

Rothamsted Repository Download

A - Papers appearing in refereed journals

Van den Bosch, F., Helps, J. and Cunliffe, N. 2024. The basic-reproduction number of infectious diseases in spatially structured host populations. *Oikos*. p. e10616. <https://doi.org/10.1111/oik.10616>

The publisher's version can be accessed at:

- <https://doi.org/10.1111/oik.10616>

The output can be accessed at: <https://repository.rothamsted.ac.uk/item/98zv0/the-basic-reproduction-number-of-infectious-diseases-in-spatially-structured-host-populations>.

© 18 June 2024, Please contact library@rothamsted.ac.uk for copyright queries.

OIKOS

Research article

The basic-reproduction number of infectious diseases in spatially structured host populations

Frank van den Bosch¹, Joe Helps² and Nik J. Cunliffe³✉

¹Department of Plant Pathology, Quantitative Biology and Epidemiology Group, University of California, Davis, CA, USA

²Net Zero and Resilient Farming, Rothamsted Research, Harpenden, Hertfordshire, UK

³Department of Plant Sciences, University of Cambridge, Cambridge, UK

Correspondence: Nik J. Cunliffe (njc1001@cam.ac.uk)

Oikos

2024: e10616

doi: [10.1111/oik.10616](https://doi.org/10.1111/oik.10616)

Subject Editor: Thorsten Wiegand

Editor-in-Chief: Dries Bonte

Accepted 14 May 2024



The spatial structure of a host population has a profound effect on the dynamics of infectious diseases. The basic reproduction number, a central quantity in the study of epidemic dynamics, is affected by host clustering as well as host density. Several authors have developed methods to quantify the basic reproduction number in a spatially structured host population. The methods used and the expressions derived are however difficult to apply to real life spatial host structures. In this paper we introduce an explicit expression for the basic reproduction number using the *O*-ring statistic, developed in spatial statistics, that quantifies the host density as a function of the distance from a randomly selected host individual. The *O*-ring statistic is frequently used in the study of the ecology of spatially structured plant populations, being a convenient summary of the properties of a landscape by way of a single function. The connection we develop between spatial statistics and epidemic dynamics can be used to study the effect of host spatial pattern on the basic reproduction number of infectious diseases. As well as showing how explicit expressions for the basic reproduction number can be derived for landscapes with standard structures, our expression for the basic reproduction number is tested against a simulation model. The model structure in our simulation is motivated by the spread of a plant disease epidemic, although it is applicable more broadly. The agreement between our analytic expression for the basic reproduction number and the corresponding numeric quantity extracted from simulations is close to perfect across a wide range of landscape structures and model parameterisations, and including cases in which more than one species of host is at risk of infection.

Keywords: host clustering, moment closure techniques, Neyman–Scott process, *O*-ring statistic, pathogen dispersal function

Introduction

The effects of host spatial structure on the dynamics of infectious diseases is increasingly recognised. For example, several authors, using epidemiological models, have found that in a spatially clustered host population epidemic incidence increases faster



www.oikosjournal.org

© 2024 The Authors. Oikos published by John Wiley & Sons Ltd on behalf of Nordic Society Oikos. This is an open access article under the terms of the Creative Commons Attribution License, which permits use, distribution and reproduction in any medium, provided the original work is properly cited.

at the start of the epidemic than in host populations with a random spatial distribution (Bolker 1999, Brown and Bolker 2003, Bauch 2005). This finding has been experimentally verified by Burdon and Chilvers (1976) in a controlled environment experiment using the pathogen *Pythium irregulare* infecting garden cress *Lepidium sativum*. Bolker (1999), Brown and Bolker (2003) and Bauch (2005) also found that the critical transmission rate above which an epidemic will spread depends on both the host distribution and the shape of the dispersal distribution of infectious units around a host.

These results relate to one of the key concepts in epidemiology, the existence of a threshold for epidemic development. This threshold, called the basic reproduction number, R_0 , is defined as the average number of secondary cases produced by one infected individual introduced into a population of susceptible individuals. When $R_0 > 1$ the number of infected individuals is expected to increase over time, resulting in an epidemic. When $R_0 < 1$ the disease will not cause an epidemic and the pathogen eventually disappears from the host population.

Expressions for the basic reproduction number, in terms of the underlying host and pathogen population dynamics and epidemiological parameters, have been derived in a wide range of cases. These include vector transmitted diseases, the effect of age structure, host behavioural heterogeneity and genetic heterogeneity (Keeling and Grenfell 2000, Madden et al. 2000). The most generic method for the derivation of basic reproduction number in terms of the pathogen and host parameters was developed by Diekmann et al. (1990) and popularised as the next generation method some years later (Diekmann et al. 2009). However, virtually all models leading to an explicit expression for R_0 make the underlying assumption of mass action, the assumption that any infectious individual in the population has an equal chance of infecting any susceptible individual, thereby lacking spatial heterogeneity.

Epidemiological models, formulated as computer simulation programmes, where the host population has an explicit spatial structure and pathogen dispersal is modelled by a contact distribution (dispersal distribution), have been developed for a wide range of purposes. Bolker (1999) introduced a spatially explicit epidemiological modelling framework that allows a detailed specification of the spatial arrangement of the host. Each host individual is treated as an individual unit located in continuous space (a point pattern). The transmission of the pathogen is governed by an infectious unit's dispersal distribution defined as the probability of an infectious unit produced by a host located at coordinates (x_1, x_2) infecting a susceptible host located at (y_1, y_2) . In most models developed so far, the dispersal distribution is rotationally symmetric. This modelling methodology, either in its deterministic or stochastic form, has been widely adopted by theoretical epidemiologists (Bolker 1999, Tildesley et al. 2006, Meentemeyer et al. 2011, te Beest et al. 2011, Cunliffe 2015, 2016).

Although these models allow for a full specification of the host spatial pattern and realistic pathogen dispersal, the main drawback is that they are far less analytically tractable than models of well mixed host populations. In particular, although

the basic reproduction number can be estimated from simulation outputs, there is as yet no simple, biologically intuitive, analytical expression for the basic reproduction number for spatial host distributions other than random distribution. For spatially random host distributions Suprunenko et al. (2021) and Wadkin et al. (2024) derive expressions for R_0 . They include other aspects such as host depletion (Wadkin et al. 2024) and the effect of disease control (Suprunenko et al. 2021). An expression for R_0 in non-random host distributions would make it easier, compared to simulation runs, to understand the effects of pathogen life-cycle parameters and host spatial structure characteristics on the basic reproduction number. Ideally, such an expression would not need a full specification of the host landscape (as simulations need) but instead would enable the calculation of R_0 on the basis of some simplified landscape characteristics.

