Spatial Epidemic Models: Theory and Simulations

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Denis Mollison
Kari Kuulasmaa[1]
Department of Actuarial Mathematics and Statistics,
Heriot-Watt University,
Riccarton, Edinburgh, Scotland

I. Introduction

The aim of this chapter is to survey theoretical results on spatial models for epidemics, and to discuss how they can help us in understanding, and if possible controlling, diseases such as rabies. In the first half of this chapter we give a general survey of work on spatial models for epidemics. In the second half we discuss some simulations carried out by Kuulasmaa (1983), aimed at exploring general aspects of endemic fox rabies; these provide evidence of the importance of incorporating stochastic and spatial features. We conclude with some discus-

[1]Present address: National Public Health Institute, Mannerheimintie 166, SF-00280 Helsinki, Finland.

sion of the relations between simple general spatial models as considered here, and more detailed specialised models such as those described in other chapters by Bacon, Ball, and Voigt and Tinline.

We first introduce the three main aspects of epidemics with which we shall be concerned, namely *thresholds, velocities* and *endemicity*.

A population is said to be 'above threshold' for a particular disease if, once started, the disease has a chance of spreading widely through the population; and 'below threshold' if it will die out with only a small proportion of the population infected. The practical problems are to identify when a population is above threshold when threatened by a particular disease such as rabies, and to estimate whether various control strategies, such as vaccination or culling, could bring the population below threshold.

The second question, of velocities, relates to how fast the disease will spread if it does become established (in an above threshold population), and how this depends on factors such as the territorial range of individuals. Practical questions include the likelihood of success of a spatially selective control strategy, such as clearing a control zone of a certain width in front of the epidemic.

The third question, of endemic behaviour, is the most difficult theoretically because our models must allow for the introduction of new susceptibles, without which the disease would die out. Here again the most important practical questions relate to the possible elimination of the disease. However, because of the greater difficulties of modelling, we need first to improve our understanding of spatial endemic models. For instance, the pattern of endemic fox rabies in Europe shows much spatial heterogeneity ('wandering patches', see e.g. Sayers, Chapter 10, this volume), and it is an important question how much this is due, if at all, to heterogeneities in the population.

II. Theory of Spatial Models

A. BASIC SPATIAL MODELS

We begin by introducing a simple spatial epidemic model motivated by the study of fox rabies. Rabies, especially among the fox population of western Europe, is a disease which spreads through local interactions among territorial animals. It therefore seems important that our model should be *spatial* and *stochastic*, and should include the *carrying capacity* or some other kind of limit on the population density. Models which are non-spatial or deterministic, or which allow populations of unbounded density, are not fully adequate (Mollison, 1981); although they have been and will continue to be extremely useful as stepping stones towards better, more complex, models.

We envisage space as a two-dimensional array of sites (see Fig. 1); in the

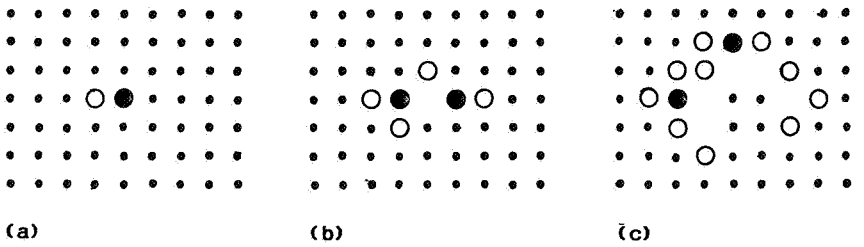

Fig. 1. The spatial epidemic model of Equation (1). (a) Part of the two-dimensional array of sites shown just after the start of an epidemic outbreak: this started with a single introduced infectious individual (●), who has now infected one neighbour (now incubating, marked ○). All other sites are still occupied by susceptibles (•). (b) The same sites, a little later: the site originally infected is now vacant ( ), and there are several incubating (○) and infectious (●) individuals. (c) Still later: note that two sites which had become vacant have now been recolonised by susceptibles.

context of fox rabies, these may be taken to represent square territories. Each site may either be empty (E), or occupied by an individual who may be susceptible (X), incubating (I), or infectious (Y). The development of the epidemic is then prescribed by stochastic change rates (formally 'instantaneous transition rates' or *ITR*s: when we say that a possible change has ITR equal to λ, we mean that in any short interval of time dt, the probability of its occurring is λdt), as follows:

	Change	Change rate
Infection	$XY \to IY$	$\beta/4$
Becoming infective	$I \to Y$	σ
Death	$Y \to E$	α
Recolonisation	$EX \to XX$	$r/4$

(1)

Here β is the overall rate at which an infective makes contacts; the change rates α and σ correspond exactly to those of the simple deterministic non-spatial model described in Chapter 9 [Equation (1)], this volume, and lead to a generation gap with probability distribution given by Equation (4) of Chapter 9 (a sum of two exponential distributions), with mean $\tau = 1/\alpha + 1/\sigma$. The 'recolonisation' term represents population regrowth, net of natural mortality (see later).

A simpler alternative model, corresponding to Equation (2) of Chapter 9, omits the incubating state:

	Change	Change rate
Infection	$XY \to YY$	$\beta'/4$
Death	$Y \to E$	α'
Recolonisation	$EX \to XX$	$r/4$

(2)

Here the infectious period and the generation gap both have exponential distribution with mean $\tau = 1/\alpha'$.

