to be checked (if not, the earlier models have to be scaled accordingly) and it also gives a clear idea of how to check the new submodel: it should, if it is scaled up again to the coarser description, produce at least the same kind of distribution or (for example) whatever. If it does not, both the submodel and the earlier full model have to be reconsidered. The essence of this modelling strategy is that it is 'pattern-oriented' (Grimm et al., 1996b) and that it scales down from coarser to finer scales, whereas cellular automaton (or, more generally, grid-based) models are usually only supposed to support the other approach, from the local to the global scale.

One might argue that the applicability of our modelling strategy is limited because we usually do not have neat patterns such as that in the spread of rabies. This may be true, but it may also be true that when tackling applied problems, modellers do not even consider large-scale patterns at all. What we wanted to show here is that it is always worth thinking in terms of patterns in space, time or any level of organization. Patterns may only exceptionally be as marked as in the case of rabies, but less distinct patterns can be used in the same way. For instance, modellers often use various kinds of patterns (i.e. time-series, frequency distributions, habitat use patterns) in the calibration process and for the validation of their models (Stephan et al., 1998; Wiegand, K. et al., 1998; Wiegand, T. et al., 1998). In addition, a combination of two or more weak patterns might be as useful or even more useful than one single spatial pattern (Staubach et al., 1998). Such a kind of pattern may be used as a 'fingerprint' of the system. In any system and with any question, there must be some kind of pattern, because without a pattern we would not be able to perceive the system or the question. 'Pattern' is anything beyond random variation; without a pattern there would be only noise (Grimm, 1996, 1998a; Jax et al., 1998). The modelling strategy presented

here means gearing ecological modelling and, more generally, ecological research towards the patterns we perceive in nature (Grimm, 1994; Weiner, 1995; Den Boer and Reddingius, 1996; Grimm et al., 1996b).

Acknowledgements

The authors are grateful to two anonymous reviewers for their stimulating comments.

References

Allen, S.H., 1983. Comparison of red fox litter size determined from counts of embryos and placental scars. J. Wildl. Manage. 47, 860–863.

Allen, S.H., Sargeant, A.B., 1993. Dispersal patterns of red foxes relative to population density. J. Wildl. Manage. 57, 526–533.

Anderson, R.M., Jackson, H.C., May, R.M., Smith, A.D.M., 1981. Population dynamics of fox rabies in Europe. Nature 289, 765–770.

Ansorge, H., 1990. Daten zur Fortpflanzungsbiologie und zur Reproduktionsstrategie des Rotfuchses, *Vulpes vulpes*, in der Oberlausitz. Säugetierkd. Inf. 3, 185–199.

Aubert, M.F.A., 1992. Epidemiology of fox rabies. In: Bögel, K., Meslin, F.X., Kaplan, M. (Eds.), Wildlife Rabies Control, Wells Medical, Kent, pp. 9–18.

Bacon, P.J., 1981. The consequences of unreported fox rabies. J. Environ. Manage. 13, 195–200.

Bacon, P.J., 1985. A systems analysis of wildlife rabies epizootics. In: Bacon, P.J. (Ed.), Population Dynamics of Rabies in Wildlife, Academic Press, London, pp. 109–130.

Ball, F.G., 1985. Front-wave velocity and fox habitat heterogeneity. In: Bacon, P.J. (Ed.), Population Dynamics of Rabies in Wildlife, Academic Press, London, pp. 197–222.

Barlow, N.D., 1995. Critical evaluation of wildlife disease models. In: Grenfell, B.T., Dobson, A.P. (Eds.), Ecology of Infectious Diseases in Natural Populations. Cambridge University Press, Cambridge, pp. 230–259.

Berger, J., 1976. Model of rabies control. Lect. Notes Biomath. 11, 74–91.

Brandl, R., Jeltsch, F., Grimm, V., Müller, M.S., Kummer, G., 1994. Modelle zu lokalen und regionalen Aspekten der Tollwutausbreitung. Z. Ökol. Natur. 3, 207–216.

Braunschweig, A.V., 1980. Ein Modell für die Fuchspopulation in der Bundesrepublik Deutschland. In: Zimen, E. (Ed.), Biogeographica Vol. 18—The Red Fox, Dr. W. Junk, The Hague, pp. 97–106.

Cain, M.L., Pacala, S.W., Fortin, M.J., 1995. Neighborhood models of clonal growth in the white clover *Trifolium repens*. Am. Nat. 145, 888–947.

