Host–pathogen systems in a spatially patchy environment

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SUMMARY

A discrete model for a host–pathogen system is developed and is used to represent the dynamics in each patch within a landscape of $n \times n$ patches. These patches are linked by between-generation dispersal to neighbouring patches. Important results (compared to similar 'coupled map lattice' studies) include an increase in the likelihood of metapopulation extinction if the natural loss of pathogen particles is low, and the observation of a radial wave pattern (not previously reported) where the wavefront propagates uniformly from a central focus. This result has additional significance in that it permits the system to exhibit 'intermittency' between two quasi-stable spatial patterns: spirals and radial waves. With intermittent behaviour, the dynamics may look consistent when viewed at one time scale, but over a longer time scale they can alter dramatically and repeatedly between the two patterns. There is also evidence of clear links between spatial structure and temporal metapopulation behaviour in both the intermittent and 'pure' regions, verified by results from an algorithmic complexity measure and a spectral analysis of the temporal dynamics.

1. INTRODUCTION

Recently, Comins *et al.* (1992) (see also Hassell *et al.* 1991) examined a model for a host–parasitoid system in a spatially sub-divided, or patchy, environment, and found a remarkable range of dynamical behaviour when: (i) within each patch the host–parasitoid dynamics are represented by the Nicholson–Bailey difference equations (which are unstable with diverging oscillations leading to extinction); and (ii) host and parasitoid movement are incorporated between generations by allowing a specified fraction of individuals from each patch to move to 'neighbouring' patches. Such a modelling process is termed a coupled map lattice. The density of the host–parasitoid subpopulations in the two-dimensional array of patches could exhibit so-called spiral waves, spatial chaos, or a 'crystal-lattice' pattern, all of which allowed the metapopulation (the sum of the subpopulations from all patches) to persist. However, regardless of the pattern exhibited there was a slight possibility of the metapopulation becoming extinct. Hence, such an approach allows investigation of the potential role that simple but explicit spatial dynamics may play not only in the spatial patterns exhibited by populations across a landscape but also in the emerging temporal dynamics of the metapopulation as a whole.

Further studies have generalized and adapted these ideas, in the context of single species models (Bascompte & Sole 1994; Hastings & Higgins 1994), of predator prey systems (Pascual 1993), of host–parasitoid systems with density-dependent parasitoid aggregation (Rohani & Miramontes 1995) and of the evolutionary consequences of the spatial structuring in host–parasitoid systems (Boerlijst *et al.* 1993). A consensus view suggests that complex temporal dynamics in the metapopulation may arise through inclusion of the spatial dimension (Ruxton 1995), and also that 'transient behaviour' may be common, whereby the system exhibits unpredictable behaviour over many generations before settling into one state. In spite of these elaborations, however, the range of spatial patterns observed has remained the same.

Here, we adopt a coupled map lattice approach to a host–pathogen system, where discrete-time within-patch dynamics represent the interaction between the host and a pathogen with free-living infective stages. The completed map allows not only for the recruitment of new free pathogen particles produced as a result of infections in the immediately preceding generation but also for long-term survival of free pathogen produced in previous generations (a significant feature of free pathogen particles). Within and between generation decay of pathogen is also included. The map has a limiting case where pathogen survival from previous generations is zero, in which it is analogous to the Nicholson–Bailey equations. This allows a direct comparison with the study of Comins *et al.* (1992). Previously unreported features are observed, particularly: (i) the appearance of a new spatial pattern; (ii) shifting metapopulation behaviour as the system can exhibit intermittency between two quasi-stable patterns; and (iii) an especially clear link between spatial patterns observed on the arena and the temporal behaviour exhibited by the metapopulation.

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2. A DISCRETE INFECTION MODEL

Initially we neglect spatial effects and concentrate on the within-patch dynamics. All patches are assumed equal and we consider host–pathogen interactions where the host has discrete generations and the pathogen has free-living infective stages. Several other simplifying assumptions are necessary and are discussed during model development.

Within the generation, we assume hosts can only die as a result of infection, a reasonable simplification (see also Anderson & May 1981) provided the natural mortality of hosts is considerably less significant than disease induced mortality, and that hosts can only contract the infection for a period of T_L time units at the start of the generation (Briggs & Godfray 1996). The death of the host and ultimate release of the pathogen particles occurs T_I time units after contracting the infection, where $T_I > T_L$; i.e. the newly released free-particles cannot cause infection until the following generation, implying secondary infections cannot occur (Briggs & Godfray 1996), and a generation is sufficiently long to allow all infected individuals to die and release the pathogen. In addition, the consumption of pathogen by the host is assumed to be a negligible component in the overall rate of pathogen loss (Anderson & May 1981; Dwyer 1992, 1994).

