

REPORT

Extinction times for closed epidemics: the effects of host spatial structure

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Abstract

Although of practical importance, the relationship between the duration of an epidemic and host spatial structure is poorly understood. Here we use a stochastic metapopulation model for the transmission of infection in a spatially structured host population. There are three qualitatively different regimes for the extinction time, which depend on patch population size, the within-patch basic reproductive number and the strength of coupling between patches. In the first regime, the extinction time for the metapopulation (i.e. from all patches) is approximately equal to the extinction time for a single patch. In the second regime, the metapopulation extinction time is maximal but also highly variable. In the third regime, the extinction time for the metapopulation (T_E) is given by $T_E = a + bn^{1/2}$ where a is the local extinction time (i.e. from last patch), b is the transit time (i.e. the time taken for infection to spread from one patch to another) and n is the total number of patches.

Keywords

Epidemiology, metapopulation, persistence, stochastic model.

Ecology Letters (2002) 5: 747–755

INTRODUCTION

Parasite persistence is a central issue in infectious disease epidemiology. Establishing the factors that influence the persistence of infection has led to significant results, notably the concept of the critical community size (Bartlett 1956), below which there are not enough hosts to sustain a disease. More recently, epidemiologists have analysed how the spatial structure of the host population impacts on epidemic development. By identifying and analysing key epidemiological processes governing parasite persistence, such as coupling between subpopulations, criteria have been developed for human diseases (e.g. measles – Bolker & Grenfell 1995), animal diseases (e.g. phocine distemper virus – Swinton *et al.* 1998; foot and mouth disease – Keeling *et al.* 2001) and plant diseases (Onstad & Kornkven 1992; Park *et al.* 2001).

Given that a parasite will not persist indefinitely in a population, the question of extinction times naturally arises. Much early work focused on investigating the duration of epidemics in homogeneously mixed populations (Ridler-

Rowe 1967; Barbour 1975). Recent work has extended this to consider the effects of the contact structure on extinction time, for example, by structuring the host population into households (Ball *et al.* 1997; Ball & Lyne 2002) or by considering the effects of spatial structure of the host population (Swinton 1998; Swinton *et al.* 1998).

The need to estimate the duration of an epidemic in a spatially structured host population was highlighted by the recent foot and mouth epidemic in Great Britain (Ferguson *et al.* 2001; Keeling *et al.* 2001; Morris *et al.* 2001). However, the highly heterogeneous nature of the GB farm landscape makes it difficult to draw general conclusions about how spatial structure affects the duration of an epidemic. Analytical results for extinction times in a spatial *SEIR* model have been derived for a linear metapopulation structure with nearest neighbour mixing (Swinton 1998). This work was undertaken as part of a study of phocine distemper virus in the North Sea (Swinton *et al.* 1998) for which a linear metapopulation is appropriate because of the behaviour of ‘haul out’ of seals along the coastline. More generally, the effects of spatial structure on extinction times

are not well understood. Specifically, there is a pressing need to investigate extinction times in two-dimensional landscapes and to relate local processes, such as the build up of infection at small spatial scales, to the host population structure.

Here we use a simple epidemic model to analyse the effects of the spatial structure of a host population and the restricted movement of the parasite on the dynamics of parasite extinction in a two-dimensional landscape. We consider a closed system in which the only processes are infection and disease-induced host mortality or, equivalently, recovery from infection; there are no host births nor deaths. The model is, however, generic and can easily be extended to consider vital dynamics. It can also be adapted for heterogeneous landscapes. The model described here applies to host-parasite interactions in which the demographic processes such as host population growth occur on a significantly longer time scale than the epidemic and can therefore be ignored. It allows us to isolate the effects of spatial structure on the epidemic processes of infection and mortality caused by that infection from the vital dynamics of the host.

Moreover, many seasonal systems are effectively closed between reproduction events. Indeed such seasonal systems provide a major motivation for studying extinction times as we can address the question: Will the parasite survive in the population until host reintroduction via a birth cohort for animals or planting for many agricultural crops? After this time new susceptible hosts are likely to facilitate parasite survival. Consequently, survival between these reintroductions forms a minimum requirement for parasite persistence (Gubbins & Gilligan 1997). This is the case for animal populations that have a short annual birth season, such as seals (Swinton 1998). It also applies to many plant-parasite systems in which there are annual hosts (Gubbins & Gilligan 1997).