Charlton, K.M., 1988. The pathogenesis of rabies. In: Campball, J.B. (Ed.), Rabies. Cluever, Boston, pp. 101–150.

Curk, A., 1991. Seasonal and cyclic patterns of fox rabies in Slovenia. Vet. Arhiv. 61, 19–24.

Czárán, T., Bartha, S., 1992. Spatiotemporal dynamic models of plant populations and communities. Trends Ecol. Evol. 7, 38–42.

David, J.M., Andral, L., Artois, M., 1982. Computer simulation model of the epi-enzootic disease of vulpine rabies. Ecol. Modell. 15, 107–125.

Den Boer, P.J., Reddingius, J., 1996. Regulation and Stabilization Paradigms in Population Ecology. Chapman and Hall, London.

Durrett, R., 1995. Spatial epidemic models. In: Mollison, D. (Ed.), Epidemic Models—Their Structure and Relation to Data. Cambridge University Press, Cambridge, pp. 187–201.

Englund, J., 1980. Population dynamics of the red fox (Vulpes vulpes L., 1758) in Sweden. In: Zimen, E. (Ed.), Biogeographica Vol. 18–The Red Fox, Dr. W. Junk, The Hague, pp. 107–121.

Ermentrout, G.B., Edelstein-Keshet, L., 1993. Cellular automata approaches to biological modelling. J. Theor. Biol. 160, 97–133.

Glenn, S.M., Collins, S.L., 1993. Experimental analysis of patch dynamics in tall-grass prairie plant communities. J. Veg. Sci. 4, 157–162.

Goretzki, J., 1995. Opportunist und Gewinner. Unsere Jagd 2/95, 8–10.

Goretzki, J., Ahrens, M., Stubbe, C., Tottewitz, F., Sparing, H., Gleich, E., 1997. Zur Ökologie des Rotfuches (*Vulpes vulpes* L., 1758) auf der Insel Rügen: Ergebnisse des Jungfuchsfanges und der Jungfuchsmarkierung. Beitr. Jagd. Wildforsch. 22, 187–199.

Goretzki, J., Paustian, K.-H., 1982. Zur Biologie des Rotfuchses, Vulpes vulpes (L., 1758), in einem intensiv landwirtschaftlich genutzten Gebiet. Beitr. Jagd. Wildforsch. 12, 96–107.

Green, D.G., 1989. Simulated effects of fire, dispersal and spatial pattern on competition within forest mosaics. Vegetatio 82, 139–153.

Grimm, V., 1994. Mathematical models and understanding in ecology. Ecol. Model. 75/76, 641–651.

Grimm, V., 1996. A down-to-earth assessment of stability concepts in ecology: dreams, demands and the real problems. Senkenb Marit. 27, 215–226.

Grimm, V., 1998a. To be or to be essentially the same: the Self-identity of ecological units. TREE 13, 298–299.

Grimm, V., 1998b. Ten years of individual-based modelling in ecology: what have we learned and what could we learn in the future? Ecol. Model. 115, 129–148.

Grimm, V., Brandl, R., Jeltsch, F., Müller, M.S., 1996a. Ein gitterbasiertes Simulationsmodell als Werkzeug zur Untersuchung der Tollwutausbreitung und -bekämpfung. Beitr. Jagd. Wildforsch. 21, 277–285.

Grimm, V., Frank, K., Jeltsch, F., Brandl, R., Uchmánski, J., Wissel, C., 1996b. Pattern-oriented modelling in population ecology. Sci. Total Environ. 183, 151–166.

Harris, S., Smith, G.C., 1990. If rabies comes to Britain. New Sci. 128, 20–21.

Hengeveld, R., 1989. Dynamics of Biological Invasions. Chapman and Hall, London.

Holling, C.S., 1966. The strategy of building models of complex ecological systems. In: Watt, K.E.F. (Ed.), Systems Analysis in Ecology. Academic Press, New York, pp. 195–214.

Jackson, H.C., 1982. Rabies in the Federal Republic of Germany. Comp. Immunol. Infect. Dis. 5, 309–313.

Jax, K., Jones, C.G., Pickett, S.T.A., 1998. The self-identity of ecological units. OIKOS 82, 253–264.

Jeltsch, F., Wissel, C., Eber, S., Brandl, R., 1992. Oscillating dispersal patterns of tephritid fly populations. Ecol. Model. 60, 63–75.