In spatial statistics a range of measures have been developed to characterise spatial point patterns (Cressie 1991, Diggle 2003, Baddeley et al. 2015). Ecologists have used these measures to study the ecology of plant populations (Law et al. 2009, Ben-Said 2021). One of the most commonly used measures is Ripley's $K(r)$ function (Ripley 1976, 1977). Ripley's $K(r)$ is defined as the expected density of individuals in a circle with radius r around a randomly chosen plant individual.

$$K(r) = \lambda^{-1} \times E \{ \text{the number of individuals within a distance } r \text{ of a randomly chosen individual} \}, \quad (1)$$

where λ is the plant density (number per unit area). Another well known statistic is the pair correlation function $g(r)$ which is defined as the probability of finding a host at distance r from a randomly chosen host (given that there is a host). The pair correlation function is related to Ripley's K -statistic as

$$g(r) = \frac{1}{2\pi r} \frac{dK(r)}{dr}. \quad (2)$$

For our purpose a more useful statistic, derived from the above statistics, was introduced by Wiegand and Moloney (2004) as the O -ring statistic, $O(r)$. The O -ring statistic is defined as the expected density of individuals at a distance r from a randomly chosen individual and is related to the pair correlation function by $O(r) = \lambda g(r)$. So, $2\pi r O(r) dr$ is the expected number of individuals in a ring of width dr and radius r from a random individual.

The $O(r)$ statistic is a convenient and concise summary of the entire structure of a landscape, and describes how many potential hosts an infectious unit encounters as a function of distance. This leads us to suggest that the dispersal density, $D(r)$, defined as the probability that an infectious unit is deposited at a distance r from the infectious host and the O -ring statistic, $O(r)$, are the key ingredients of the basic reproduction number of infectious diseases in spatially structured host populations.

We derive the expression for the basic reproduction number of an infectious disease in a spatially structured host population and test the expression using a spatially explicit stochastic epidemiological model. Both pathogens with a single host species and pathogens with multiple host species will be discussed.

The expression for R_0

In this section we derive the expression for the net-reproductive number R_0 . To introduce the use of the O -ring statistic in calculating R_0 we first describe the one host species case, before generalising to the multiple host case.

One host species

The basic reproduction number, R_0 , quantifies the growth of a population according to a discrete process mapping from one generation to the next. If the number of infected hosts in generation t is given by I_t , the dynamics of the infected population is given by

$$I_{t+1} = R_0 I_t. \quad (3)$$

We assume one infected host produces on average θ infectious units (such as spores or vectors carrying the pathogen) per time unit, the probability per time unit for the infected host to be removed (e.g. die or reach the end of the infectious period) is μ . The mean total number of infectious units produced by a host during the infectious period is thus θ/μ . The dispersal density, $D(r)$, is the probability density that an infectious unit is deposited at distance r from the host. At distance r from the host, in an otherwise uninfected population, the density of susceptible hosts equals $O(r)$. The probability density that an infectious unit is deposited on a susceptible host at distance r is thus given by $O(r)D(r)$. Therefore, the density of hosts receiving an infectious unit at a distance r from a randomly chosen infectious host equals $2\pi r O(r)D(r)$, and the total number of hosts receiving an infectious unit may be calculated by integrating over r . An infectious unit deposited on a susceptible host infects the host with probability β , the infection efficiency. The basic reproduction number then is given by

$$R_0 = \frac{\theta}{\mu} \beta \int_0^\infty 2\pi r O(r) D(r) dr. \quad (4)$$

In the standard non-spatial SIR model, the basic reproductive number would be given by

$$R_0 = \frac{\theta}{\mu} \beta \lambda. \quad (5)$$

Later on (Eq. 18) we will show that this is also the expression for R_0 in a randomly distributed host population.

Multiple host species

The approach is easily generalised to multiple host species. The multi-variate Ripley's K -function for hosts of type j around a host of type i is defined as: $K_{ij}(r) = \lambda_j^{-1} E \{ \text{number of individuals host type } i \text{ within distance } r \text{ of a randomly chosen host of type } j \}$.

This characterises the density of the population of species i around hosts of species j . Next, the O -ring statistic for species i around a host of type j is

$$O_{ij}(r) = \lambda_j \frac{1}{2\pi r} \frac{dK_{ij}(r)}{dr}. \quad (6)$$

We assume that the dispersal distribution around an infected host i and host j are the same, $D(r)$, although this is easily generalised. The number of infectious units produced per time unit by one host of type i is θ_i , the probability per time unit for the host to die is μ_i , and the probability that an infectious unit causes an infection on host i is β_i .

Next, we define the bi-variate basic reproduction number as the average number of secondary cases in species i produced by one infected individual of species j introduced into a population of susceptible individuals, R_{ij} ,

$$R_{ij} = \frac{\theta_j}{\mu_j} \beta_i \int_0^\infty 2\pi r O_{ij}(r) D(r) dr. \quad (7)$$

The dynamics of the number of infected hosts of all host types, $\mathbf{I}_{i,t}$ (i is 1 to K) in pathogen generation $t + 1$ then is described by

$$\mathbf{I}_{i,t+1} = \mathbf{A} \mathbf{I}_{i,t}. \quad (8)$$

where

$$\mathbf{I}_{i,t} = (I_{1,t}, I_{2,t}, I_{3,t}, \dots, I_{K,t})^T, \quad (9)$$

and

$$\mathbf{A} = \begin{pmatrix} R_{11} & \dots & R_{K1} \\ \vdots & \ddots & \vdots \\ R_{1K} & \dots & R_{KK} \end{pmatrix}. \quad (10)$$

Matrix \mathbf{A} is called the next-generation matrix. The pathogen's basic reproduction number then is calculated as the largest eigenvalue of the matrix \mathbf{A} (Diekmann et al. 1990, 2009, van den Bosch et al. 2008).

A numerical test

In this section we compare values of R_0 calculated using the expression derived above with numerical simulations of the epidemic process. The calculations use the Neymann–Scott

process for which the equations are given in the section ‘A closer analysis of the R_0 in the example case’.

The landscape

We consider cases with one host species and two host species. The host landscape is generated using a Neyman–Scott process (Neyman and Scott 1958, see also the section ‘A closer analysis of R_0 in the example case’).

Number of hosts and number of clusters

For host type 1 and host type 2 the total number of hosts are fixed numbers ($n_1 = 800$, $n_2 = 1200$). For each landscape the number of clusters (ρ_1 , ρ_2) is drawn from a uniform distribution on the interval $[2, 10]$. For each landscape generated the clusters are distributed randomly through space.

Landscape and dispersal scaling

The scale of the landscape is to some extent interchangeable with the scale of the pathogen dispersal. A landscape where we increase the distance between hosts by a factor of 2, but also increase the pathogen dispersal distances by a factor of 2, are dynamically the same landscapes. It therefore suffices to keep one of these fixed and vary the other. For simplicity in the programme we chose to keep the dispersal scale of the pathogen fixed and vary the scale of the landscape. To this end a landscape scaling factor, s , is introduced. All distances of hosts in the landscape are scaled with s . For each landscape the value of s is drawn from the interval $[0.1, 5]$. To ensure that half the landscapes are small and half the landscapes are large we selected half of the landscapes to have $s > 1$, and the other half to have $s \leq 1$, with uniform distributions used within each range (i.e. with $[0.1, 1]$ and $(1, 5]$). For each cluster separately its width is drawn from a uniform distribution on the interval $[0.025s, 0.2s]$, resulting in different cluster widths.