The model also addresses the effects of the host population structure on the critical population sizes necessary to sustain infection locally and at larger spatial scales. This is done by focusing on the local processes and properties (i.e. infection within a patch, coupling to other patches, host population size in a patch) and integrating these into a realistic, two-dimensional landscape. Earlier work has successfully identified key processes involved in the invasion and persistence of parasites in spatially structured populations (Park *et al.* 2001). Hence we naturally begin by considering local (within-patch) transmission of infection and coupling between patches. This builds on the work of Swinton (1998). Working with a one-dimensional metapopulation Swinton (1998) proposed that the parasite spreads from the initial site of infection, potentially through the whole population, eventually dying out from one last subpopulation. This suggests that there are two components to the parasite extinction time in a spatially structured

metapopulation: (i) the spreading component, which depends on the parasite transit time between patches and the number of patches in the metapopulation; and (ii) the within-patch component, which (for a fixed disease-induced mortality rate) depends on the size of a patch. This hypothesis was examined by Swinton (1998) in the context of a one-dimensional metapopulation with nearest neighbour mixing (an appropriate framework in which to use the *SEIR* model for phocine distemper virus). Here, we extend the concept to a more general framework. Using a simple *SIR* model (applicable to many animal and plant diseases) we show how short- and long-range spread of infection govern the parasite extinction times in a two-dimensional metapopulation.

THE MODEL

The model describes the stochastic dynamics of a closed epidemic in a spatially structured host population. Spatial structure is represented by subdividing the host population into a number of patches (or subpopulations) with interactions taking place between neighbouring patches. The host population within each patch is divided into susceptible (i.e. uninfected) and infected classes where S_j and I_j are the number of hosts in each class in the j th patch, respectively. The index j identifies the position of each patch within the metapopulation. For simplicity we assume that the patches are arranged in a square array and, hence, the index is $j = (x_j, y_j)$ which describes the horizontal (x_j) and vertical (y_j) position of patch j within the square array of subpopulations.

In the model there are two transitions, infection and removal of infected hosts (for example, through disease-induced mortality or recovery from infection), both of which are stochastic processes. The number of transitions of each type that occurs during a small time interval, δt , is drawn from a binomial distribution, $B(m, p)$, where m is the population size and p is the probability that an individual is affected by that particular process. For infection the appropriate population size and probability are S_j and $\lambda_j \delta t$, respectively, and for removal they are I_j and $\mu \delta t$, respectively, where λ_j is the force of infection (defined below) and μ is the removal rate. An approximating normal or Poisson distribution is used wherever possible to reduce computation time (Evans *et al.* 1993).

The force of infection in patch j , λ_j , is the per capita rate of the acquisition of infection and comprises two components reflecting transmission within and between patches, respectively. It is given by

$$\lambda_j = \beta I_j + \varepsilon \beta \sum_{k \in N_{\text{neigh}}(j)} I_k \quad (1)$$

where β is the rate of transmission within a patch, ε represents the strength of coupling between patches

($0 < \varepsilon < 1$) and $N_{\text{neighbourhood}}$ is the neighbourhood of interaction (i.e. those patches which interact with patch j). Here, we assume that mixing only occurs between nearest neighbours and, thus, a patch $k = (x_k, y_k)$ is in the neighbourhood of patch $j = (x_j, y_j)$ if $|x_j - x_k| + |y_j - y_k| = 1$. This formulation for the force of infection, eqn 1, assumes that there is no saturation in the contact rate for an infected host and, consequently, the addition of the between-patch component increases the number of potential contacts. Conversely, if the contact rate was saturated a conservative form for the force of infection would be more appropriate (cf. Swinton 1998; Swinton *et al.* 1998). However, for the range of values of the coupling parameter, ε , considered in this paper, exploratory analyses indicated that any differences between the two formulations were negligible and, hence, the conclusions we draw are applicable to both forms.

The basic reproductive number is a key epidemiological parameter and is defined as the average number of secondary infections caused by an infected individual introduced to a wholly susceptible population at demographic equilibrium (Diekmann *et al.* 1990). For a single, isolated patch in our model, the basic reproductive number is given by

$$R_p = \beta\kappa / \mu, \tag{2}$$

where κ is the patch population size. Heuristically, this expression is the potential number of new infections, $\beta\kappa$, that an infected individual is capable of making in a wholly susceptible population per unit time multiplied by the infectious period, $1/\mu$. In the results that follow, the within-patch basic reproductive number (eqn 2), is shown to be an important parameter with respect to the duration of an epidemic.

EXTINCTION TIMES

If a population is spatially subdivided, either due to discrete host communities or restricted parasite dispersal in a continuous population, then it is natural to consider within- (i.e. local extinction) and between-patch (i.e. transit) components to extinction. To investigate the effects of spatial structure on these two components, we simulated 100 replicate epidemics (each initiated by introducing 10 infected hosts into the population) for a range of values for the patch size, κ , the within-patch basic reproductive number, R_p , and the strength of coupling between patches, ε .

Local extinction

Homogeneously mixed populations have been extensively studied in epidemiology (Anderson & May 1991). Early results on extinction times in such populations were derived using applied probability theory and showed that the

average time to extinction depends on the logarithm of the population size (Ridler-Rowe 1967; Barbour 1975). Introducing this idea into a spatial context, Swinton (1998) extended the definition of the critical community size (Bartlett 1956) to a critical distribution of patch population sizes (Swinton *et al.* 1998) and an associated local extinction time that depends on the logarithm of the patch population size (Swinton 1998; Swinton *et al.* 1998).