Jeltsch, F., Wissel, C., 1994. Modelling dieback phenomena in natural forests. Ecol. Modell. 75/76, 111–121.

Jeltsch, F., Milton, S.J., Dean, W.R.J., van Rooyen, N., 1996. Tree spacing and coexistence in semiarid savannas. J. Ecol. 84, 583–595.

Jeltsch, F., Müller, M.S., Grimm, V., Wissel, C., Brandl, R., 1997a. Pattern formation triggered by rare events: lessons from the spread of rabies. Proc. R. Soc. London Ser. B 264, 495–503.

Jeltsch, F., Milton, S.J., Dean, W.R.J., van Rooyen, N., 1997b. Simulated pattern formation around artificial waterholes in the semi-arid Kalahari. J. Veg. Sci. 8, 177–188.

Källen, A., Arcuri, P., Murray, J.D., 1985. A simple model for the spatial spread and control of rabies. J. Theor. Biol. 116, 377–393.

Kareiva, P., Wennergren, U., 1995. Connecting landscape patterns to ecosystem and population processes. Nature 373, 299–302.

Levin, S.A., 1992. The problem of pattern and scale in ecology. Ecology 73, 1943–1967.

Levins, R., 1966. The strategy of model building in population biology. Am. Sci. 54, 421–431.

Lima, S.L., Zollner, P.A., 1996. Towards a behavioural ecology of ecological landscapes. TREE 11, 131–135.

Lloyd, M., Jensen, B., Haaften, J.L.v., Niewold, F.J.J., Wandeler, A.I., Bögel, K., Arata, A.A., 1976. The annual turnover of fox populations in Europe. Zentralbl. Veterinärmed., Reihe B 23, 580–589.

Lubina, J.A., Levin, S.A., 1988. The spread of a reinvading species: range expansion in the California sea otter. Am. Nat. 131, 526–543.

Macdonald, D.W., Voigt, D.R., 1985. The biological basis of rabies models. In: Bacon, P.J. (Ed.), Population Dynamics of Rabies in Wildlife. Academic Press, London, pp. 71–108.

MacInnes, C.D., 1987. Rabies. In: Novak, M., Baker, J.A., Obbard, M.E., Malloch, B. (Eds.), Wild Furbearer Management and Conservation in North America. Trappers Association, Ontario, pp. 910–929.

Maron, J.L., Harrison, S., 1997. Spatial pattern formation in an insect host-parasitoid system. Science 278, 1616–1619.

Masson, E., Aubert, M.F.A., Barrat, J., Vuillaume, P., 1996. Comparison of the efficacy of the antirabies vaccines used for foxes in France. Vet. Res. 27, 255–266.

May, R.M., 1973. Stability and Complexity in Model Ecosystems. Princeton University Press, Princeton, NJ.

Meia, J.S., Weber, J.M., 1995. Home ranges and movements of red foxes in central Europe: stability despite environmental changes. Can. J. Zool. 73, 1960–1966.

Meslin, F.M., 1997. Zoonoses in the world—current and future trends. In: Information Circular 42, WHO Mediterranean Zoonoses Control Centre, pp. 2–4.

Mollison, D., 1991. Dependence of epidemic and population velocities on basic parameters. Math. Biosci. 107, 255–287.

Mollison, D., Levin, S.A., 1995. Spatial dynamics of parasitism. In: Grenfell, B.T., Dobson, A.P. (Eds.), Ecology of Infectious Diseases in Natural Populations. Cambridge University Press, Cambridge, pp. 384–398.

Murray, J.D., Stanley, E.A., Brown, D.L., 1986. On the spatial spread of rabies among foxes. Proc. R. Soc. London Ser. B 229, 111–150.

Murray, J.D., Seward, W.L., 1992. On the spatial spread of rabies among foxes with immunity. J. Theor. Biol. 156, 327–348.

Nisbet, R.M., Gurney, W.S.C., 1982. Modelling Fluctuating Populations. Wiley, New York.

Okubo, A., Murray, J.D., Williamson, M., 1989. On the spatial spread of grey squirrels in Britain. Proc. R. Soc. London Ser. B 238, 113–125.

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

Perrins, J., Fitter, A., Williamson, M., 1993. Population biology and rates of invasion of three introduced Impatiens species in the British Isles. J. Biogeogr. 20, 33–44.

Peters, R.H., 1991. A Critique for Ecology. Cambridge University Press, New York.