The model is:

$$X_{t+1} = R\,X_t\,e^{-\nu W_t}, \tag{1}$$

$$W_{t+1} = \sigma_1\,\sigma_2\,W_t + \sigma_2\,\Lambda\,X_t(1-e^{-\nu W_t}), \tag{2}$$

where X_t and W_t are respectively the initial numbers of uninfected host individuals and pathogen particles in generation t, R is the average number of viable offspring produced between each generation per host individual, ν is the scaled transmission efficiency of the infection (scaled because the pathogen decays throughout the period when the host is susceptible), σ_1 represents the proportional survival of pathogen from previous generations during the first T_I time units of the present generation (i.e. until newly created pathogen is released), σ_2 represents the proportional survival of all pathogen from T_I time units until the start of the next generation and Λ represents the number of free pathogen particles released per infected individual.

This model is derived by integrating a set of ordinary differential equations over a generation, under the assumptions stated above (see Appendix 1), but can also be described phenomenologically as follows.

In equation (1) the $\exp(-\nu W_t)$ term represents the proportion of host individuals who escaped infection in generation t. Thus, the whole equation multiplies the number of host individuals surviving a generation by the average number of viable offspring per host. Equation (2) consists of two parts. The second represents the amount of pathogen newly created in the immediately preceding generation, as $X_t(1-\exp(-\nu W_t))$ individuals become infected per generation, with each one releasing Λ pathogen particles of which a proportion σ_2 survive until the start of the present generation. The first represents the amount of pathogen created in previous generations that has survived until the start of the present generation.

One advantage of this model is that although it describes a host–pathogen system, the Nicholson–Bailey equations are contained as a limit (achieved by setting $\sigma_1 = 0$, effectively allowing no long-term pathogen survival from previous generations) allowing direct comparisons to be made with the study of Comins *et al.* (1992). Although the model contains a number of assumptions its formulation can still be described biologically and it retains important features, such as long-term pathogen survival, which are fundamental to host–pathogen systems. A stability analysis of the system, equations (1) and (2), indicates that there is one equilibrium which is unstable with diverging oscillations. Hence, as for the Nicholson–Bailey model, extinction is the inevitable outcome in a single patch or in a homogeneous (not patchy) habitat.

3. THE SPATIALLY EXTENDED MODEL

The environment is modelled as a two-dimensional arena, of width n, in which host and pathogen are distributed amongst a square grid of 'cells' or patches. Within each patch the dynamics are represented by equations (1) and (2) and between generations a fraction μ_X of hosts and μ_W of the pathogen disperse outwards to colonize equally the patches which are their eight nearest neighbours. We employ reflective boundary conditions so at the arena's edge individuals reflect or 'rebound' back into the appropriate cell. Thus, we follow the procedure of Comins *et al.* (1992) who give a detailed account of a coupled map lattice, of dispersal to neighbouring patches and reflective boundary conditions.

4. RESULTS

To lay bare the effects of pathogen survival, we increase σ_1 from 0 (equivalent to the Comins *et al.* (1992) study) to 1, as we increase μ_X over its range, while keeping all other parameters fixed, and observe whether the dynamics are altered. An identical study, exchanging σ_2 for σ_1 was also undertaken and the results were very similar. The only significant difference arose trivially, in the region as $\sigma_2 \to 0$. Here total pathogen survival is negligible and unable to restrain unbounded host growth. Due to close agreement for all other corresponding values of σ_1 and σ_2, we have chosen to report only the results for σ_1, thereby stressing the importance of the ratio between the survival of long-lived pathogen particles and that of newly released pathogen particles. In addition, by choosing σ_1 as the parameter to vary, the model contains the Nicholson–Bailey equations as a limit.

To determine the combined effects of long-term pathogen survival (σ_1) and spatial dispersal (μ_X) on the population dynamics of the system we use the parameter values most commonly employed by Comins *et al.* (1992) (and for which most of their results are published; for values see figure 1). Here, μ_X is chosen as a variable parameter since for the fixed values of the

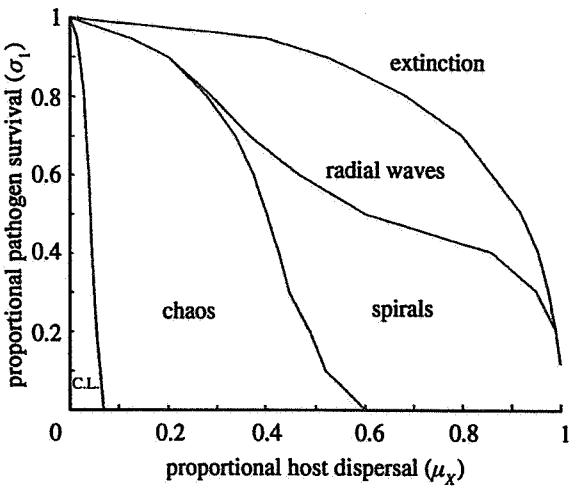

Figure 1. A portrait displaying the likely spatial patterns in μ_X–σ_1 parameter space. C.L. represents the region where crystalline structure are expected. The parameter values used allow comparisons with the study of Comins *et al.* (1992) and are $n = 30$, $r = 2$, $\sigma_2 = 1$, $\Lambda = 1$, $\nu = 1$ and $\nu = 0.89$. The boundaries were obtained by simulation.

other parameters, and $\sigma_1 = 0$, all the possible dynamic patterns can be attained.