Calculating the time to extinction for a range of patch population sizes, κ , shows that the extinction time for a single, isolated patch exhibits the expected $\log(\kappa)$ dependence, and that the variability is effectively constant (Fig. 1a). Furthermore, there is a marked dependence of the extinction time on the within-patch basic reproductive

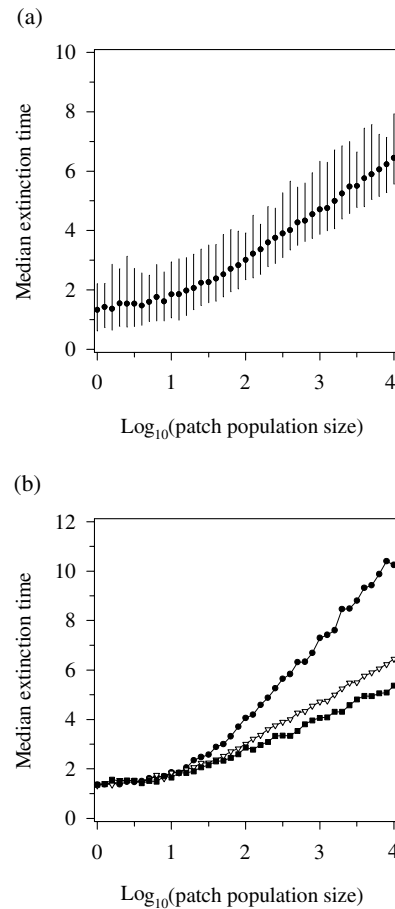


Figure 1 Extinction times and their dependence on population size, κ , for a single patch. (a) Median extinction time (circles) and the 5 and 95 percentiles (error bars). (b) Median extinction time for different values of the within-patch basic reproductive number: $R_p = 2$ (circles); $R_p = 4$ (triangles); and $R_p = 8$ (squares). Results are based on the simulation of 100 replicates of the model (with $\delta t = 10^{-3}$). Default parameters are $\mu = 3.0$, $R_p = 4$ and $\varepsilon = 0.01$, and the initial populations are $S = \kappa$ and $I = 10$.

number, R_p (Fig. 1b). Parasite extinction occurs more quickly for large values of R_p because the rate at which the parasite can colonize susceptible hosts is larger.

Transit times

A key component of the parasite extinction time in a closed, spatially structured population is the time it takes for the parasite to spread from the site of its introduction to the furthest edges of the system (or, for more complex interactions, to the least connected subpopulations). In a metapopulation this is achieved by patch-to-patch transits, associated with which are the probability of a transit and a characteristic transit time. Here we define the transit time to be the time taken for infection to first appear in a patch which is directly connected to an infected patch.

Using a two-patch version of the model, the probability of a transit and the median transit time are calculated. Both depend on the within-patch basic reproductive number, R_p (Fig. 2) and the strength of coupling between the patches, ε (Fig. 3). Note that where percentiles are not shown (Fig. 3b–d), fewer than 20 replicates gave rise to a transit and, hence, percentiles are not properly defined.

Increasing the within-patch basic reproductive number, R_p , increases the probability of a transit (Fig. 2). This increase is most significant at smaller patch population sizes, where the probability of a transit is generally low due to the small number of infected hosts. An increase in R_p also leads to a reduction in the characteristic transit time (Fig. 2). By varying the between-patch strength of coupling, ε , we see that there is a critical patch population size below which the probability of a transit is very low (Fig. 3). Increasing ε has the effect of reducing both the critical patch size for a transit and the transit time (Fig. 3). When varying either the within-patch basic reproductive number, R_p , or the between-patch strength of coupling, ε , the median transit time is robust to changes in the patch population size, and the greatest variability in the transit time occurs at low patch population sizes (i.e. when the probability of a transit is smallest) (Figs 2 and 3).

Extinction times for the metapopulation

The relationship between the duration of an epidemic and the patch population size (Figs 4 and 5) was investigated for a metapopulation arranged in a square array with each epidemic initiated by introducing 10 infected hosts into a central patch. The extinction time for the metapopulation (i.e. the time taken until infection dies out from all patches) has three distinct phases depending on the patch population size (Figs 4 and 5). For small patch population sizes the probability of a transit is small (Figs 2 and 3) and, hence, transits are rare. Consequently there is only a local (i.e.