In both models, the spatial element is involved in the other two types of

change. Thus *XY* indicates a pair of neighbours, one of whom is susceptible and the other infectious; the first type of change represents infection of the former by the latter. Since each individual has four neighbours (see Fig. 1), each infectious individual will be making potentially infectious contacts at an overall rate β [for Model (1), β' for Model (2)], these contacts being divided equally among its four neighbours, and of course succeeding only if the neighbour chosen is susceptible. The parameter β thus corresponds to βN, in the simple non-spatial models described in Chapter 9 (Equations 1–3). Similarly, the parameter r, which represents the per capita net population growth rate at low densities, corresponds roughly to the parameter r of those models (but see comments in Section III,C). An important concept in spatial models is the *contact distribution* (Mollison, 1972, 1977), which describes the spatial distribution of the potentially infectious contacts made by an infectious individual. Here we have taken the contact distribution as concentrated on an individual's four nearest neighbours. This simple distribution is probably adequate at least for the initial exploration of endemic conditions, but if we are interested in the velocity of spread of an epidemic, as in Section II,C, we will need to allow for longer range contacts as well. In models for endemic conditions, we need a secondary contact distribution as well, to describe the process of recolonisation of empty sites: here this has also been assumed to be a nearest-neighbour distribution, and again in a more detailed analysis we should consider the effect of allowing for longer range movements by recolonising animals.

The type of model introduced here is about as simple as seems possible for a stochastic spatial model suitable for endemic disease. Ideally, one would like to achieve a broad understanding of such models, and then introduce further realistic details, such as seasonal and social variability. However, even such simple models are not well understood, and initially we shall go in the other direction, and consider the problems of thresholds and velocities in the context of even simpler models.

B. SPATIAL MODELS: THRESHOLDS

The 'threshold' for a disease has been defined previously as the dividing line between conditions in which the disease will die out with only a small proportion of the population infected, and conditions in which there is a chance of the disease spreading widely through the population. This should perhaps be called the 'pandemic threshold', to distinguish it from the 'endemic threshold', which may be defined as the initial conditions such that the disease will persist indefinitely. The endemic threshold, to which we shall return in Section II,D, will depend crucially on the rate of regrowth of the susceptible population, and will in general be higher than the pandemic threshold.

In considering the basic (pandemic) threshold it seems reasonable in the first

instance to neglect the regrowth of susceptibles, since this is slow compared with the initial velocity of the disease. (A detailed analysis of this requires consideration of the rate of advance of the front, its depth, and the typical dispersal distances of each year's young foxes.) If we do neglect the regrowth of susceptibles, we can simplify our analysis of threshold conditions in one important respect. It is then possible at least in the slightly simplified models usually studied, to make a *list* of each individual's potential contacts (in a stochastic model, this will involve random choices) without having to consider time or whether that individual will in fact be infected. The set of those eventually infected by the disease then consists precisely of those for whom we can find a 'chain of infection', with each individual in the previous one's list, which begins with one of those initially infected. Thus, if we are only interested in who will and who will not be infected, as we are when considering thresholds, we need take no direct account of the time structure.

The classic threshold theorem is due to Kermack and MacKendrick (1927). It refers to a non-spatial deterministic epidemic model with homogeneous mixing, and says that a pandemic will occur if and only if the basic reproductive rate C, which is essentially the average size of each individual's list, is greater than unity. The further the population is initially above threshold, the further the remaining susceptibles at the conclusion of the pandemic will be below threshold (Kermack and Mackendrick, 1927; Kendall, 1965).

A similar stochastic model behaves similarly, except that there is a chance that the disease will fail to get established even though the population is above threshold. The probability of failure can be estimated by comparison with a simpler model which allows an unlimited pool of suceptibles, and is approximately C^{-Y_0}, where Y_0 is the number initially infected (Whittle, 1955; Kendall, 1965).

In models incorporating the introduction of fresh susceptibles, the disease may either settle into endemic equilibrium, or into a cyclic pattern, with each peak of infection behaving much like one of Kermack and McKendrick's pandemics (Bartlett, 1960; see also Chapters 6, 7 and 9 of this volume).

We next turn to models for spatially distributed populations. Most work on the velocity of epidemics has been restricted to one-dimensional models (see next section). However, it can be shown for quite a general class of models that pandemics in one dimension are impossible, provided only that infectious cases are subject to eventual removal (F. Kelly in discussion of Mollison, 1977, pp. 318–319). While this is a rather theoretical result (there does appear to be a 'pseudo-threshold' above which the disease can spread a great distance), it suggests that for a realistic consideration of thresholds we do need to study two-dimensional models.