Figure 1 shows clearly the influence of long-term pathogen survival on the spatial pattern. At $\sigma_1 = 0$ we obtain the results of Comins *et al.* (1992). As we increase σ_1, pathogen survival increases the possibility of extinction of both host and pathogen. In addition, a new spatial pattern, that of a radial wave, which has previously not been reported, is revealed. This is a wave with a circular wavefront, propagating at a fixed rate from a focus. Such waves occur for virtually all values of host mobility but require intermediate to high values of pathogen survival. Furthermore, their appearance is observed in arenas' with boundary conditions other than reflective.

The radial wave pattern becomes more distinct as μ_X increases. As figure 2*a* shows, for low μ_X the patterns resemble a thin wavefront of relatively low host density propagating across the arena, whereas for higher μ_X (figure 2*b*) the pattern involves higher host densities and contains more than one peak, reminiscent of circular ripples on water. In all cases the waves occur in a repeating pattern, although the details of the period, the intensity and precise shape of the wave vary slightly over time for any fixed set of parameter values.

The increased likelihood of extinction shown in figure 1 occurs when pathogen levels remain high for a sustained period, making host regeneration impossible. At low μ_X this only occurs when σ_1 is high (i.e. very slow decay of pathogen levels). However, as host mobility increases, extinction can occur for much lower values of σ_1. For higher μ_X extinction usually occurs as a direct result of an unusually large host outbreak (figure 3*a*), resulting in the production of such large quantities of pathogen that the host cannot recover and becomes extinct. Such extreme host outbreaks arise as metapopulation levels fluctuate with greater amplitude, which they do as host mobility increases. Higher host mobility is likely to increase the hosts' efficiency in exploiting regions of the arena where pathogen levels are low enough to permit increases in host abundance. This could explain the unusually high host outbreaks observed for higher μ_X and the associated increase in the likelihood of extinction.

Particularly notable is figure 3*b*, *c* which illustrates how metapopulation behaviour and spatial patterns can differ depending on initial conditions, even when parameter values are identical. The values of μ_X and σ_1 in figure 3*b*, *c* are very close to the boundary between spirals and radial waves in figure 1 and here both patterns are possible (on either side of the boundary line the respective pattern is more likely, but not the only possibility). The present boundary is unlike others in figure 1 (and those in Comins *et al.* 1992) as the patterns either side of it (spiral or radial wave) are distinct. At all other boundaries the patterns merge, so that close to the boundary it is difficult to distinguish one pattern from another and characteristics associated with both patterns are often visible simultaneously.

In the case of a spiral pattern, once the spiral has formed (after approximately 400 generations) the

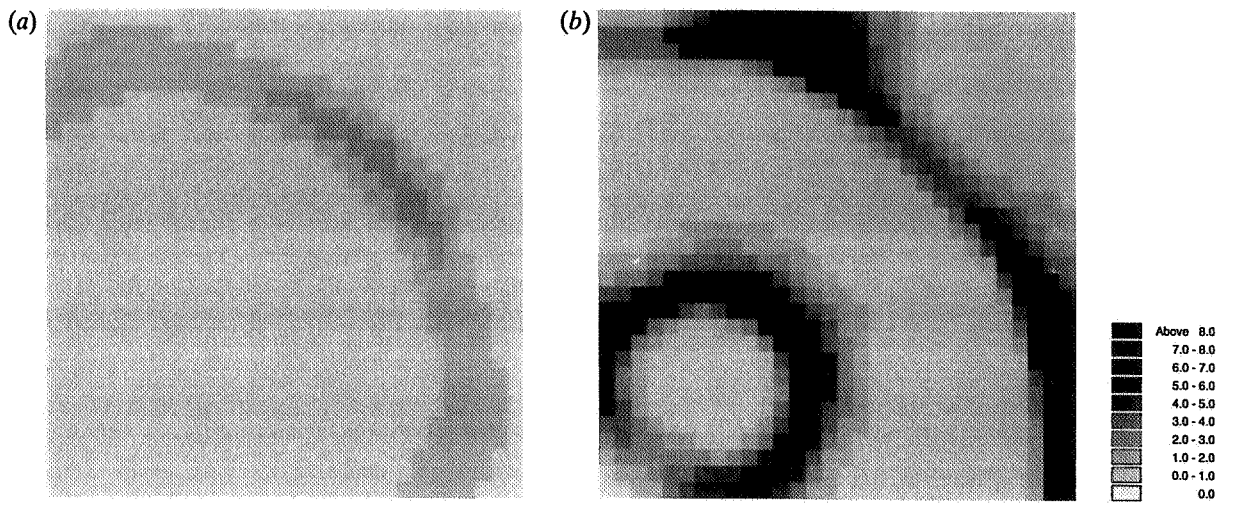

Figure 2. Instantaneous map of host population density displaying the radial wave patterns: (*a*) $\mu_X = 0.2$, $\sigma_1 = 0.92$ and (*b*) $\mu_X = 0.8$, $\sigma_1 = 0.5$ with other parameters as in figure 1.