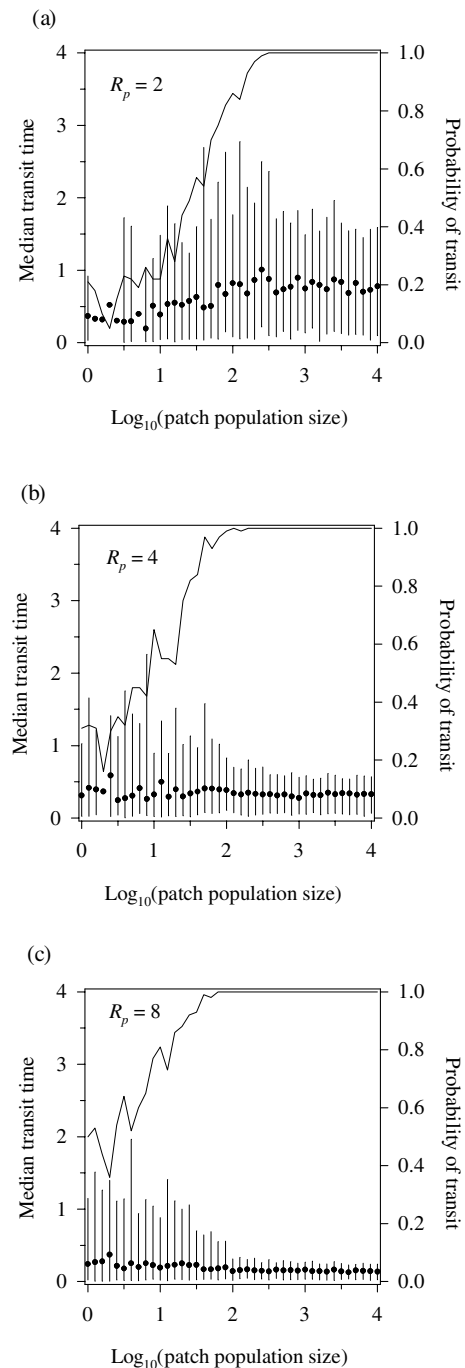


Figure 2 Transit times for infection and their dependence on patch population size, κ , for different values of the within-patch basic reproductive number: (a) $R_p = 2$; (b) $R_p = 4$; and (c) $R_p = 8$. Each figure shows the median transit time (circles) and the 5 and 95 percentiles (error bars) together with the probability of a transit occurring (solid line). Results are based on the simulation of 100 replicates of the model with two patches (with $\delta t = 10^{-3}$). The remaining parameters are $\mu = 3.0$ and $\varepsilon = 0.01$, and the initial populations are $S_1 = S_2 = \kappa$, $I_1 = 10$ and $I_2 = 0$.