We consider here only models without recolonisation of empty sites. Perhaps the simplest case is that where the infectious period is of fixed length, rather than

exponentially distributed as in Models (1) and (2) of the previous section. This is because in this case the infections made by an individual are statistically independent, each having probability p say. The epidemic model is therefore formally equivalent to the well-known *bond percolation* model of physics, which has threshold value $p_0 = \frac{1}{2}$ (Broadbent and Hammersley, 1957; Kesten, 1980). The threshold value of the basic reproductive rate is then $C_0 = 4p_0 = 2$. If, on the other hand, the infectious period is extremely variable, we tend towards the following case: with probability p the infectious period is very long and the individual infects all four neighbours, while with probability 1-p, the infectious period is very short and it infects none. This corresponds to *site percolation*, for which the critical value p_0 has been estimated to be 0.6; thus in this case C_0, which is again $4p_0$, is equal to 2.4. For intermediate infectious periods, we may suspect that C_0 will lie between these two values, i.e. between 2 and 2.4. By using a comparison technique for epidemic models which differ only in the distribution of their infectious periods, Kuulasmaa (1982) has shown that this is true for all such distributions. In particular, it is true for the exponential distribution, as in Models (1) and (2) (without recolonisation, i.e. with $r = 0$), and simulations show that for this case $C_0 \approx 2.12$.

These results refer to the case where each individual only interacts with its four nearest neighbours. Asymptotic results (e.g. Ball, 1983) suggest that C_0 will be closer to unity when the number of potential contacts is larger.

C. SPATIAL MODELS: VELOCITIES

The velocity of spread of a disease will clearly depend to a large extent on the *contact distribution*, which describes the spatial distribution of the potentially infectious contacts made by an individual. This dependence has only been studied in any depth for very simple one-dimensional models, namely *simple epidemics*, in which infected individuals remain permanently infective: this corresponds to Model (2) with $\alpha' = 0$ (r is then irrelevant, since sites never become vacant). This work is reviewed in Mollison (1977). There has also been some thorough work on two-dimensional models with the contacts restricted to an individual's nearest neighbours, mostly on percolation models; this field is reviewed by Smythe and Wierman (1978).

Most work even in the one-dimensional case has been on deterministic models, in the form of nonlinear convolution or diffusion equations. These can be shown (McKean, 1975; Mollison, 1977) to be closely related to 'linear' stochastic models, which make the simplifying but unrealistic assumption of an unlimited pool of susceptibles, and in which the density of infectives consequently can grow exponentially. It is thus perhaps not surprising that these deterministic models turn out to be a poor guide to the behaviour of more realistic stochastic models.

The earliest work on velocities appears in two classic papers which appeared independently in 1937, one by Fisher and the other by Kolmogoroff, Petrovsky and Piscounov. Their work concerned the advance of an advantageous gene but translates fairly straightforwardly to epidemic models. They used a diffusion approximation rather than a contact distribution, which yields a characteristic velocity of $\beta\sigma\sqrt{2}$; here β is the rate at which individuals make contacts, and σ the standard deviation of the contact distribution. For the exact model the characteristic velocity is rather higher, varying between about $1.5\beta\sigma$ and $1.85\beta\sigma$ for contact distributions with mainly local concentration; but for more widely spread distributions, the characteristic velocity may be infinite, so that the epidemic spreads at arbitrarily increasing speed (Mollison, 1972, 1977; the exact condition for finite velocity is that the contact distribution should have exponentially bounded tails). In cases where the velocity is finite, the behaviour of the corresponding linear stochastic model is unrealistically well-behaved (see Mollison, 1977, esp. pp. 323–324).

For the stochastic simple epidemic model itself, the velocity can be shown to be infinite only if the contact distribution has infinite standard deviation. Simulations confirm that the manner of advance is less regular than for the linear model; indeed, for intermediate contact distributions (with finite standard deviation but with tails not exponentially bounded), the epidemic appears to advance in a mixture of steady progress and 'great leaps forward' (Mollison, 1972). Where the linear model (and the deterministic simple epidemic) have finite velocity, however, the stochastic simple epidemic advances in a relatively steady manner, at a rather lower velocity than the linear process. The difference in velocity appears to be greatest when an individual's potential contacts are concentrated on a small number of neighbours, the ratio being over 3 to 1 in the extreme case where an infective can only contact a single individual to either side.

In two dimensions, for the linear stochastic model and its associated deterministic models, the velocity in each direction can be found simply from the one-dimensional analysis, and thus varies between about $1.5\beta\sigma'$ and $1.85\beta\sigma'$ for contact distributions with exponential tails, where σ' denotes the standard deviation of the contact distribution in the direction considered (that is, of the projection of the contact distribution in that direction).

A number of results have been obtained for simple stochastic epidemic models, particularly percolation models, showing that the infected area expands with a characteristic shape and velocity; if, as in lattice models, the contact distribution is direction dependent, the velocity will not be exactly the same in each direction (Richardson, 1973; Mollison, 1978; Schürger, 1980). Actual velocities have only been estimated from simulations. As in one dimension, they appear to be significantly lower than for linear or deterministic models. For instance, for nearest neighbour contact distributions, simulations suggest that the velocity is $0.83\beta\sigma'$ on a square lattice and $0.89\beta\sigma'$ on a hexagonal lattice; on an irregular

lattice the velocity is found to be a little higher (P. J. Green, in discussion of Mollison, 1977, pp.317–318). [Incidentally, for nearest neighbour models with just one individual at each site, the velocity is proportional to the *crinkliness* of the boundary of the infected area (Mollison, 1974; see also Downham and Green, 1977).] As to contact distributions without exponentially bounded tails, the conditions for the simple epidemic in two dimensions to have finite velocity are unknown.