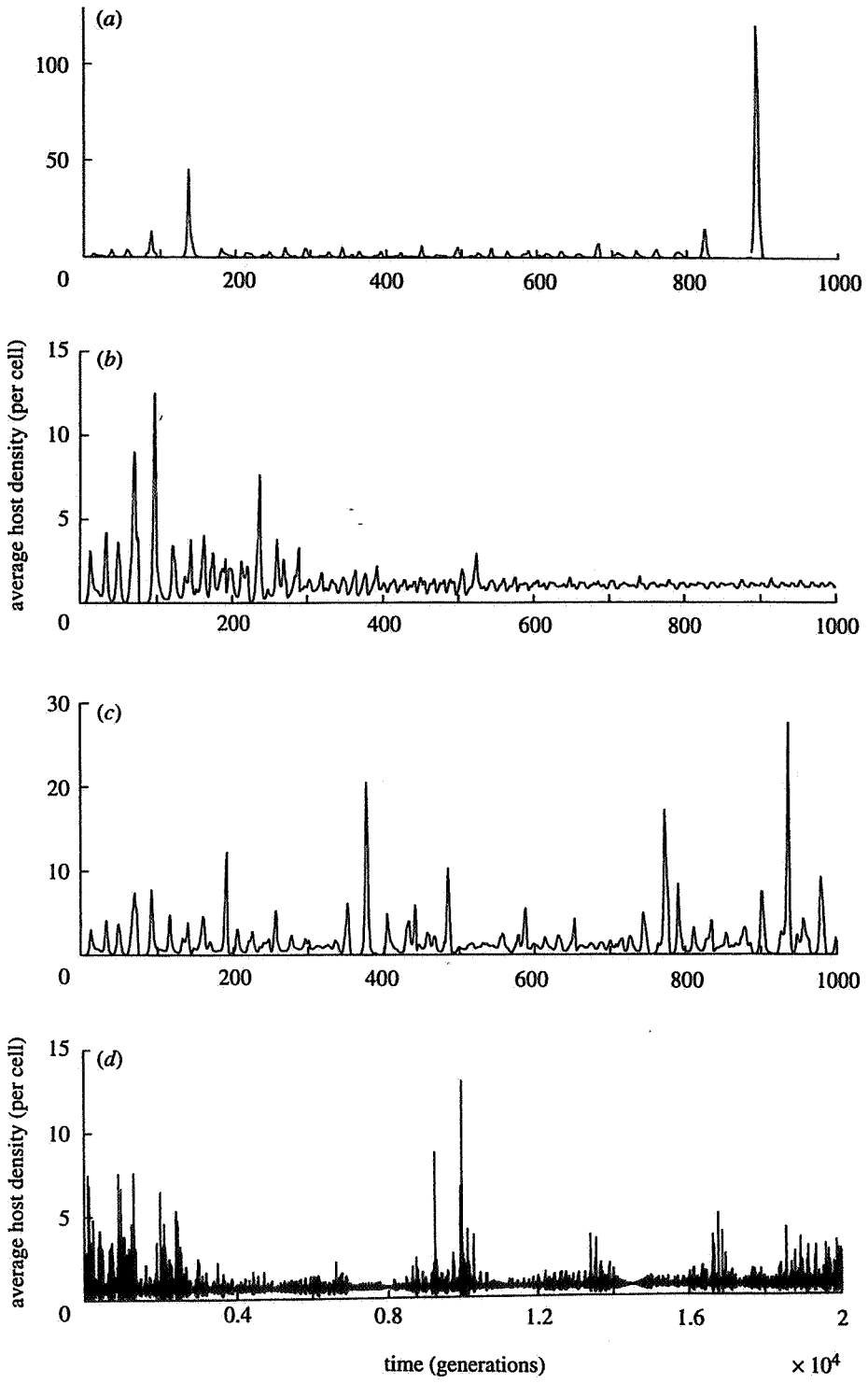

Figure 3. Average host density per cell (equivalent to metapopulation density) plotted against time. Parameters are as in figure 1 except: (*a*) $\mu_X = 0.8$, $\sigma_1 = 0.6$ and the figure portrays a large host outbreak followed by host extinction; (*b*,*c*) $\mu_X = 0.8$, $\sigma_1 = 0.4$ and the figures display how the metapopulation behaviour differs depending on the spatial pattern even though parameter values are identical (in (*b*) the spatial pattern is a spiral and in (*c*) the spatial pattern is a radial wave). In (*d*) $\mu_X = 0.7$, $\sigma_1 = 0.6$ and the portrait depicts the long-term transients in the model in terms of metapopulation levels. Shifts in metapopulation behaviour from wide oscillations to narrow indicate a change from a radial wave spatial pattern to that of a spiral and vice versa.

metapopulation fluctuates within a narrow range (figure 3*b*). However, when a radial wave pattern is observed, the metapopulation fluctuates with far greater intensity and unpredictability (figure 3*c*). When the σ_1–μ_X parameter combination is well within the spiral or radial wave parameter space regions of figure 1, the respective metapopulation behaviour described above for each pattern occurs. Nearer the boundary on the other hand, both types of spatial behaviour are possible, depending on initial conditions, with each associated with its appropriate metapopulation behaviour.