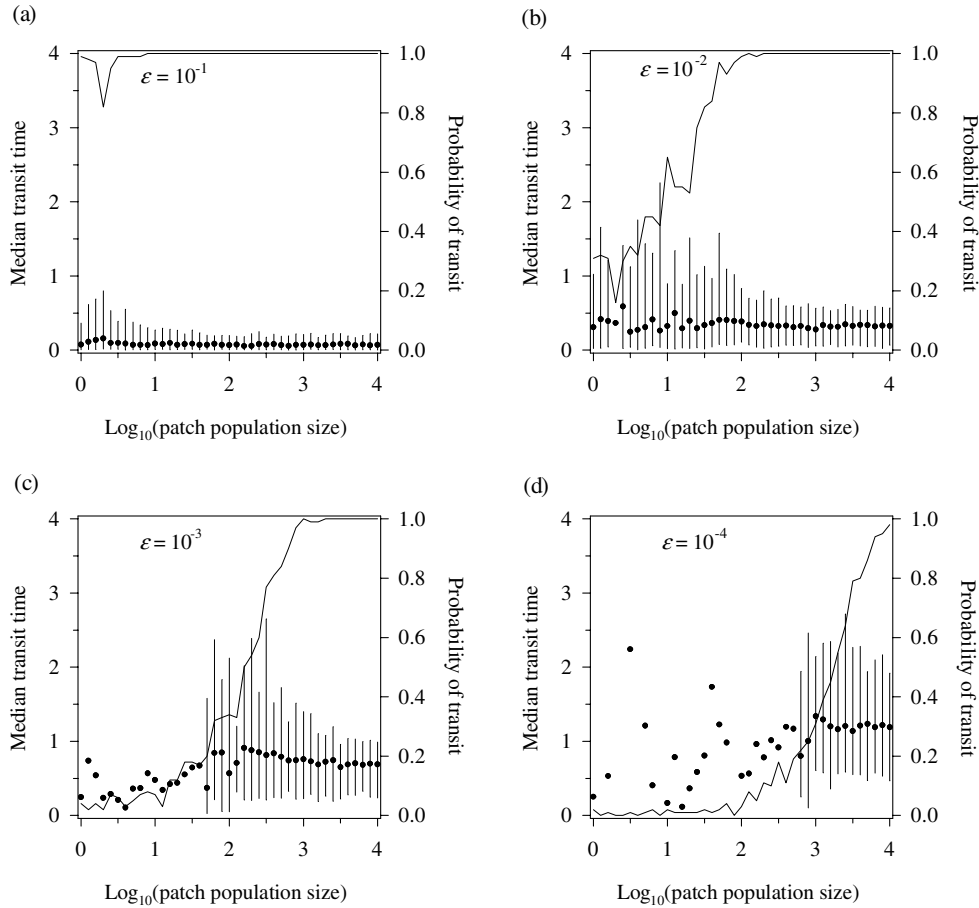


Figure 3 Transit times for infection and their dependence on patch population size, κ , for different values of the strength of coupling between patches: (a) $\varepsilon = 10^{-1}$; (b) $\varepsilon = 10^{-2}$; (c) $\varepsilon = 10^{-3}$; and (d) $\varepsilon = 10^{-4}$. Each figure shows the median transit time (circles) and the 5 and 95 percentiles (error bars) together with the probability of a transit occurring (solid line). Results are based on the simulation of 100 replicates of the model with two patches (with $\delta t = 10^{-3}$). The remaining parameters are $\mu = 3.0$ and $R_p = 4.0$ and the initial populations are $S_1 = S_2 = \kappa$, $I_1 = 10$ and $I_2 = 0$.

within-patch) epidemic and the metapopulation extinction time is approximately equal to the extinction time for a single patch (cf. Fig. 1b). At intermediate patch population sizes, the probability of a transit increases to intermediate values (Figs 2 and 3) and the metapopulation extinction time is larger because the epidemic spreads from the initial site. In this regime the variability in extinction times is greatest (Fig. 4a) and the extinction times are the longest (even longer than at large patch population sizes when the epidemic typically spreads through the whole metapopulation; see below) (Figs 4 and 5). A similar result was observed by Swinton (1998), who speculated that this is due to transits occurring later in the local epidemic when the patch population is at an intermediate level (cf. Figs 2 and 3). Finally, as the patch population size increases further, the probability of a transit is close to one (Figs 2 and 3) and the epidemic spreads rapidly through the whole metapopulation (i.e. there is a global epidemic). The extinction time is

shorter than in the intermediate regime (Figs 4 and 5) largely because transits occur more frequently (Figs 2 and 3).

The metapopulation extinction times show characteristic dependencies on the transmission parameters in the model (Figs 4 and 5). Increasing either the within-patch basic reproductive number, R_p , or the strength of coupling, ε , decreases the critical patch sizes for transition from local to global epidemics (Figs 4 and 5), as predicted by the transit dynamics (Figs 2 and 3). Below the critical patch size, neither parameter has much effect on the extinction time, whilst above it increasing either parameter leads to a reduction in extinction times (Figs 4 and 5).

Partitioning the components of extinction times

When global epidemics occur in the metapopulation (i.e. for sufficiently large patch sizes), it is possible to identify a relationship between the metapopulation extinction time