The work discussed so far concerns continuous-time models, in which an infective makes contacts at rate β from the moment at which it becomes infected. What little is known of simple epidemics with different time structure, as for instance with a fixed generation gap between the infection times of an infective and its victims (i.e. a discrete-time model), suggests that similar results will hold as regards velocities and the conditions to ensure a finite velocity. However, velocities will no longer be simply proportional to the infectiousness of individuals, as measured by β or some similar parameter; the velocity is likely to rise more slowly than proportionately, the exact relation depending on the contact distribution.

For epidemics with removal [Model (2) without recolonisation, i.e. $r = 0$], the velocity of the linear and deterministic models is approximately $\sqrt{(1 - \alpha/\beta)}$ times the velocity of the model without removal (D. G. Kendall, in discussion of Bartlett, 1957, pp. 64–67; Atkinson and Reuter, 1976). For the nonlinear stochastic model, however, unpublished simulations by one of the authors suggest that the reduction in velocity is rather greater. Some of these simulations, incidentally, show a pattern in which the front breaks down into a number of 'arcs of infection' (as conjectured by D. G. Kendall, in discussion of Bartlett, 1957, pp. 64–67), with no infectives on the stretches in between. However, this seems to occur principally in simulations where the epidemic is dying out.

D. SPATIAL MODELS: ENDEMICITY

In discussing thresholds in Section II,B, we restricted attention mainly to the initial spread of a disease in the case where removed individuals are not replaced. If the infection is to become *endemic*, it is of course essential that new susceptibles should be introduced.

One of the simplest spatial models exhibiting endemic behaviour is the 'contact process' introduced by Harris (1974) (there are two surveys by Griffeath, 1979, 1981; see also Durrett and Griffeath, 1982). This process may be regarded as a simplified version of Model (2) in which infectives, instead of being removed and leaving vacant sites, simply recover and become susceptible again. We thus have the following:

	Change	Change rate
Infection	$XY \to YY$	$\beta'/4$
Recovery	$Y \to X$	α'

(3)

[Note that this is nearly the same as is obtained by setting $r = \infty$ in Model (2).] For this process the parameter $\lambda = \beta'/4\alpha'$ has a threshold value λ_0, which (for a two-dimensional model) is known to lie between $\frac{1}{3}$ and 1 [Holley and Liggett, 1978; Harris, 1974; the lower bound here can be improved marginally, to 0.359 (Griffeath, 1975, p. 191)]. Above this threshold value, the process may tend to a stochastic equilibrium in which both infectives and susceptibles are present.

An apparently rather similar model, but exhibiting very different behaviour, is that introduced by Williams and Bjerknes (1972) for two competing cell populations. In this model 'susceptibles' can replace neighbouring infectives, so that the process is symmetrical between the two types:

	Change	Change rate
Infection	$XY \to YY$	$\beta'/4$
'Recovery'	$Y \to X$	α'

(4)

Even though this model includes the introduction of new susceptibles, it appears to have only trivial equilibria. If $\beta < \alpha$ the infection is certain to become extinct (Kelly, 1977b) (we are assuming that the initial set of infectives is finite). If $\beta > \alpha$ the infection may survive forever, but in that case the infected area expands as an approximate disk of linearly growing radius (i.e. at fixed velocity), so that all sites eventually become infected (Bramson and Griffeath, 1980, 1981).

Few theoretical results are available for even the simplest models for endemic disease, such as our Model (2). One technique which is worth mentioning is that of 'balance equations,' in which we consider the density of each type of individual and of each type of pair of neighbours: for instance, let $\pi(Y)$ denote the proportion of infected sites, and $\pi(XY)$ the density of neighbouring XY pairs. In endemic equilibrium, if such is possible, the creation and removal of infected cases must be in balance: thus, taking into account the change rates of Model (2), we have that

$$\alpha'\pi(Y) = (\beta'/4)\pi(XY) \tag{5}$$

Such equations, together with the fact that all such proportions must lie between 0 and 1, can be used to find bounds on the parameter values for which endemic equilibrium is possible [note that $\pi(XY)$ = density = 2 × (proportion of XY pairs), so that $0 \leqslant \pi(XY) \leqslant 2$]. For instance, we can show that $\lambda_0 > \frac{1}{3}$ for Model (2) (Kuulasmaa, 1983; this approach was applied to Harris's contact process by Clifford and Sudbury, 1979).

In the next section we report the results of some simulations. One final technique which is worth mentioning derives from physics, and lies in a sense intermediate between theory and simulations. In this approach a specific model, usually a power law, is derived heuristically, and parameter values are then estimated from simulations. This yields surprisingly precise values for param-

eters such as λ_0 for various percolation type processes (Grassberger, 1983). While this approach is not strictly rigorous, it must at least be regarded as producing very interesting conjectures.

III. Simulations of a Spatial Model for Endemic Fox Rabies

A. METHODOLOGY

Simulations of the endemic models introduced previously [Models (1) and (2) of Section II,A] have been performed on a finite rectangular area (typically of 60 × 60 sites). To avoid edge effects, we assume that the pattern repeats outside the rectangle considered. (In precise mathematical terminology, we identify opposite pairs of edges of the rectangle, so that our area is topologically a torus.)