Structure of a cluster

For each cluster the number of hosts is drawn from a Poisson distribution with mean N_i/ρ_i , where ρ_i is the number of clusters for host type i , and where for the last cluster the size is determined by the fixed number of hosts of that type in the landscape minus the total number in all the clusters drawn up to the last cluster (landscapes which already had more than N_i hosts in the first $\rho_i - 1$ clusters due to stochastic effects were simply discarded). Individual hosts have displacement $(\Delta x, \Delta y)$ from the centre. The displacements are drawn from a Normal distribution (with zero mean and standard deviation given by the cluster width).

Figure 1A shows one realisation of this process. For this landscape the bi-variate O -ring statistics, calculated in R using the ‘spatstat’ package ver. 3.0-6 (Baddeley et al. 2015) are shown in Fig. 1C.

The pathogen dynamics

Each host can be in one of three states, uninfected/susceptible, S , infected and infectious, I , or removed and no longer

infectious, R . Infected hosts are removed at a constant rate μ . A host i that is susceptible becomes infected and infectious with a probability per time unit, φ_i , given by

$$\varphi_i = \beta_i \sum_{\text{all } I_j} D(r_{ij}). \quad (11)$$

where $D(r_{ij})$ is the probability that an infectious unit produced by an infected and infectious host j is deposited at a distance r_{ij} between host j and host i , and β_j is the infection efficiency. So, the rate at which a susceptible becomes infected depends on the sum of the rates at which infectious units are transferred from infected hosts j to the susceptible host i .

As a description of the dispersal density, we use the power-exponential density (Rieux et al. 2014, Fabre et al. 2021)

$$D(r) = \frac{ce^{-\left(\frac{r}{\alpha}\right)^c}}{2\pi\alpha^2\Gamma\left(\frac{2}{c}\right)}. \quad (12)$$

This flexible density, parameterised by a scale parameter, α , and shape parameter, c , can describe both thin tailed and fat tailed dispersal densities, depending on the value of c . For a value of $c = 2$, the dispersal density is a Normal density, for a value $c = 1$ it is an exponential density and for a value $c < 1$ the density is fat tailed (meaning the tail decreases slower than exponentially with r for large values of r). Figure 1B shows the shape of the dispersal distribution for three values of c .

Simulating the epidemics

After the landscape has been generated, and the number of infectious units produced per unit time per host, θ , the host death rate, μ_1 , and the infection efficiency, β , as well as the dispersal density parameters, c and α , have been defined, the simulated epidemic is started. Each run starts with seeding a small number of infectious hosts into the host landscape, Fig. 1D. The state and generation of each host individual is recorded through time, Fig. 1D–E.

Estimating the basic reproduction number

The method to estimate R_0 from the simulated data is based on the idea of fitting a non-spatial branching process type model to estimate directly the individual elements of the next generation matrix.

If the data in generation t are $\bar{I}_t = (I_{1,t}, I_{2,t})^T$ then the number of infected hosts of type 1 in the next generation will be the sum of two random variables, $A + B$. Lloyd-Smith et al. (2005) shows how exponentially-distributed infectious periods in a continuous time model cause the number of infections due to single infected individual to be distributed as Geometric(p), where $p = 1/(1 + R_0)$. Because the sum of X identically independent Geometric(p) distributions follows a negative binomial distribution with parameters X and p