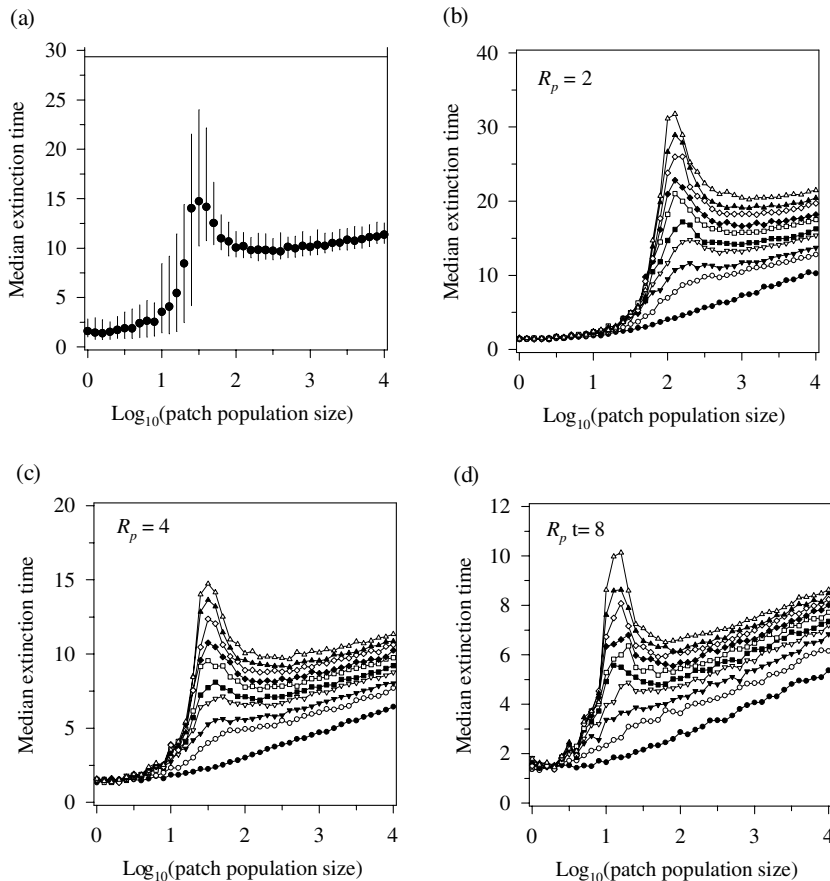


Figure 4 Extinction times and their dependence on patch population size, κ , for different values of the within-patch basic reproductive number. (a) Median extinction time (circles) and the 5 and 95 percentiles (error bars) for $R_p = 4$. (b)–(d) Median extinction times: (b) $R_p = 2$; (c) $R_p = 4$; and (d) $R_p = 8$ for a different number of patches: $n = 1$ (solid circles); $n = 4$ (hollow circles); $n = 9$ (solid downward triangles); $n = 16$ (hollow downward triangles); $n = 25$ (solid squares); $n = 36$ (hollow squares); $n = 49$ (solid diamonds); $n = 64$ (hollow diamonds); $n = 81$ (solid upward triangles); and $n = 100$ (hollow upward triangles). Results are based on the simulation of 100 replicates of the model (with $\delta t = 10^{-3}$). The remaining parameters are $\mu = 3.0$ and $\varepsilon = 0.01$ and the initial populations are $S_j = \kappa$ and $I_j = 0$, except for a central patch in which $I = 10$.

and the combination of local extinction times, the number of patches and the transit time. Swinton (1998) proposed such a relationship for a one-dimensional metapopulation which can be summarized as $T_E = a + bn$, where a is the local extinction time (i.e. extinction from one patch) which depends on the patch population size (cf. the section on local extinction; Fig. 1), b is the transit time (cf. the section on transit times; Figs 2 and 3) and n is the number of transits. Here we extend the model to take account of the two-dimensional structure, thus $T_E = a + bn^{1/2}$. The power of $1/2$ arises because of the two-dimensional structure of the metapopulation.

To test the hypothesis, extinction times were calculated for increasingly large systems (up to a 50×50 patch array) for patch population sizes of $\kappa = 10^3$ and $\kappa = 10^4$ (Fig. 6). The extinction times predicted by the hypothesis show close agreement when superimposed over the calculated extinction times (Fig. 6). The parameter estimates (and their 95% confidence intervals) for $\kappa = 10^3$ are $a = 6.21$ (5.73,6.69) and $b = 0.39$ (0.32,0.48), whilst for $\kappa = 10^4$ they are $a = 7.68$ (7.13,8.23) and $b = 0.37$ (0.28,0.46). Estimates for a (the local extinction time) show the predicted dependence on patch size but are a little larger than

expected, lying in the upper tail (95th percentile) of the distribution (cf. Fig. 1). This reflects the dependence of this component of the extinction time on those local epidemics that are of longer duration. Estimates for the transit times, b , are very close to those derived from transit simulations (cf. Figs 2 and 3). Approximate F -tests (Ross 1990) show that allowing the power to vary (i.e. fitting the relationship $T_E = a + bn^c$ rather than $T_E = a + bn^{1/2}$) does not significantly reduce residual deviance. Moreover, the estimates (and their 95% confidence intervals) for the power, c , are $c = 0.49$ (0.47,0.52) for $\kappa = 10^3$ and $c = 0.48$ (0.45,0.51) for $\kappa = 10^4$, and estimates for the other parameters are comparable.

DISCUSSION

Using a simple, stochastic model we have shown how the spatial structure of a host population affects the duration of epidemics. Starting from populations comprising one or two patches we have identified two key processes governing parasite extinction in a spatial context. These are local (i.e. within-patch) extinction and transits (i.e. the transmission of infection between patches). Local extinction is strongly