It is convenient to look on the process of infection from the susceptible's point of view. At any moment, each susceptible is independently subject to an infection at rate $\beta/4$ times the number of neighbouring infectives. In this way unsuccessful infections are omitted, and hence a considerable amount of computing time is saved. The filling of vacant sites by reproduction of neighbouring susceptibles works in a similar way. In Model (1), incubating sites are liable to becoming infected, with change rate σ, and infectives to becoming vacant at rate α; in Model (2) we only have infective sites, which are liable to become vacant at rate α'.

Since, given the present state of the process, the types of the sites change in independent Poisson processes, the time to the next change in the process has exponential distribution with mean 1/(sum of the change rates of the sites). Furthermore, the probability that the next change occurs at a given site is the ratio of the change rate for that site to the total change rate, independently of the waiting time. Hence we can first decide what is the next change and then, if we are interested in it, find out the time of this change. In practice, since the number of changes will be large in any period of interest, we usually get very accurate timing by taking the time between successive changes to equal the mean value, 1/(total change rate).

The simulation algorithm used is the following:

1. Give the necessary initial values. The main arrays needed are TYPE and RATE, where TYPE(I,J) indicates the current type of site (I,J), and RATE(I,J) indicates its change rate (ITR). Also, a variable TOTALRATE for the total change rate is needed. We store the time in variable TIME.
2. Let TOTALRATE = $\Sigma_{I,J}$ RATE(I,J).
3. Choose the site, (I,J) say, where the next change occurs; the probability of choosing this particular site is RATE(I,J)/TOTALRATE.
4. Replace TYPE(I,J) by its new value and update RATE for (I,J) and its neighbours.

5. Increase TIME, for exact timing by a random value from the exponential distribution of mean 1/TOTALRATE, or for approximate timing, simply by 1/TOTALRATE.
6. Output as required: for instance, proportions of different types of sites, or a plot of the state of the process to printer, VDU or film.
7. To continue the simulation, go to step 2; otherwise
8. The simulation is concluded.

B. SIMULATION RESULTS

Simulations of both models were carried out for a wide range of parameter values. These are presented here with an assumed timescale such that the generation gap of the disease matches rabies data, as were the non-spatial models of Chapter 9. Thus for Model (1) we take $\sigma = 13$, $\alpha = 73$ (year^{-1}); while for Model (2) we take $\alpha' = 11$. We shall plot the infectivity in terms of $\mu = \beta/\alpha$ [for Model (1), $= \beta'/\alpha'$ for Model (2)], since this ratio to a good approximation determines the basic reproductive rate of the infection, $C \approx \mu/(1 + \mu/4)$ (the approximation involved here is that we neglect the possibility of infecting two or more susceptibles in succession on the same neighbouring site).

Both models show broadly similar patterns. If the infection is started from an initial focus, it spreads at first with a fairly regular front, behind which occurs a 'silent' phase (compare Macdonald, 1980, Fig. 3.5, showing the advance of rabies in France). This regularity disappears in the subsequent endemic phase, and one can no longer observe any direction for the infection, except very locally.

The simulations indicate that there is a unique endemic equilibrium for certain parameter values. Figure 2 shows the estimated proportions of susceptibles and infected cases in this equilibrium. [Note that for Model (1) the proportion of incubating cases in equilibrium is always $\frac{73}{13} \approx 5.6$ times the proportion of infective cases.] The size of the simulation area has no observed effect on the mean proportions, but it does affect their fluctuation (see below).

Except possibly for very low values of r, there is a clear critical value C_0 of the basic reproductive rate, such that for $C < C_0$ extinction is certain. For larger values of r, the critical value C_0 is close to its theoretical lower bound of $\frac{4}{3}$ (see Section II,D); for values of r appropriate to fox rabies ($r < 1$), C_0 is considerably higher.

When r is large, and C near C_0, the proportion of empty sites is negligible. This supports the conjecture that for such values the process can be approximated by Harris's contact process. In particular, we would then have that $\inf_r C_0(r)$ is the same as the critical C_0 of Harris's process, for which the best known lower bound is $4 \times 0.359 = 1.44$.

Figures 3 and 4 show states of the two models in apparent equilibrium, in each case for two different choices of parameters. These patterns are again reminis-