Ratz, A., 1995. Long-term spatial patterns created by fire: a model oriented towards boreal forests. Int. J. Wildland Fire 5, 25–34.

Reichman, O.J., 1993. Distinct animal-generated edge effects in a tallgrass prairie. Ecology 74, 1281–1284.

Rhodes, C.J., Anderson, R.M., 1997. Epidemic threshold and vaccination in a lattice model of disease spread. Theor. Popul. Biol. 52, 101–118.

Sargeant, A.B., 1972. Red fox spatial characteristics in relation to waterfowl predation. J. Wildl. Manage. 36, 225–236.

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

Sayers, B.M., Mansourian, B.G., Phantan, T., 1977. A pattern analysis study of a wildlife rabies epizootic. Med. Inform. 2, 11–34.

Sayers, B.M., Ross, J.A., Saengcharoenrat, P., Mansourian, B.G., 1985. Pattern analysis of the case occurrences of fox rabies in europe. In: Bacon, P.J. (Ed.), Population Dynamics of Rabies in Wildlife. Academic Press, London, pp. 235–254.

Schenzle, D., 1995. Zur Frage der weiteren Tollwutbekämpfung in Deutschland. Dtsch. Tierärztl. Wochenschr. 102, 419–454.

Schippers, P., Verboom, J., Knaapen, J.P., van Apeldoorn, R.C., 1996. Dispersal and habitat connectivity in complex heterogeneous landscapes: an analysis with GIS-based random walk model. Ecography 19, 97–106.

Schlüter, H., Müller, T., Staubach, C., Fröhlich, A., 1997. Rabies control in Central Europe-results after more than 10 years of oral immunization of foxes. Epidémiol Santé Anim. 31–32 (1.01), 1–3.

Schlüter, H., Müller, T., 1995. Tollwutbekämpfung in Deutschland. Ergebnisse und Schlußfolgerungen aus über 10-jähriger Bekämpfung. Tierärztl. Umsch. 50, 748–758.

Scott, T.G., 1943. Some food coactions of the northern plains red fox. Ecol. Monogr. 13, 427–479.

Selhorst, T., Schlüter, H., 1997. Cost-benefit analysis of the oral immunization strategy for the control of rabies in fox populations. Epidémiol Santé Anim. 31–32 (10.20), 1–3.

Sikes, R.K., 1970. Rabies. In: Trainer, D.O. (Ed.), Infectious Diseases of Wild Mammals. Iowa State University Press, Ames, IA, pp. 3–19.

Silvertown, J.W., 1992. Introduction to Plant Population Ecology. Longman Scientific and Technical, Essex, England.

Skellam, J.G., 1951. Random dispersal in theoretical populations. Biometrika 38, 196–218.

Smith, G.C., 1995. Modelling rabies control in the UK: the inclusion of vaccination. Mammalia 59, 629–637.

Smith, G.C., Harris, S., 1991. Rabies in urban foxes *(Vulpes vulpes)* in Britain: the use of a spatial stochastic simulation model to examine the pattern of spread and evaluate the efficacy of different control regimes. Phil. Trans. R. Soc. London Ser. B 334, 459–479.

Starfield, A.M., Bleloch, A.L., 1991. Building Models for Conservation and Wildlife Management, 2. ed. Burgess International, Edina, MN.

Staubach, C., Tackmann, K., Löschner, U., Mix, H., Busse, W., Thulke, H.-H., Territo, B.M., Conraths, F.J., 1998. Geographic information system—aided analysis of factors potentially influencing the spatial distribution of *Echinococcus multilocularis* infections of foxes. In: Thrusfield, M.V., Goodall, E.A. (Eds.), Proceedings of the SVEPM Conference, Ennis, Ireland, pp. 40–47.

Steck, F., Wandeler, A., 1980. The epidemiology of fox rabies in Europe. Epidemiol. Rev. 2, 72–96.

Stephan, T., Jeltsch, F., Wiegand, T., Wissel, C., Breiting, H.A., 1998. Sustainable farming at the edge of Namib: analysis of land use strategies by a computer simulation model. In: Usó, J.L., Patten, B.C., Brebbia, C.A. (Eds.), Ecosystems and Sustainable Development. Computational Mechanics Publications, Southampton, pp. 41–50.

Storm, G.L., Montgomery, G.G., 1975. Dispersal and social contact among red foxes: results from telemetry and computer simulation. In: Fox, M.W. (Ed.), The Wild Canids, Reinhold, New York, pp. 237–246.