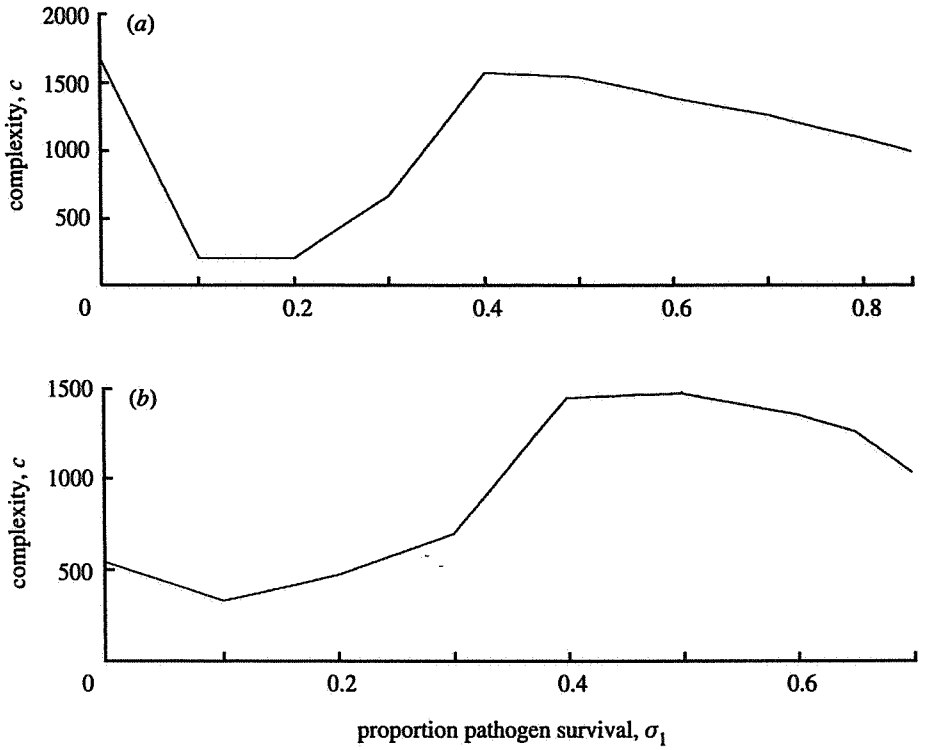

Figure 4. The Kolmogorov complexity measure (c) plotted against proportional pathogen survival. Parameters are as in figure 1 except (a) $\mu_X = 0.55$ and (b) $\mu_X = 0.7$. In (a) complexity decreases as we leave chaos (σ_1: 0–0.1) and both (a) and (b) display how complexity increases as we cross the boundary between spirals and radial waves, which coincides with the region in which intermittency occurs, then decreases as we move away from this boundary. The measure is estimated using a time series of length 45000 generations beginning from generation 20000.

Furthermore, in the long term, both patterns can occur from the same set of initial conditions. Figure 3*d* shows how for one set of values, radial wave behaviour is observed until approximately 3000 generations (i.e. until the metapopulation fluctuations become smaller) after which the behaviour evolves into a spiral pattern, and spirals are clearly observed by 4000 generations, accompanied by small metapopulation fluctuations. These spirals occur until approximately 9000 generations, when radial waves are once again observed on the lattice associated with a marked increase in metapopulation fluctuations. When viewed over several hundred generations, the dynamics may look consistent. However, on a longer time scale the spatial patterns may switch, indicating that the system can exhibit intermittency between two quasi-stable spatial patterns. Such intermittency is impossible in the simpler pure Nicholson–Bailey system (Comins *et al.* 1992). Therefore, as the result is new, it was checked – successfully – that intermittency also occurs for larger arena sizes. The observation that pattern switches alter the nature of the temporal metapopulation dynamics provides evidence of a clear link between this and the spatial pattern.

To extract more information about the nature of the dynamical system and the intermittent behaviour in particular, mathematical techniques involving Lyapunov exponents, complexity measures and spectral analysis may be considered. The calculation of Lyapunov exponents may be inappropriate, as they provide evidence of trajectory divergence in chaotic systems but are unlikely to be helpful in the categorizing of spatial structures, and also difficult, since model systems such as ours typically have high dimensionality.