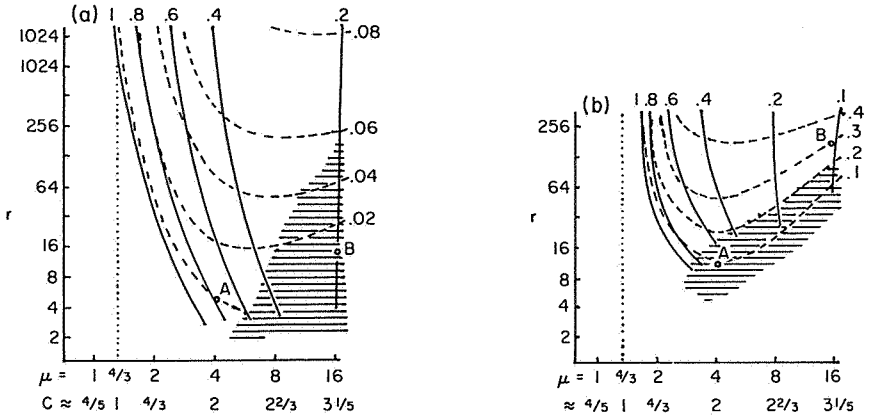

Fig. 2. Estimated proportions of susceptibles (——) and infected cases (– – –) in endemic equillibrium, for Models (1) and (2). In the shaded regions fluctuations of the proportions are significant on a 60 × 60 lattice. (a) Model (1), with $\alpha = 73$, $\sigma = 13$. The horizontal scale is given in terms both of $\mu = \beta/\alpha$ and of the (approximate) basic reproductive rate $C \approx \mu/(1 + \mu/4)$. Simulations with the parameter values marked A and B are shown in Figs 3 and 4. (b) Model (2), with $\alpha' = 11$. Similarly, with $\mu = \beta'/\alpha'$.

cent of those observed in real endemic conditions (compare for instance, Macdonald, 1980, Figs 3.5a and 5.18).

The relative proportions, in equilibrium, of each type of site deserve further comment; we shall denote them here simply by (e.g.) X rather than $\pi(X)$. The basic balance equations for equilibrium are then, for Model (1),

$$\beta XYq_{XY} = \sigma I = \alpha Y = rEXq_{EX} \tag{6}$$

and for Model (2),

$$\beta' XYq_{XY} = \alpha' Y = rEXq_{EX} \tag{7}$$

Here q_{XY} denotes the density of neighbouring XY pairs relative to the expected value assuming homogeneous mixing ($= 4XY$), and similarly for q_{EX}; setting the q's both $= 1$ we thus recover the balance equations for the non-spatial models of Chapter 9. The level of prevalence, $(I + Y)/X$, is then not $Er\tau$ as found for the non-spatial model, but q_{EX} times as much. Typically, we find a lower level of prevalence; for example, for the parameter values of Fig. 3a, $q_{EX} \approx 0.4$, and the level of prevalence averages 3%, compared with an expected value from the non-spatial analysis of nearly 8%.

From Eq. (6) we can also deduce that $X = 1/(\mu q_{XY})$. The tendency of Xs and Ys to avoid each other appears even more marked than that of Xs and Es. Again in Fig. 3a, we find that $q_{XY} \approx 0.3$, so that the proportion of susceptibles is approximately 80%, rather than 25% as expected assuming homogeneous mixing. The low values of q_{XY} can be attributed to a combination of factors. A new

infective is likely to begin with fewer than average susceptible neighbours (for a start, the site which infected it is unlikely to be susceptible again yet); and if it infects one it is similarily unlikely to get a replacement, so that an individual who has been infectious for some time is even less likely to have susceptible neighbours (this latter factor has already been referred to in explaining why $C < \mu$). It is easy to guess from this that $q_{XY} \leqslant \frac{3}{4}C/\mu$, and hence $X \geqslant \frac{4}{3}C^{-1}$, $= 67\%$ in the present case.

When the parameters approach the shaded regions of Fig. 2, the proportions begin to fluctuate until finally extinction occurs. If r is decreased the infection eventually becomes extinct, while if C is increased the population becomes extinct. However, in this parameter region the proportion of infectives is small, and the few that there are tend to group together. Thus, the fluctuations, and perhaps the ensuing extinction, may be due only to the finite size of the simulation area. Interestingly, the more realistic model [Model (1)] appears stable down to lower values of r.

C. DISCUSSION OF SIMULATIONS

The models we have simulated were chosen to include only the most basic features essential for a study of spatial patterns of an endemic disease of territorial animals. We have omitted many features, and made considerable approximations in the features we have included. Hence, before we interpret the results of our simulations, we must discuss some of the shortcomings of our models.

We have apparently neglected natural mortality. However, this is largely taken into account if we assume that most vacancies occurring through natural mortality are soon filled by the offspring of nearby sites, thus keeping the population at the carrying capacity, at least in rabies free areas.

We have only been able to simulate with values of r down to about 2 year^{-1}, even for Model (1). As mentioned in the previous section, we find that the level of prevalence fluctuates for small values of r, but we conjecture that this may only be due to the finite size of the simulation area and the consequent small total population of infectives in these cases; to investigate the stability of the equilibrium for smaller values of r would require simulations on a considerably larger lattice. On the face of it, this is a considerable shortcoming of our present simulations, since values of r appropriate to foxes are about 0.5 year^{-1}. However, against this we must note that our present assumption that territories can only be recolonised by their immediate neighbours 'disenfranchises' a large proportion of susceptibles, whose offspring might in reality be prepared to travel considerable distances in search of empty sites. Even at low population densities, the susceptibles tend to group together, and thus in our model the offspring of the individuals at the edges of such a group really represent the offspring of the entire group. Thus the lower values of r in our simulations ($r = 2$–5, say) may in fact