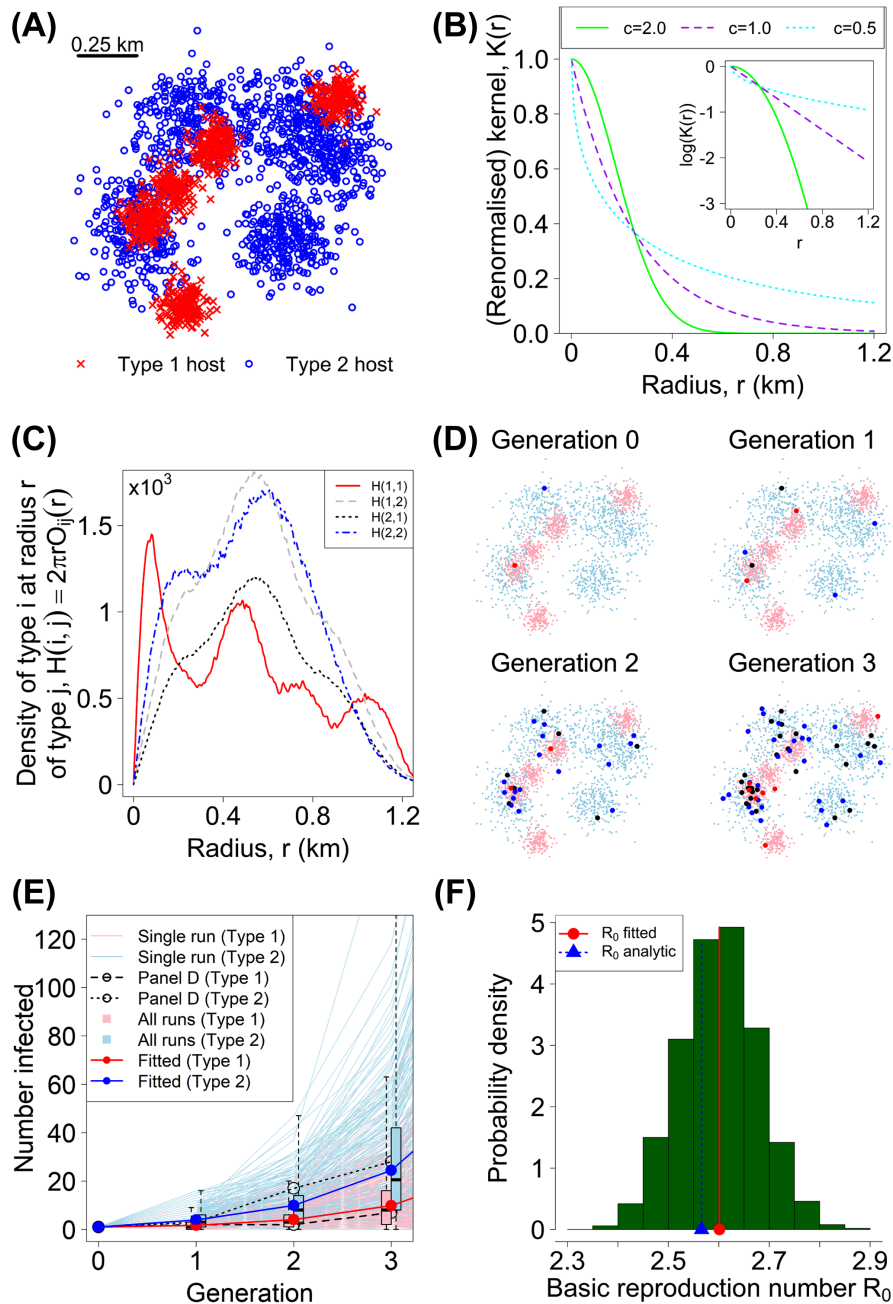


Figure 1. An overview of the processes in this paper. (A) An example of a two host species spatial pattern generated using the Neyman–Scott process. (B) The shape of the dispersal density, Eq. 12, for various values of the shape parameter c . The distributions are normalised such that all three go through $(0,1)$. This is done for illustrative purposes in this figure only. (C) The O-ring statistics for the two species host distribution in figure A: note the values on the y-axis are multiplied by 1000, and so range from 0 to about 1750. (D) The development of the first few generations of a single exemplar epidemic on the two host species distribution of (A). The hosts infected in each generation are shown by larger red (type 1) and blue (type 2) dots; black dots show post-infectious hosts infected in previous generations. (E) The dynamics of the number of infected hosts of type 1 and type 2 in a set of 250 simulations, on the two species host distribution in (A). The thick lines with filled dots show the results of fitting the non-spatial branching process model to the data from these 250 simulations: estimated parameters $R_{11}=1.240$, $R_{12}=2.580$, $R_{21}=0.486$, $R_{22}=1.394$ and so $R_0=2.440$. The box plots summarise the values of all 250 replicate simulations. The dotted lines with open dots show the numbers of infected hosts of each type in the single simulation shown in (D). (F) The distribution of the simulated basic reproduction number, R_0 , for 1000 sets of 250 simulations (i.e. 1000 sets of ensembles of simulations exemplified by those shown in (E)). The vertical lines and symbols on the x-axis show the values of the mean basic reproduction number as estimated by averaging the values as found in each of 1000 sets of 250 simulations and as calculated from the integral expression and the O-ring statistic (average value of $R_0^{\text{fitted}}=2.601$ and $R_0^{\text{analytic}}=2.566$). Parameter values used: $c=1$, $\theta_1=0.1$, $\theta_2=0.1$, $\rho_1=0.05$, $\rho_2=0.1$, $\mu_1=2.0$, $\mu_2=4.0$, $\alpha_1=0.25$, $\alpha_2=1.0$. Initial number of infected hosts of type 1 is 1, initial number of infected hosts of type 2 is 1.

(Boswell and Patil 1970), and assuming there is no density dependence, the distributions of A and B are therefore

$$\begin{aligned} A &\sim \text{NegBin}\left(I_{1,t}, \frac{1}{1+R_{11}}\right) \\ B &\sim \text{NegBin}\left(I_{2,t}, \frac{1}{1+R_{12}}\right) \end{aligned} \quad (13)$$

This allows us to calculate the contribution to the likelihood from infected hosts of type 1 in generation $t+1$, $L_{1,t+1}$, by convolution

$$L_{1,t+1} = \sum_{i=0}^{I_{1,t+1}} P(A=i)P(B=I_{1,t+1}-i), \quad (14)$$

which takes the sum over all possible ways $A+B$ could equal $I_{1,t+1}$.

For a single simulation, the likelihood, L , calculated using data up to generation F is therefore

$$L = \prod_{i=1}^F L_{1,i} L_{2,i}, \quad (15)$$

which is a function of the R_{ij} . Given data from M ($=250$) done with the same parameter values and done on the same landscape simulations – where in each simulation a single individual of each type was infected initially – we can then calculate an overall likelihood function over the ensemble of simulations by multiplying M expressions of the form above (with $F=2$). This allows us to estimate the best-fitting values of R_{ij} using standard numerical techniques to maximise the log-likelihood and to use these estimates to estimate the overall value of R_0 as the largest eigenvalue of the next generation matrix.

Comparing the simulated and analytically calculated R_0

A first test of the expression for R_0 was done using the landscape in Fig. 1A and the dispersal densities shown in Fig. 1B. One realisation with a seed of two randomly placed infected hosts – one infected host of each type – in Generation 0 of our epidemic simulation (written in the C programming

language; available online at <https://github.com/nikcuniffe/rZeroORing>) is shown in Fig. 1D and the densities of the two infected host types in the first three generations in a single set of 250 replicated simulations are shown in Fig. 1E. Figure 1F shows the distribution of R_0 values calculated with the method explained above, with initial condition of one infected host for each host type, over 1000 independent sets of 250 simulations. The mean value of the R_0 calculated from the 1000 sets of 250 simulations (red dot) is very close to the R_0 calculated from the analytical expression (blue triangle); numerical integration was done in R using the 'sfsmisc' package ver. 1.1-16 (Maechler 2023).

The results of sets of simulations for the three dispersal distributions (Normal, exponential and fat-tailed) are summarised in Table 1. There is a close correspondence between the simulated R_0 and the R_0 calculated from the analytic expression both in the case of one host and in the case of two host types.

To assess how far landscape structure affects the accuracy of the analytic R_0 expression, 100 replicate landscapes were generated as described above. Each landscape is generated by drawing a value for s and ρ as well as drawing values for the cluster width. For each of these 100 different landscapes, 10 replicate sets of 250 simulations were done using the baseline parameter values as in the caption of Table 1, and 10 values of R_0 calculated. The value of the simulated R_0 values and the R_0 calculated from the expression are plotted in Fig. 2A, C. Clearly there is a nearly perfect agreement between the simulated and the calculated R_0 value irrespective of the landscape and the dispersal density used.

To assess the effect of the pathogen lifecycle parameters (the number of infectious units θ_i , probability per time unit to die μ_i , probability that an infectious unit causes an infection β_i , the shape parameter of the dispersal density c and the dispersal scale parameter α) 200 sets of parameter values were drawn. Initially values were drawn from homogeneous distributions on the interval 20–200% of the baseline value of the parameter. Further parameter sets were generated by drawing only values for one of the parameters and retaining the other parameters at their baseline value. We restricted to $c=0.5$, $c=1.0$ and $c=2.0$. Using the landscape of Fig. 1A and the three dispersal densities of Fig. 1B, 10 replicate sets of 250 runs were done for each set of pathogen life-cycle parameters and 10 values of R_0 calculated. Figure 2B and D show that there is a near perfect correspondence between the simulated

Table 1. The basic reproduction number of the simulated epidemic and calculated from the analytic expression for the baseline parameter values. ($\theta_1=0.1$; $\theta_2=0.1$; $\beta_1=0.05$; $\beta_2=0.1$; $\mu_1=2$; $\mu_2=4$; $\alpha=0.25$). Numbers in the 'fitted' cells are the median values over an ensemble of 1000 sets of 250 simulations; the values in brackets are the 5 and 95 percentiles.

	Gaussian ($c=2$)		Exponential ($c=1$)		Fat tailed ($c=0.5$)	
	Fitted	Analytic	Fitted	Analytic	Fitted	Analytic
Species 1 only	1.322 (1.201–1.464)	1.286	1.365 (1.257–1.491)	1.349	1.542 (1.414–1.692)	1.551
Species 2 only	1.285 (1.150–1.401)	1.237	1.569 (1.450–1.719)	1.541	2.063 (1.926–2.193)	2.063
Both types	2.182 (1.999–2.361)	2.078	2.613 (2.439–2.807)	2.566	3.431 (3.241–3.648)	3.426