A more appropriate method may be to evaluate the 'Kolmogorov complexity' of the dynamics (Kaspar & Schuster 1987), a measure which provides useful information in quantifying pattern structure in coupled map lattice systems (Rohani & Miramontes 1995). The Kolmogorov complexity of a given process is the minimum amount of information effort required to simulate it. Kaspar & Schuster (1987) show that by converting metapopulation time series into a string of ones and zeros (by partitioning the data around the average) a quantity c (an appropriate measure of the Kolmogorov complexity) can be calculated as the number of steps required to reconstruct a given binary string by simply 'copying' and 'inserting' digits (see Rohani & Miramontes (1995) for further discussion and Kaspar and Schuster (1987) who provide an algorithm for calculating c). It has been argued that c is a better measure of order than the Lyapunov exponent and examples (Kaspar & Schuster 1987; Rohani & Miramontes 1995) show that it is indeed a useful measure by which the development of spatio-temporal patterns can be characterized. Higher complexity is observed for disordered, chaotic patterns than for ordered, spiral patterns.

In our study, plotting complexity, c, against proportional pathogen survival, σ_1 (figure 4*a*, *b*), shows that the complexity of the metapopulation is correlated

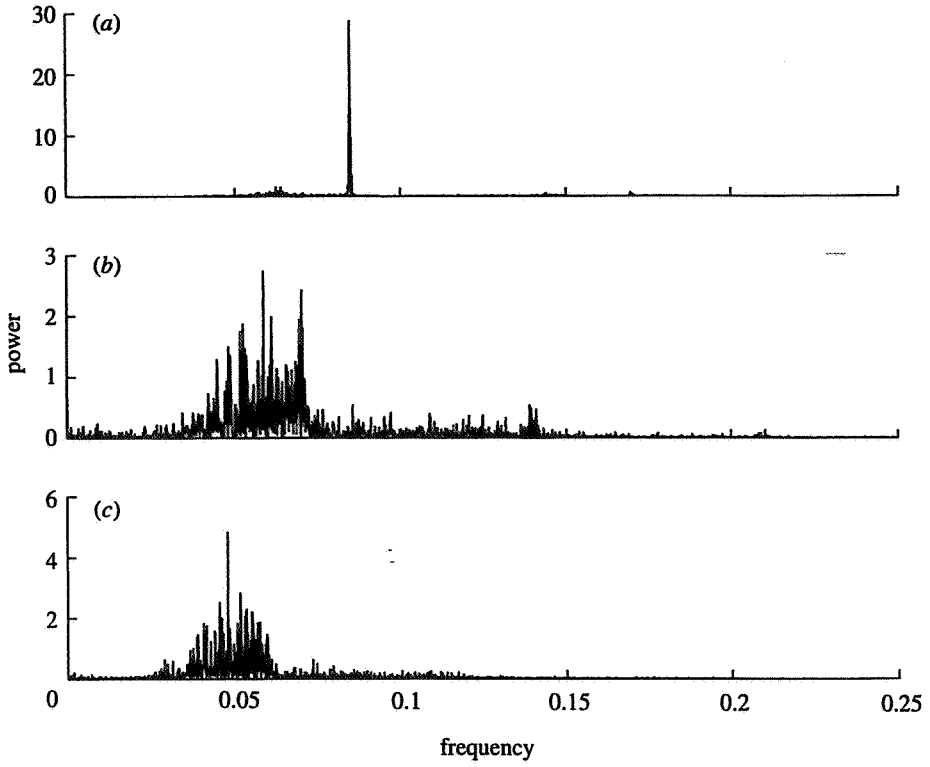

Figure 5. The power spectra of metapopulation density. The parameters are as in figure 1 with $\mu_X = 0.55$ and (*a*) $\sigma_1 = 0.2$ (spirals), (*b*) $\sigma_1 = 0.5$ (intermittency) and (*c*) $\sigma_1 = 0.7$ (radial waves). The portraits display a pronounced periodic component for regions where spirals and radial waves are expected, (*a*) and (*c*) respectively. (*b*) Near the boundary between these regions, where intermittency occurs the spectra contains more noise, a regular periodic component is absent from the dynamics.

with the spatial pattern transitions described by figure 1. Complexity decreases dramatically as we cross the boundary from chaotic patterns into the region of spiral patterns (figure 4*a*). Significantly, however, the complexity can also be observed to peak over the region which corresponds to crossing the boundary between spiral and radial waves. This peak also coincides with the region where intermittency between the two spatial patterns is exhibited, and it is this propensity to switch behaviour at irregular intervals that causes the increase in the complexity measure.

Additional information emerges from a spectral analysis of the temporal dynamics. Power spectra were calculated for metapopulation series corresponding to spirals, intermittency and radial waves (figure 5*a–c*). A single dominant frequency, characterized by high power, is clearly visible for spiral patterns (figure 5*a*). Also, a single dominant frequency is observed for radial waves, but it is of less power than that for spirals, and other frequencies, with powers which decay rapidly as we move in either direction from the dominant frequency, are also visible (figure 5*c*). Such dominant frequencies indicate that the metapopulations associated with these patterns (particularly spirals) exhibit marked temporal periodicity. No such dominant frequency is observed for parameter values in the region of intermittency (figure 5*b*), and here the distribution is flatter (more noisy) and of lower power. This indicates that there is no regular period to the metapopulation dynamics.