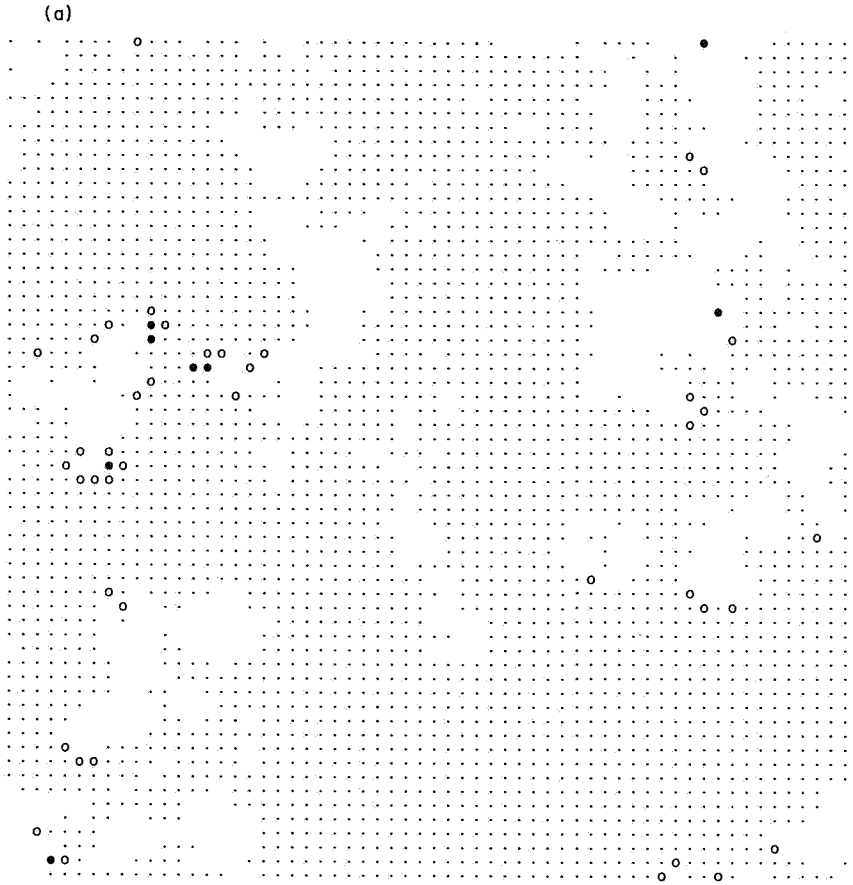

Fig. 3. (a) Typical state of Model (1), in apparent equilibrium, for parameter values $r = 4.4$, $C = 2$ (marked A in Fig. 2a). Symbols: susceptible (•), incubating (○), infectious (●), vacant ( ). (b) The same, but with $r = 14.6$, $C = 3.2$ (B in Fig. 2a).

reasonably represent fox population regrowth. However, this aspect of modelling clearly requires further consideration, particularly in respect of how far foxes travel to find new territories and how efficient they are at identifying vacant territories.

We have generally only allowed for one individual at each site, whereas fox territories in reality are occupied by family groups. Some simulations were also done of a model with two individuals per site, and for a relatively high internal contact rate the results were qualitatively similar to those for the basic model. This suggests that our model will approximately apply to sites occupied by family groups; though in interpreting results, we must allow for likely differences: for instance, the generation gap for family to family infections may be a

Fig. 3. (*Continued.*)

compound of several individual generation gaps, and will therefore have a somewhat higher mean (see Chapter 8, this volume). In favour of our basic model, we may note that its assumption that the directions of contacts made from a site are independent is rather more reasonable if these represent the contacts made by a family rather than by a single individual.

We have not allowed for heterogeneity between individuals, and in particular for the difference between settled and itinerant foxes. In so far as the latter are important, we clearly need to consider contact distributions allowing longer range contacts. It would in any case be interesting to examine how threshold levels (C_0) and endemic patterns depend on the contact distribution. For a start, we might guess that the scale of endemic patterns, and the velocities with which they spread, will be roughly proportional to the standard deviation of the contact distribution.