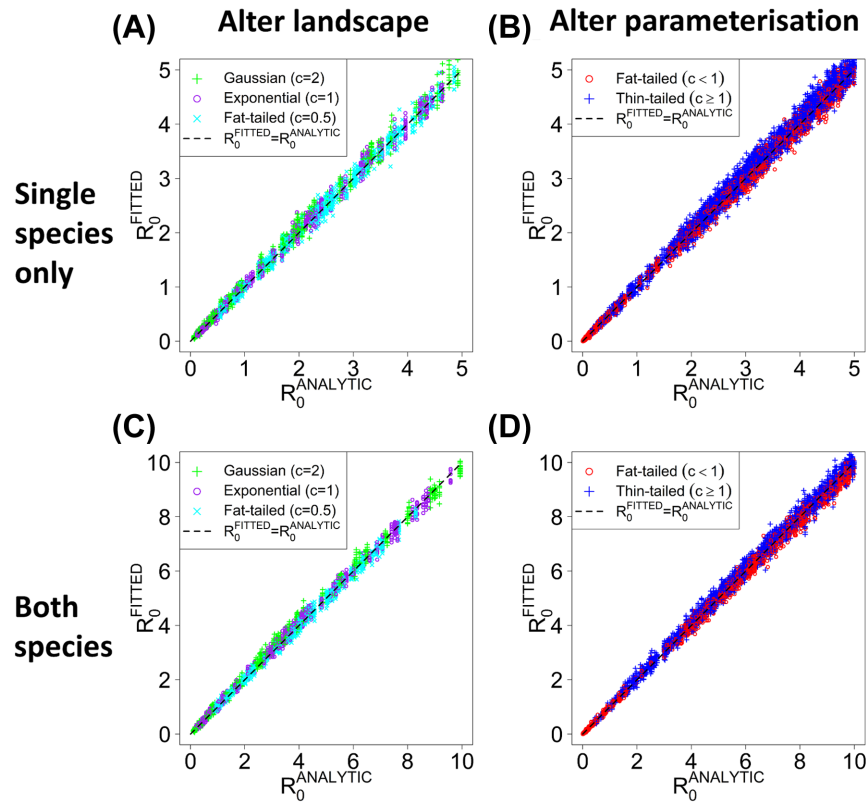


Figure 2. Comparison of the basic reproductive number calculated from simulations, R_0^{fitted} , and calculated using the integral expression and the O-ring statistic, R_0^{analytic} . (A) and (B) are for the single species case, (C) and (D) for the two species case. (A) and (C) show calculations for a range of generated host distributions, (B) and (D) show calculations for a range of epidemiological parameters. Each dot in each figure is the result of estimating R_0 from 250 simulations using one set of life cycle parameters on one single landscape; note that 10 replicate sets of 250 simulations were performed for each landscape \times parameter set combination (there are vertical lines of 10 points in each plot, most clearly visible towards the right-hand side of (C)). For the methods of generating host distributions and varying parameters see the main text.

R_0 and the R_0 calculated from the analytic expressions irrespective of the values of the pathogen life-cycle parameters.

$$R_0 = \frac{\theta}{\mu} \beta \lambda \int_0^\infty 2\pi r D(r) dr = \frac{\theta}{\mu} \beta \lambda := \tilde{R}_0. \quad (18)$$

A closer analysis of R_0 in the example case

Complete spatial randomness

The homogeneous Poisson process can be used to generate a completely spatially random host distribution. The Ripley's K function for this process has the form

$$K(r) = \pi r^2. \quad (16)$$

This is intuitively clear as the number of hosts within a circle of radius r around any host is proportional to the area of the circle. The O-ring statistic takes the form

$$O(r) = \lambda, \quad (17)$$

which indicates that at every distance from a randomly selected host the host density is the same as the mean host density. The basic reproduction number then becomes

As expected, the basic reproduction number is linearly dependent on host density and on the pathogen transmission rate. This holds for the R_0 in standard SEIR models as well (Kermack and McKendrick 1927). When infectious units disperse further from their host, increasing α , the basic reproduction number stays the same in a spatially random host population. Also, the shape of the dispersal density, whether it has a fat tail, $c < 1$, or an exponentially bounded tail, $c > 1$, does not affect the value of the basic reproduction number in a spatially random host population.

Regular host distributions

There is a variety of point processes that generates host distributions more regular than the complete spatially random, CSR, distribution. The Strauss process generates host distributions where the density of hosts within a critical distance δ of each host is smaller than expected under CSR. These distributions are generated starting with a CSR distribution of hosts

and deleting hosts within the critical distance δ of a host with probability $1 - \gamma$. An approximation to the Ripley's K function for the Strauss process is given by (Isham 1984)

$$K(r) = \begin{cases} \gamma \pi r^2 & r \leq \delta \\ \pi r^2 - (1 - \gamma) \pi \delta & r > \delta \end{cases} \quad (19)$$

The O -ring statistic takes the form

$$O(r) = \begin{cases} \lambda \gamma & r \leq \delta \\ \lambda & r > \delta \end{cases} \quad (20)$$

The basic reproduction number is given by

$$R_0 = \tilde{R}_0 \left(1 - (1 - \gamma) \int_0^\delta 2\pi r D(r) dr \right), \quad (21)$$

and substituting the dispersal density we find

$$R_0 = \tilde{R}_0 \left(1 - \frac{c}{\alpha^2 \Gamma\left(\frac{2}{c}\right)} (1 - \gamma) \int_0^\delta r e^{-\left(\frac{r}{\alpha}\right)^c} dr \right). \quad (22)$$

Note that the factor $\frac{c}{\alpha^2 \Gamma\left(\frac{2}{c}\right)} \int_0^\delta r e^{-\left(\frac{r}{\alpha}\right)^c} dr$ is always between

0 and 1, with 0 for $\delta = 0$ and 1 for $\delta = \infty$. Therefore for $\delta \rightarrow \infty$, $R_0 \rightarrow \gamma \tilde{R}_0$ and as $\delta \rightarrow 0$, $R_0 \rightarrow \tilde{R}_0$.

For $c = 2$ this becomes

$$R_0 = \tilde{R}_0 \left(1 - (1 - \gamma) \left(1 - e^{-\left(\frac{\delta}{\alpha}\right)^2} \right) \right). \quad (23)$$

For $c = 1$ this becomes

$$R_0 = \tilde{R}_0 \left(1 - (1 - \gamma) \left(1 - \left(1 + \frac{\delta}{\alpha} \right) e^{-\left(\frac{\delta}{\alpha}\right)} \right) \right). \quad (24)$$

We will first interpret the effect of parameter values on the basic reproduction number in general from the form of the Eq. 22–24, and then show a few examples for specific parameter values in Fig. 3.

Host density and regularity

The basic reproduction number is linear in the R_0 of the complete spatial randomness case, \tilde{R}_0 . This implies that

the basic reproduction number increases linearly with the mean host density, λ , and with the pathogen transmission rate. Moreover, host regularity will always decrease the basic reproduction number, and the greater the level of regularity the smaller R_0 . Increasing the area around a host in which the host density is smaller than expected, δ , always decreases R_0 . The smaller the density of hosts in the vicinity of a host, $1 - \gamma$, the smaller R_0 .

Pathogen dispersal density

In a regular host population, the basic reproduction number increases with increasing dispersal distance, α . This can be explained by considering the number of infectious units that will be deposited on a host. In a regular host distribution, infectious units will have a larger chance to be deposited on a host if they move beyond the area around the host where the host density is smaller than average in the entire population.

The effect of the shape of the dispersal density, c , on the basic reproduction number in regular host populations is more difficult to see from the expression. We therefore explore this numerically.