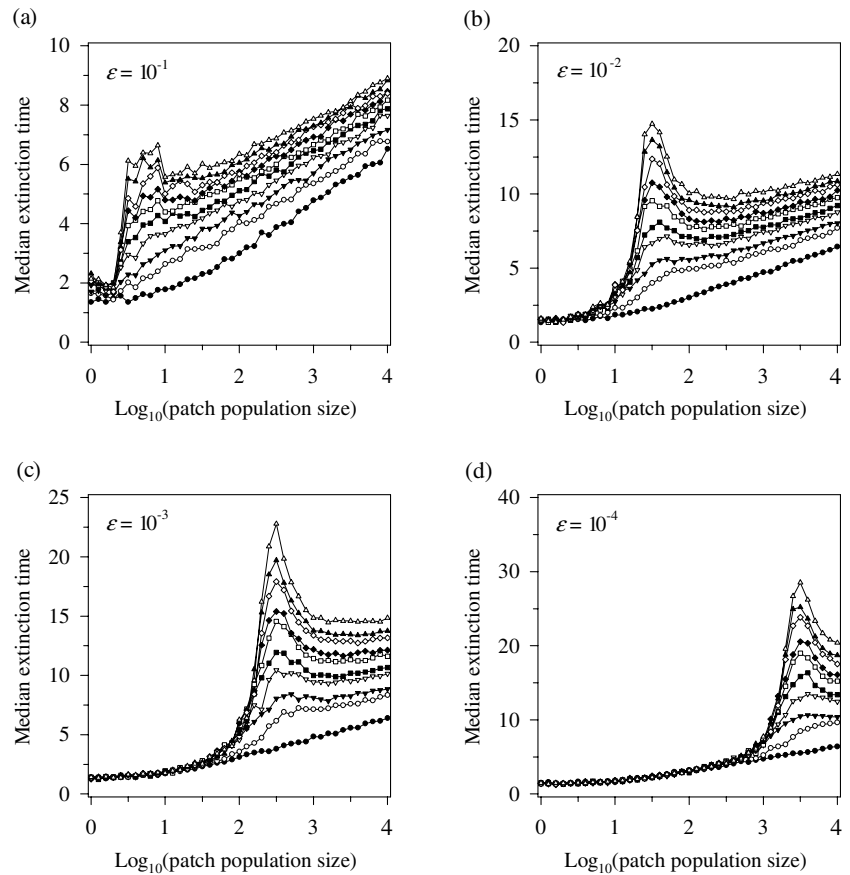


Figure 5 Extinction times and their dependence on patch population size, κ , for different values of the strength of coupling between patches: (a) $\varepsilon = 10^{-1}$; (b) $\varepsilon = 10^{-2}$; (c) $\varepsilon = 10^{-3}$; and (d) $\varepsilon = 10^{-4}$. Each figure shows the median extinction time for a different number of patches: $n = 1$ (solid circles); $n = 4$ (hollow circles); $n = 9$ (solid downward triangles); $n = 16$ (hollow downward triangles); $n = 25$ (solid squares); $n = 36$ (hollow squares); $n = 49$ (solid diamonds); $n = 64$ (hollow diamonds); $n = 81$ (solid upward triangles); and $n = 100$ (hollow upward triangles). Results are based on the simulation of 100 replicates of the model (with $\delta t = 10^{-3}$). The remaining parameters are $\mu = 3.0$ and $R_p = 4$ and the initial populations are $S_j = \kappa$ and $I_j = 0$, except for a central patch in which $I = 10$.

influenced by the population size in a patch (Fig. 1). Patch size may be determined by natural boundaries such as school catchment areas for childhood diseases or fields for plant disease. In practice, however, the size of a patch is determined by the scale at which homogeneous mixing between host and parasite remains a plausible description of the infection process. The scale varies depending on the nature of the host and the dispersal properties of the parasite (Thrall & Burdon 1999; Park *et al.* 2001). Transits play a vital role in determining the duration of an epidemic. Both the probability of a transit and the time taken for a transit to occur combine to influence the extent of spatial spread and also the extinction time. In turn, the transits depend upon the spatial parameters describing the local (R_p) and between-patch (ε) spread of infection (Figs 2 and 3).

Qualitatively, there are three regimes for extinction times that correspond with individual patch size (Figs 4 and 5). In the first regime, which corresponds with small patch sizes, the parasite is unable to spread from its initial site of introduction. Consequently, the extinction time for the whole metapopulation is the same as the extinction time within a single patch. The latter scales with $\log(\kappa)$ where κ is the patch population size (Figs 4 and 5; cf. Fig. 1). In the second regime (corresponding to intermediate patch sizes)

the probability of transmission to another patch increases (Figs 2 and 3). The epidemic typically spreads beyond a single patch but does not reach all patches in the metapopulation. Hence, both the extent of spread and the extinction time are most variable in this regime. The duration of an epidemic tends to be longest in this intermediate regime, because the infection must build up locally before it is able to spread to neighbouring patches. In the third regime (i.e. for large patch sizes) transits are sufficiently likely to occur so that an epidemic spreads through the whole population. For a regular two-dimensional patch structure the number of transits scales with $n^{1/2}$, where n is the total number of patches in the metapopulation. Using this scaling together with the results on local extinction times (Fig. 1) and transits (Figs 2 and 3) we extended the model of Swinton (1998) to predict extinction times for the metapopulation in this regime, $T_E = a + bn^{1/2}$, where a is the local extinction time and b the transit time, an hypothesis supported by simulations (Fig. 6).