Stöhr, K., Meslin, F.M., 1996. Progress and setbacks in the oral immunisation of foxes against rabies in Europe. Vet. Record 139, 32–35.

Tackmann, K., Löschner, U., Mix, H., Staubach, C., Thulke, H.-H., Conraths, F.J., 1998. Spatial distribution patterns of *Echinococcus multilocularis* (Leuckart 1863) (Cestoda: Cyclophyllidea: Taniidae) among red foxes in an endemic focus in Brandenburg (Germany). Epidemiol. Infect. 120, 101–109.

Thiery, J.M., D'Herbes, J.M., Valentin, C., 1995. A model simulating the genesis of banded vegetation patterns in Niger. J. Ecol. 83, 497–507.

Thulke, H.-H., Tischendorf, L., Staubach, C., Müller, M.S., Schlüter, H., 1997a. Simulation based investigations on the consequences of changed rabies spreading within immunised fox populations. Epidémiol Santé Anim. 31–32 (1.02), 1–3.

Thulke, H.-H., Tischendorf, L., Staubach, C., Müller, M.S., Schlüter, H., 1997b. Neue Antworten zur Frage der weiteren Tollwutbekämpfung in Deutschland. Dtsch. Tierärztl. Wochenschr. 104, 492–495.

Tischendorf, L., 1997. Modeling individual movements in heterogeneous landscapes: potentials of a new approach. Ecol. Modell. 103, 33–42.

Tischendorf, L., Thulke, H.-H., Staubach, C., Müller, M.S., Jeltsch, F., Goretzki, J., Selhorst, T., Müller, T., Schlüter, H., Wissel, C., 1998. Chance and risk of controlling rabies in large-scale and long-term immunized fox populations. Proc. R. Soc. London Ser. B 265, 839–845.

Toma, B., Andral, L., 1977. Epidemiology of fox rabies. Adv. Virus Res. 21, 1–36.

Trewhella, W.J., Harris, S., McAllister, F.E., 1988. Dispersal distance, home range size and population density in the red fox (Vulpes vulpes): a qualitative analysis. J. Appl. Ecol. 25, 423–434.

Voigt, D.R., Tinline, R.R., Broekhoven, L.H., 1985. A spatial simulation model for rabies control. In: Bacon, P.J. (Ed.), Population Dynamics of Rabies in Wildlife, Academic Press, London, pp. 311–349.

Wandeler, A.I., 1980. Epidemiology of fox rabies. In: E. Zimen (Editors), Biogeographica Vol. 18—The Red Fox, Dr. W. Junk, The Hague, pp. 237–250.

Wandeler, A.I., Capt, S., Kappeler, A., Hauser, R., 1988. Oral immunization of wildlife against rabies: concept and first field experiments. Rev. Infect. Dis. 10, S649–S653.

Weiner, J., 1995. On the practice of ecology. J. Ecol. 83, 153–158.

White, P.C.L., Harris, S., Smith, G.C., 1995. Fox contact behavior and rabies spread: a model for the estimation of contact probabilities between urban foxes at different population densities and its implications for rabies control in Britain. J. Appl. Ecol. 32, 693–706.

White, P.C.L., Saunders, G., Harris, S., 1996. Spatio-temporal patterns of home ranges use by foxes in urban environments. J. Anim. Ecol. 65, 121–125.

Wiegand, K., Jeltsch, F., Ward, D., Rohner, C., 1998. Decline of the Negev's Acacias—a spatially explicit simulation model as an aid for sustainable management. In: Usó, J.L., Patten, B.C., Brebbia, C.A. (Eds.), Ecosystems and Sustainable Development. Computational Mechanics Publications, Southampton, pp. 63–72.

Wiegand, T., Naves, J., Stephan, T., Fernandez, A., 1998. Assessing the risk of extinction for the brown bear (*Ursus arctos*) in the Cordillera Cantabrica, Spain. Ecol. Monogr. 68, pp. 539–571.

Wissel, C., 1992a. Aims and limits of ecological modelling exemplified by the island theory. Ecol. Model. 63, 1–12.

Wissel, C., 1992b. Modelling the mosaic cycle of a Middle European beech forest. Ecol. Modell. 63, 29–43.

Wolfram, S., 1986. Theory and Application of Cellular Automata. World Science, Singapore.

Yachi, S., Kawasaki, K., Shigesada, N., Teramoto, E., 1989. Spatial patterns of propagating waves of fox rabies. Forma 4, 3–12.