Numerical examples

The above discussion shows the qualitative effects of host regularity and pathogen dispersal density on the basic reproduction number. The analysis does not reveal the size of the effects nor is it possible to assess the effect of the dispersal density shape parameter, c , on the basic reproduction number. Numerical solutions of the equation for R_0 shown in Fig. 3 explores the size of the effects of host regularity, pathogen dispersal and the shape parameter on the basic reproduction number.

Figure 3A–B show that when the basic reproduction number in a spatially random host population is larger than unity, a more regular host distribution can bring the basic reproduction number below unity, preventing epidemic invasion. This requires large exclusions zones, δ , or a high strength of exclusion, $1 - \gamma$. The basic reproduction number increases with the dispersal scale, α , (Fig. 3C) and decreases with increasing value of the shape parameter (Fig. 3D). Shape parameters $c > 1$ are dispersal densities with a thin tail (the tail decreases faster than exponential with distance for large distances) and for $c < 1$ the dispersal density is fat tailed (the tail decreases slower than exponentially with distance for large distances). That a thinner tail decreases R_0 is intuitively explained because it makes the probability that an infectious unit reaches a host outside the exclusion zone to be smaller.

Spatially clustered host distributions

The Neyman–Scott process generates spatially clustered host distributions. Ripley's K function for the Neyman–Scott process is given by

$$K(r) = \pi r^2 + \frac{1 - e^{-\frac{r^2}{4\sigma^2}}}{\rho}. \quad (25)$$

where ρ is the cluster density and σ is the standard deviation of the distance of a host to the centre of the cluster it belongs to (Diggle 2003). From this we find that the O -ring statistic is given by:

$$O(r) = \lambda \left(1 + \frac{1}{\rho 4\pi\sigma^2} e^{-\frac{r^2}{4\sigma^2}} \right), \quad (26)$$

and the basic-reproduction number is

$$R_0 = \tilde{R}_0 \left(1 + \int_0^\infty 2\pi r \frac{1}{\rho 4\pi\sigma^2} e^{-\frac{r^2}{4\sigma^2}} D(r) dr \right). \quad (27)$$

Substituting the dispersal density, we get

$$R_0 = \tilde{R}_0 \left(1 + \frac{c}{\rho 4\pi\alpha^2 \Gamma\left(\frac{2}{c}\right) \sigma^2} \int_0^\infty r e^{-\left(\frac{r}{\alpha}\right)^c - \frac{r^2}{4\sigma^2}} dr \right). \quad (28)$$

For $c=2$ this becomes

$$R_0 = \tilde{R}_0 \left(1 + \frac{1}{\rho\pi(\alpha^2 + 4\sigma^2)} \right). \quad (29)$$

For $c=1$ this becomes

$$R_0 = \tilde{R}_0 \left(1 + \frac{1}{2\rho\pi\alpha^2} \left(1 - \sqrt{\pi} \frac{\sigma}{\alpha} e^{\left(\frac{\sigma}{\alpha}\right)^2} \operatorname{erfc}\left(\frac{\sigma}{\alpha}\right) \right) \right). \quad (30)$$

Where erfc is the complementary error function.

We will first interpret the effect of parameter values on the basic reproduction number in general from the form of the Eq. 28–30, and then show a few examples for specific parameter values in Fig. 3.

Host density and clustering

Interpreting the form of Eq. 28–30 we see that as in the case with host regularity, the basic reproduction number is linear in the R_0 of the complete spatial randomness case, \tilde{R}_0 . This implies that the basic-reproduction number increases linearly with the mean host density, λ , and with the pathogen transmission rate. Moreover, host clustering will always increase the basic-reproduction number and the greater the level of clustering the larger R_0 . Increasing σ decreases the level of host clustering and decreases the value of R_0 . Increasing the number of clusters, ρ , in a host population with a fixed number of hosts, decreases the level of clustering and decreases R_0 .

That R_0 decreases with the level of clustering can be explained by the fact that for any biologically relevant dispersal kernel more infectious units will be deposited closer to the infected host than further away from the infected host. In the case of a clustered host distribution this leads to more infectious units being deposited on susceptible hosts than in the case of a spatially random host distribution.

Pathogen dispersal density

Interpreting the form of Eq. 28–30 we see that in a clustered host population, the basic reproduction number decreases with increasing dispersal distance. This can be explained as before by considering the number of infectious units that will be deposited on a host. In a clustered host distribution, infectious units will have a higher probability of moving out of the cluster from which they originate when the dispersal scale is larger. Infectious units that moved out of the host cluster they originate from will have a smaller probability to be deposited on a host and cause an infection.

The effect of the shape of the dispersal density, c , on the basic reproduction number in clustered host populations is more difficult to see from the expression. We therefore explore this numerically.

Numerical examples

Numerical solutions of the equation for R_0 shown in Fig. 3 explores the size of the effects of host clustering and pathogen dispersal on the basic reproduction number.

The figure shows that when a pathogen has a basic reproduction number smaller than unity in a spatially random host population, host clustering can bring the R_0 above unity. In Fig. 3E $1/\sigma$ equals zero for a random host distribution. The figure shows that the effect of host clustering on the basic reproduction number can, depending on the values of the other parameters, be large. In the numerical example shown the basic reproduction number is smaller than unity for random host distributions and becomes four for very clustered host distributions. An increased density of host clusters, ρ , (Fig. 3F) decreases R_0 because increasing ρ at constant host density decreases the level of host clustering. The effect of the pathogen dispersal scale, Fig. 3G, can also be considerable. Increasing the dispersal distance from close to 0 to large values decreases R_0 from around 4 to less than 1.

The shape of the dispersal distribution has a pronounced effect on the basic reproduction number, with fat tailed distributions having small values of R_0 . Thinner tails imply that a larger fraction of the infectious units is deposited closer to the parent host, which increases the probability it is deposited in a host in the cluster it originates from.

Discussion

We introduced a method to calculate the basic reproduction number, R_0 , for a spatially explicit host population. The