This important result allows the prediction of extinction times in spatially extended systems using information based on local processes: the duration of a local epidemic and the probability and time taken for infection to spread between

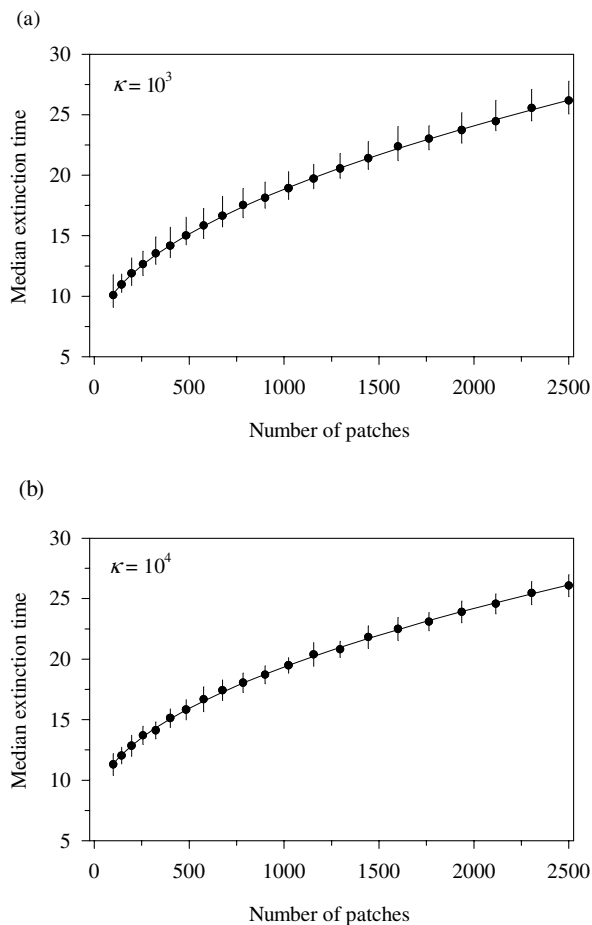


Figure 6 Extinction times and their dependence on the number of patches for different patch population sizes: (a) $\kappa = 10^3$; and (b) $\kappa = 10^4$. Each figure shows the median extinction time (circles) with 5 and 95 percentiles (error bars) together with the fitted relationship $T_E = a + bn^{1/2}$ (solid line). The estimates for a and b in each case are discussed in the section on partitioning the components of extinction times. Results are based on the simulation of 100 replicates of the model (with $\delta t = 10^{-3}$). The default parameters are $\mu = 3.0$, $R_p = 4$ and $\varepsilon = 0.01$ and the initial populations are $S_j = \kappa$ and $I_j = 0$, except for a central patch in which $I = 10$.

patches. Consequently, if data on these components are known, for example, from previous epidemics, it is possible to predict the duration of future epidemics (e.g. phocine distemper virus – Swinton 1998; Swinton *et al.* 1998). Moreover, transmission parameters are often very difficult to estimate empirically, especially the strength of coupling between patches (Kareiva 1990; Bolker & Grenfell 1995). However, epidemic duration and transit times are often more amenable to estimation from data and, hence, if the relationship between these and transmission parameters are known, they can be used to provide estimates for such parameters (see, e.g. Swinton 1998; Swinton *et al.* 1998).

Although the underlying epidemiological model used here is simple, it provides a framework into which more complexity can easily be incorporated. The regular patch structure with homogeneous patch population sizes is an obvious first step to developing theories concerning the relationship between host spatial structure and the duration of epidemics. The model can be adapted to reflect real landscapes or populations in the spirit of Keeling *et al.* (2001) in which a detailed UK farm landscape was used to investigate the spread and duration of foot and mouth disease. Although simple relationships analogous to those developed here and in Swinton (1998) are unlikely to emerge for more complicated cases, our work has shown that stochastic simulations are powerful tools for investigating the factors which influence the dynamics of epidemics in spatially structured populations. Another further step is to integrate more realistic contact structure models such as that proposed by Webb & Sauter-Louis (2002) for transmission of scrapie in sheep, with the metapopulation approach adopted here.

As we have demonstrated here, stochastic models and, in particular, those incorporating host spatial structure, provide a powerful tool with which to investigate persistence and the duration of an epidemic. Moreover, they can be used to assess the impact of management and conservation strategies (Shea *et al.* 2000; Fieburg & Ellner 2001). As spatial structure continues to be a focus for epidemiologists, more information on host structure is coming to light. This promises to enhance our understanding of why epidemics persist and, further, will give us the opportunity to test control strategies.

ACKNOWLEDGEMENTS

We gratefully acknowledge funding from the Natural Environment Research Council (AWP), Churchill College, Cambridge (SG), the Biotechnology and Biological Sciences Research Council, the Royal Society and the Leverhulme Trust (CAG). We also thank Matt Keeling for helpful discussions.

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Editor, J. C. Koella

Manuscript received 25 June 2002

First decision 28 July 2002

Manuscript accepted 11 August 2002