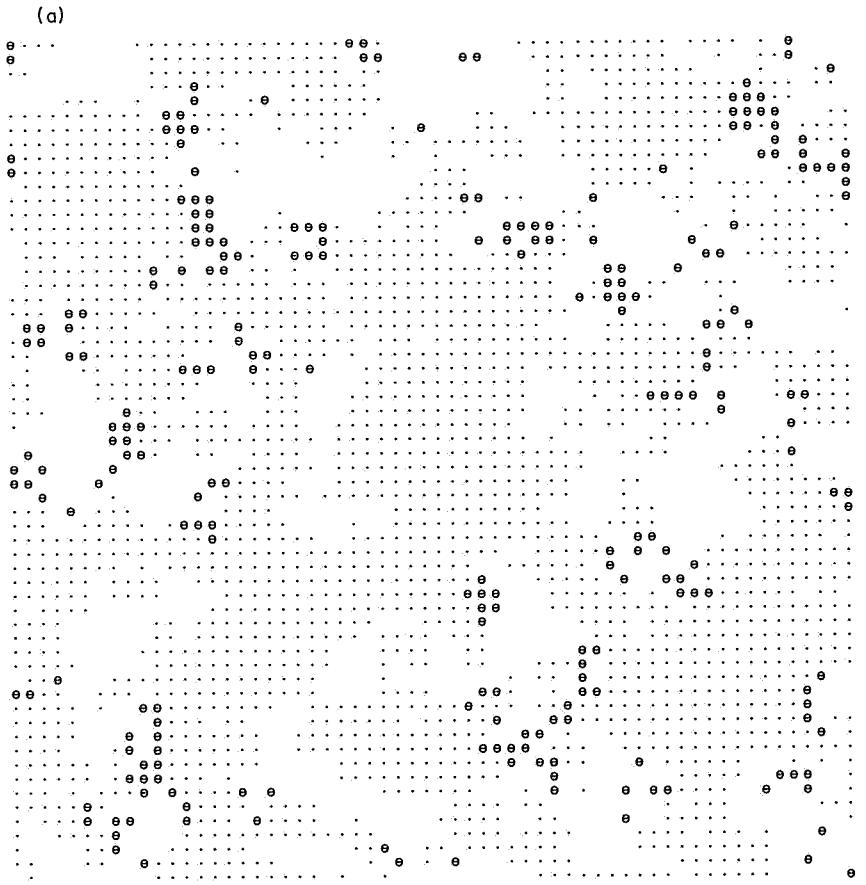

Fig. 4. (a) Typical state of Model (2), in apparent equilibrium, for parameter values $r = 11$, $C = 2$ (marked A in Fig. 2b). (b) The same, but with $r = 176$, $C = 3.2$ (B in Fig. 2b). Symbols: susceptible (•), infectious (⊖), vacant ( ).

Even allowing for all these defects, and other neglected factors such as seasonal variation, we can draw some general conclusions from these simulations. They show how an epidemic which begins by advancing in a regular manner with a fairly well defined velocity can break up into an endemic pattern of quite large wandering 'patches of infection', without any need to invoke geographic or social heterogeneity; that is, we can have heterogeneous behaviour in a homogeneous environment.

The proportions of the various types of individual in endemic equilibrium differ significantly from those expected from non-spatial models; in particular the proportion of vacant sites is much smaller.

The most interesting question raised is whether oscillations in the level of

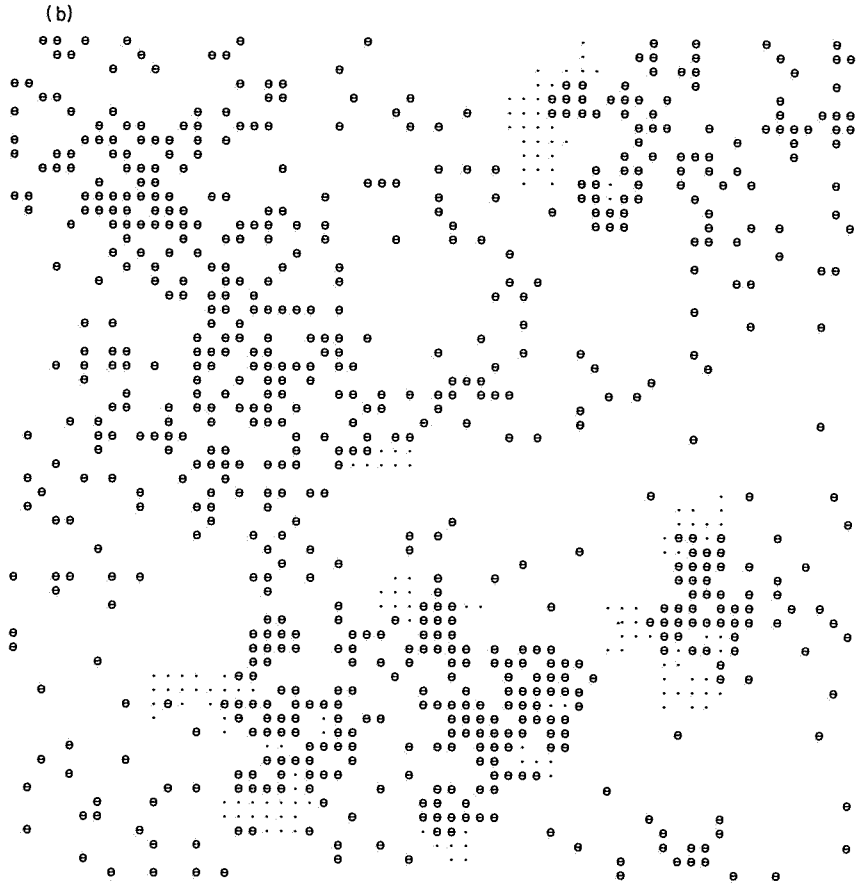

Fig. 4. (*Continued.*)

prevalence can be genuinely cyclical as in the non-spatial case (see Chapters 6 and 9, this volume), or are merely the consequence of considering a large scale random pattern over too small an area; some support for the latter view comes from Macdonald's observation that fluctuations appear more marked in data from small countries (Macdonald, 1980, Table 3.1).

IV. Discussion

The formidable task of developing models for endemic disease may be compared to building a house in a hurry. Practical workers insist on building a complete house, and are not too worried that it may need replacing later. Theoreticians insist on building reliable foundations, and are not too worried if

the house is never finished. Both points of view of course have their merits, and ideally we need to combine these.

This chapter lies towards the theoretical end of the spectrum, though it has been framed where possible in terms of parameters with straightforward ecological interpretations, such as the basic reproductive rate and contact distribution. If we are to use complex models to explain rather than just imitate reality, we need to understand which assumptions are crucial for particular results: we need to be able to take a model apart and see what makes it tick. If this chapter assists such understanding, it will have served its purpose as part of the foundations.

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