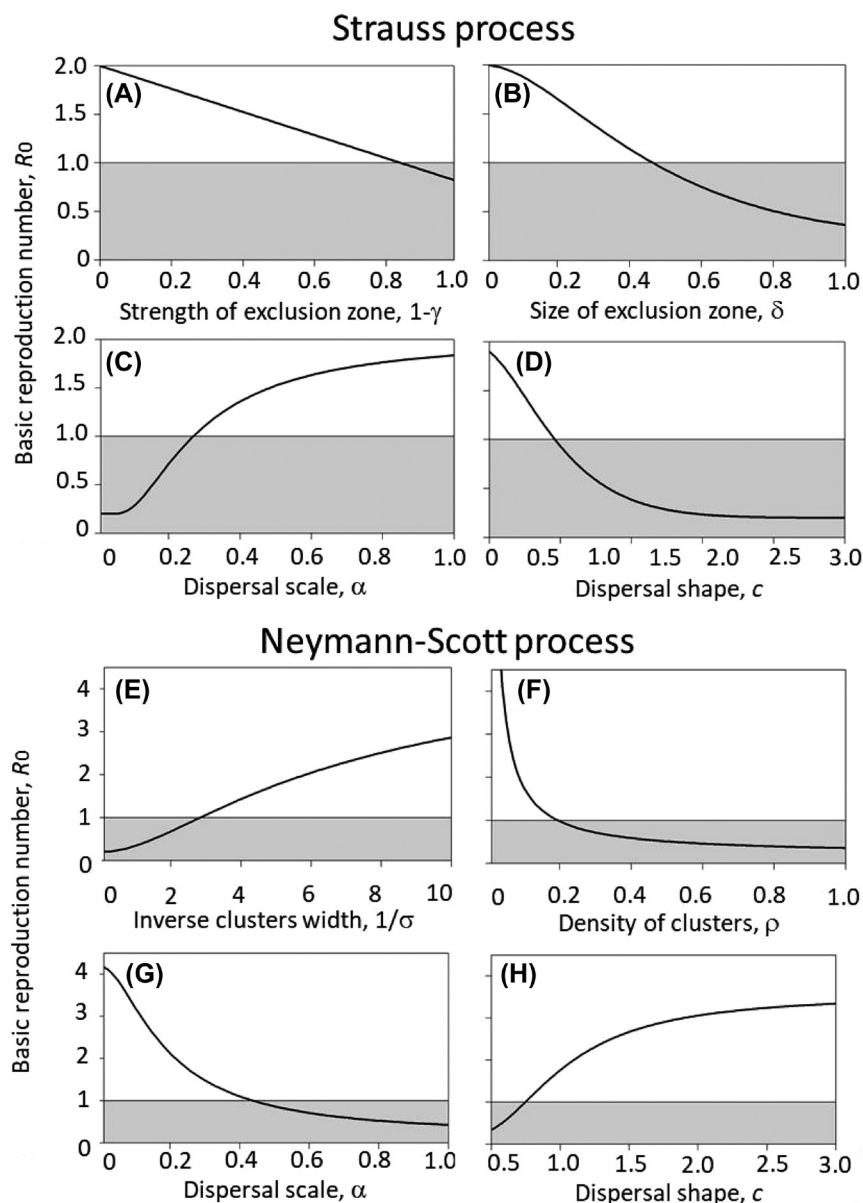


Figure 3. The effect of parameter values on the basic reproduction number. (A–D) are for the Strauss process (regular host distributions), (E–H) for the Neyman–Scott process (clustered host distributions). (A) shows the effect of the strength of the exclusion zone on R_0 , (B) the effect of the size of the exclusion zone on R_0 , (C) the effect of the dispersal scale on R_0 , and (D) the effect of the dispersal density shape parameter on R_0 . (E) shows the effect of the inverse of the cluster width on R_0 , (F) the effect of the density of clusters on R_0 , (G) the effect of the dispersal scale on R_0 , and H the effect of the dispersal density shape parameter on R_0 . For this figure for the Strauss process $\delta=0.5$, $\gamma=0.1$, and the other parameters are as given in the subscript of Fig. 1. For the Neyman–Scott process and the other parameters as in Fig. 1.

spatial host distribution is characterised via the O -ring statistic which is interpreted as the density of hosts at a distance r from a randomly selected host. The O -ring statistic is a measure from spatial statistics frequently used in the study of the ecology of plant spatial population dynamics (Wiegand and Moloney 2004, Law et al. 2009). The method to calculate R_0 thus establishes a connection between the epidemiology of infectious diseases in spatially structured host populations and spatial statistics methods from spatial ecology.

We presented the calculation of R_0 for a single host species as well as for multiple host species. In the derivation of the R_0

for multiple host species we used the next-generation matrix approach as developed by Diekmann et al. (1990, 2009). We note here that this does not imply that the method is only valid for discrete time epidemic models. The next generation matrix approach is valid for both continuous time and discrete time models (Diggle 2003): indeed the simulations on which we tested the analytic method were continuous time models.

The expression for R_0 has a clear intuitive biological interpretation that allows it to be used in the analysis of epidemics on realistic spatial host patterns. Basically, the product $D(r)O(r)$ describes how a pathogen ‘sees’ the host population

around and infected host individual. $D(r)O(r)$ is the probability that an infectious unit released from an infectious host lands on another host at distance r from the originating host. Calculating the basic reproduction number then is a matter of adding up all of the $D(r)O(r)$ contributions through the entire space and multiplying with the number of effective infectious units, $\theta\beta/\mu$.

The values of the parameters needed to calculate R_0 can be obtained experimentally, though some are complicated to estimate. Values are published for some patho-systems. The pathogen life cycle parameters 'the number of infectious units', θ , the 'probability per time unit to die', μ , and the probability that an infectious unit causes an infection, β , appear in all expressions for R_0 as the basic reproductive number in a spatially random host distribution, $R_0 = \frac{\theta}{\mu}\beta\lambda$, which is the same as the basic reproductive number in the SIR models. Values of R_0 for plant pathogens have been published (Wadkin et al. 2024). Dispersal densities of the infectious units are notoriously difficult to quantify as these infectious units can disperse over large distances which then are hard to quantify using sampling. Some dispersal densities for plant pathogens based on direct measurement of dispersal distances have been published (Rieux et al. 2014), and indirect estimates, usually based on fitting a model to spatially refined epidemiological data can also be found in the literature (Neri et al. 2014). Finally, the O -ring statistic has to be derived from a spatially resolved host map. This must be done on a case-by-case basis, but since the host is stationary this is relatively easy to do.

Several authors have studied the effect of spatial host distribution on epidemic dynamics using moment closure techniques (Bolker and Pacala 1999, Filipe and Maule 2003, Brown and Bolker 2004, Bauch 2005). These methods result in expressions for R_0 from which valuable insight is gained into the effects of spatial host structure on the dynamics of infectious pathogens and give insight into the situation when the epidemic runs into density dependence. Of these Filipe and Maule introduced a function describing the mean host density as function of distance from a randomly chosen host. This function is mathematically similar to the O -ring statistics. Due to the use of the moment closure technique the expression arrived for R_0 does however have a very complex structure and can not easily be applied to realistic spatial host distributions. Filipe and Maule's R_0 expression also does not allow for an intuitive interpretation along the lines as the expression for R_0 introduced in this paper.

The results of our qualitative and quantitative analysis generally agree with the results by other authors. Bolker (1999), Brown and Bolker (2004) and North and Ovaskainen (2007), showed that the initial epidemic growth rate is larger in a clustered host population compared to that in a spatially random population. Since the generation time is kept constant in their simulations this implies that R_0 in a clustered host population is larger than in a spatially random host population, which agrees with our findings.

Brown and Bolker (2004) pay special attention to the local depletion of susceptible hosts and its effect on the

net-reproductive number. They show that for very concentrated dispersal kernels the high infection rate of neighbouring hosts can deplete the availability of susceptible host to such an extent that the epidemic does not develop where on basis of an R_0 calculation it is expected to develop. They develop a moment closure method that incorporates this local host depletion into the calculation of the basic reproduction number. We have in our simulations not noticed that a concentrated dispersal distribution affects the numerically derived value of R_0 to deviate significantly from the value calculated using the O -ring statistic.