Thus, the dynamic spatial structures of spirals and radial waves can be characterized by low complexity measures, implying a relatively higher degree of temporal order and by a marked degree of periodicity. The region of intermittency between spiral and radial waves, on the other hand, is characterized by high complexity and the lack of particular dominant frequencies in the power spectra. The latter observations (in heightened form) are also a characteristic of chaotic (unstructured) patterns, highlighting the problem of categorizing the state of a system without information on both spatial and temporal dynamics.

5. DISCUSSION

By analysing a spatial host–pathogen model which allows direct comparisons to be drawn with the study of Comins *et al.* (1992), we have shown how long-term pathogen survival from previous generations can alter the overall dynamics expected, particularly by allowing the possibility of radial wave patterns and increasing the likelihood of host extinction. Another general result, for all values of host mobility, is that increasing σ_1 has the effect of reducing the metapopulation levels and increasing the period between metapopulation fluctuations. This is intuitively obvious, since greater long-term pathogen survival will result in slower pathogen decay. Hence the host is restrained at low levels for a greater length of time before another outburst can occur.

The repeated radial waves observed in our study vary a little in period, intensity and shape over time for

any fixed set of parameter values. These small and apparently random fluctuations in the details of the repeated waves mean they differ from the repeated waves associated with reaction–diffusion models (Murray 1986; Dwyer 1994). There, the waves arise only when the temporal dynamics alone would exhibit cyclic behaviour, and all wave details are identical. The temporal dynamics in our model – equations (1) and (2) – can only exhibit unstable oscillatory behaviour, implying that the radial waves are not simply a result of the underlying temporal dynamics. However, the occurrence of repeated waves on both the coupled map lattice and in host–pathogen systems represented by reaction–diffusion equations (Dwyer 1994) can be attributed to comparable processes. During an outbreak, propagating hosts produce an abundance of pathogen which is deposited behind the wave. Hosts cannot increase in abundance while pathogen levels are high so the landscape behind the wave contains only a low density of hosts. When the pathogen levels have decayed sufficiently (this depends on σ_1 in our model) another host outbreak can occur which creates a focus from which the host can once again propagate.

Another noticeable feature of figure 1 is that for low values of μ_X (i.e. hosts with limited mobility) increasing the proportion of pathogen survival has little effect on the type of pattern observed, whereas at intermediate and high values of host mobility σ_1 plays a significant role in determining the spatial dynamics. In particular this implies that σ_1 has less effect on the crystal lattice and spatial chaos patterns. Hanski (1994) argues that of the range of dynamical behaviours possible, 'spatial chaos' would be most robust against changes in model assumptions. Neglecting the crystal lattice pattern (which only occurs in a small region of parameter space) our study provides some evidence to substantiate Hanski's statement.

Perhaps the most interesting results in this study concern the ability of the host–pathogen model to display switches in behaviour at both the spatial and the metapopulation level. Previous studies (Hastings & Higgins 1994) have demonstrated transient behaviour, whereby rich patterns of population density variation are generated in space, which shift unpredictably and take thousands of generations to settle into a steady pattern. Hendry & McGlade (1995) remark how transient states may persist for sufficiently long to become the only relevant behaviour in the system. This calls into question the traditional approach to biological modelling, which focuses on equilibria and long-term dynamics; it also generates dilemmas as to whether model-fitting and trend analyses are reliable: a problem discussed by Kareiva & Wennergren (1995) and a cause for concern for ecological policy makers relying on long-term data (Holsinger 1995).

The intermittent behaviour observed in the present study furthers the idea of 'transient' behaviour in a number of important respects. First, the system may never settle to one particular pattern. With appropriate parameter values it may switch repeatedly between patterns, so that all behaviour of the system may be thought of as 'transient'. Moreover, an observer monitoring the system in the relatively short term might observe only one consistent pattern, or only the second, or a switch from one to the other: in spite of a consistency in the underlying biological characteristics. This raises the possibility of similar difficulties in real ecological systems, further undermining faith in the reliability of model-fitting exercises applied to population data. Here, to obtain such behaviour, it is necessary to assume only that identical patches containing host–pathogen dynamics are linked by dispersal.

Furthermore, it is notable that when intermittent behaviour occurs, and more generally, there is a clear connection between spatial and metapopulation dynamics. This emphasizes that explicitly spatial models may further our understanding of the details not only of spatial but also of temporal dynamics.

To conclude, by modelling a host–pathogen system spatially, previously unreported behaviour is obtained and we are driven to extend our view as of what spatially explicit models are capable of achieving. This study reveals the possibility of new spatial patterns, it provides strong evidence of a connection between spatial patterns and metapopulation behaviour and introduces the concept of intermittency, where the metapopulation may incur long-term shifts in behaviour indefinitely.

APPENDIX 1

In this appendix, we develop a discrete form map to represent a host–pathogen system. We recall that hosts can only be infected for a period T_L at the start of a generation, of length T, and will release Λ pathogen particles T_I time units after contracting the infection (where $T_I > T_L$ and $T \geqslant T_I + T_L$).

We begin by defining the rate of infection at time s in generation t as

$$\frac{\nu' X_t(s) W_t(s)}{T_L}, \tag{A 1}$$

where $X_t(s)$ is the number of susceptible individuals, $W_t(s)$ is the number of pathogen particles, ν' is the transmission efficiency of the pathogen. Thus

$$\frac{\mathrm{d}X_t(s)}{\mathrm{d}s} = \begin{cases} \dfrac{-\nu' X_t(s) W_t(s)}{T_L} & \text{for} \quad 0 \leqslant s \leqslant T_L, \\ 0 & \text{for} \quad T_L \leqslant s \leqslant T. \end{cases} \tag{A 2}$$

In addition, as $W_t(s) = W_t(0) \exp(-\mu s)$ for $0 \leqslant s \leqslant T_L$, where μ is the decay rate of the pathogen we can substitute for $W_t(s)$ in (A2) and integrate over the generation. By including between generation reproduction of R viable offspring per host we obtain equation (1):

$$X_{t+1} = R X_t \mathrm{e}^{-\nu W_t}, \tag{A 3}$$

where $\nu = \nu'(1 - \exp(-\mu T_L))/\mu T_L$ is the scaled transmission rate of the infection, with $X_t = X_t(0)$ and $W_t = W_t(0)$.

The equation for the rate of change of the pathogen particles is:

$$\frac{dW_t(s)}{ds} = \begin{cases} -\mu W_t(s) & \text{for } 0 \leqslant s \leqslant T_I, \\ \Lambda \dfrac{v' X_t(s-T_I) W_t(s-T_I)}{T_L} - \mu W_t(s) & \\ & \text{for } T_I \leqslant s \leqslant T_L + T_I, \\ -\mu W_t(s) & \text{for } T_L + T_I \leqslant s \leqslant T, \end{cases}$$

where Λ is the number of free particles released per infected individual. In general, the integration of equation (A4) in closed form is not possible. However, by employing a first-order approximation to $\exp(-\mu t)$, integration over the generation can be achieved and by including proportional pathogen loss, p, between generations we obtain equation (2):

$$W_{t+1} = \sigma_1 \sigma_2 W_t + \sigma_2 \Lambda X_t (1 - e^{-\nu W_t}), \qquad (A\,5)$$

where $\sigma_1 = \exp(-\mu T_I)$ and $\sigma_2 = p \exp(-\mu T_L)$.

REFERENCES

Anderson, R. M. & May, R. M. 1981 The population dynamics of microparasites and their invertebrate hosts. *Phil. Trans. R. Soc. Lond.* B **291**, 451–524.

Bascompte, J. & Sole, R. V. 1994 Spatially induced bifurcations in single-species population dynamics. *J. Anim. Ecol.* **63**, 256–264.

Boerlijst, M. C., Lamers, M. E. & Hogeweg, P. 1993 Evolutionary consequences of spiral waves in a host–parasitoid system. *Proc. R. Soc. Lond.* B **253**, 15–18.

Briggs, C. J. & Godfray, H. C. L. 1996 The dynamics of insect–pathogen interactions in seasonal environments. *Theor. Popul. Biol.* (In the press.)

Comins, H. N., Hassell, M. P. & May, R. M. 1992 The spatial dynamics of host–parasitoid systems. *J. Anim. Ecol.* **61**, 735–748.

Dwyer, G. 1992 On the spatial spread of insect viruses: theory and experiment. *Ecology* **73**, 479–494.

Dwyer, G. 1994 Density dependence and spatial structure in the dynamics of insect pathogens. *Am. Nat.* **143**, 533–562.

Hanski, I. 1994 Spatial scale, patchiness and population dynamics on land. *Phil. Trans. R. Lond.* B **343**, 19–25.

Hassell, M. P., Comins, H. N. & May, R. M. 1991 Spatial structure and chaos in insect population dynamics. *Nature, Lond.* **353**, 255–258.

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

Hendry, R. J. & McGlade, J. M. 1995 The role of memory in ecological systems. *Proc. R. Soc. Lond.* B **259**, 153–159.

Holsinger, K. E. 1995 Population biology for policy makers. *BioScience Sup.* S10–S20.

Kareiva, P. & Wennergren, U. 1995 Connecting landscape patterns to ecosystems and population processes. *Nature, Lond.* **373**, 299–302.

Kaspar, F. & Schuster, H. G. 1987 Easily calculable measure for the complexity of spatiotemporal patterns. *Phys. Rev.* A **36**, 842–848.

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

Pascual, M. 1993 Diffusion-induced chaos in a spatial predator–prey system. *Proc. R. Soc. Lond.* B **251**, 1–7.

Rohani, P. & Miramontes, O. 1995 Host–parasitoid metapopulations: the consequences of parasitoid aggregation on spatial dynamics and searching efficiency. *Proc. R. Soc. Lond.* B **260**, 335–342.

Ruxton, G. D. 1995 Temporal scales and the occurrence of chaos in coupled populations. *Trends Ecol. Evol.* **10**, 141–142.

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