In the example studied we use three cases 1) complete spatial randomness, 2) over dispersed host patterns generated by the Strauss process and 3) clustered host distributions generated by the Neyman-Scott method to generate the spatial host distribution. The method is however equally well applicable to any real-life spatial host distribution from which the O -ring statistic can be calculated numerically (Wiegand and Moloney 2004). This opens the possibility to use the method in the analysis of practically relevant cases. For example, the expression for R_0 can be used in land use planning for nature development and agriculture to reduce the risk of disease outbreaks. For example, increased tree cover has been widely acknowledged as a fundamental requirement to mitigate the effects of climate change and restore natural habitats (Griscom et al. 2017, Chazdon and Brancalion 2019). However, tree pests and diseases are a continuous threat to tree health (Boyd et al. 2013). Any increase in forested area will increase the opportunity for pathogens to invade and spread. The question is thus how these new forests could be spatially structured to minimise the risk of outbreaks of pests and diseases and/or be resilient when such outbreaks do occur. Using the expression for R_0 it is possible, for known pathogens, to plan the spatial arrangement of forest lots such that the risk of epidemic damage is minimised. The same holds for agricultural fields threatened by crop pathogens.

Our method to calculate the basic reproduction number of an infectious disease in a spatially structure host population forms a natural connection between the spatial statistics measure used in the study of the spatial ecology of plant populations and the dynamics of pathogen on these plant populations. Using the method introduces it is possible to study the effect of real-life spatial host distributions on epidemic dynamics.

Funding – JH: Rothamsted Research receives strategic funding from the Biotechnological and Biological Sciences Research Council of the United Kingdom. We acknowledge support from the Growing Health (BB/X010953/1) Institute Strategic Programme. For the purpose of open access, the author has applied a Creative Commons Attribution (CC BY) licence to any Author Accepted Manuscript version arising from this submission.

Author contributions

Frank van den Bosch: Conceptualization (equal); Formal analysis (equal); Investigation (equal); Methodology (equal);

Writing – original draft (equal). **Joe Helps:** Conceptualization (equal); Methodology (equal); Supervision (equal); Writing – review and editing (equal). **Nik J. Cuniffe:** Formal analysis (equal); Investigation (equal); Software (equal); Visualization (equal); Writing – review and editing (equal).

Data availability statement

Data are available from the Dryad Digital Repository: <https://doi.org/10.5061/dryad.kh18932fw> (van den Bosch et al. 2024).

References

- Baddeley, A., Rubak, E. and Turner, R. 2015. Spatial point patterns: methodology and applications with R. – Chapman and Hall/CRC Press.
- Bauch, C. T. 2005. The spread of infectious diseases in spatially structured populations: an invasy pair approximation. – *Math. Biosci.* 198: 217–237.
- Ben-Said, M. 2021. Spatial point-pattern analysis as a powerful tool in identifying pattern–process relationships in plant ecology: an updated review. – *Ecol. Process.* 10: 56.
- Bolker, B. M. 1999. Analytic models for the patchy spread of plant disease. – *Bull. Math. Biol.* 61: 849–874.
- Bolker, B. M. and Pacala, S. W. 1999. Spatial moment equations for plant competition: understanding spatial strategies and the advantages of short dispersal. – *Am. Nat.* 153: 575–602.
- Boswell, M. T. and Patil, G. P. 1970. – In: Patil, G. P. (ed.), *Random counts in models and structures*, vol. 7–8. Pennsylvania State Univ. Press.
- Boyd, I. L., Freer-Smith, P. H., Gilligan, C. A. and Godfray, H. C. J. 2013. The consequence of tree pests and diseases for ecosystem services. – *Science* 342: 1235–1237.
- Brown, D. H. and Bolker, B. M. 2004. The effects of disease dispersal and host clustering on the epidemic threshold in plants. – *Bull. Math. Biol.* 66: 341–371.
- Burdon, J. J. and Chilvers, G. A. 1976. The effect of clumped planting patterns on epidemics of damping-off disease in cress seedlings. – *Oecologia* 23: 17–29.
- Chazdon, R. and Brancalion, P. 2019. Restoring forests as a means to many ends, An urgent need to replenish tree canopy cover calls for holistic approaches. – *Science* 365: 24–25.
- Cressie, N. 1991. *Statistics for spatial data*. – Wiley & Sons.
- Cuniffe, N. J., Stutt, R. O. J. H., DeSimone, R. E., Gottwald, T. R. and Gilligan, C. A. 2015. Optimising and communicating options for the control of invasive plant disease when there is epidemiological uncertainty. – *PLoS Comput. Biol.* 11: e1004211.
- Cuniffe, N. J., Cobb, R. C., Meentemeyer, R. K. and Gilligan, C. A. 2016. Modeling when, where, and how to manage a forest epidemic, motivated by sudden oak death in California. – *Proc. Natl Acad. Sci. USA* 113: 5640–5645.
- Diekmann, O., Heesterbeek, J. A. P. and Metz, J. A. J. 1990. On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations. – *J. Math. Biol.* 28: 365–382.
- Diekmann, O., Heesterbeek, J. A. P. and Roberts, M. G. 2009. The construction of next-generation matrices for compartmental epidemic models. – *J. R. Soc. Interface* 7: 873–885.
- Diggle, P. J. 2003. *Statistical analysis of spatial point patterns*, 2nd edn. – Arnold.
- Fabre, F., Coville, J. and Cuniffe, N. J. 2021. Optimising reactive disease management using spatially explicit models at the landscape scale. – In: Scott, P., Strange, R., Korsten, L. and Gullino, L. (eds), *Chapter 4 plant diseases and food security in the 21st century*. Springer.
- Filipe, J. A. N. and Maule, M. M. 2003. Analytical methods for predicting the behaviour of population models with general spatial interactions. – *Math. Biosci.* 183: 15–35.
- Griscom, B. W. et al. 2017. Natural climate solutions. – *Proc Natl Acad. Sci. USA* 114: 11645–11650.
- Isham, V. 1984. Multitype Markov point processes: some approximations. – *Proc. R. Soc. A* 391: 39–53.
- Keeling, M. J. and Grenfell, B. T. 2000. Individual-based perspectives on R_0 . – *J. Theor. Biol.* 203: 51–61.
- Kermack, W. O. and McKendrick, A. G. 1927. A contribution to the mathematical theory of epidemics. – *Proc. R. Soc. A* 115: 700–721.
- Law, R., Illian, J., Burslem, D. F. R. P., Gratzner, G., Gunatilleke, C. V. S. and Gunatilleke, I. A. U. N. 2009. Ecological information from spatial patterns of plants: insights from point process theory. – *J. Ecol.* 97: 616–628.
- Lloyd-Smith, J. O., Schreiber, S. J., Kopp, P. E. and Getz, W. M. 2005. Superspreading and the effect of individual variation on disease emergence. – *Nature* 438: 355–359.
- Madden, L. V., Jeger, M. J. and van den Bosch, F. 2000. A theoretical assessment of the effects of vector-virus transmission mechanisms on plant virus disease epidemics. – *Phytopathology* 90: 576–594.
- Maechler, M. 2023. *sfsmisc: utilities from ‘Seminar fuer Statistik’ ETH Zurich*. – R package ver. 1.1-16, <https://CRAN.R-project.org/package=sfsmisc>.
- Meentemeyer, R. K., Cuniffe, N. J., Cook, A. R., Filipe, J. A. N., Hunter, R. D., Rizzo, D. M. and Gilligan, C. A. 2011. Epidemiological modeling of invasion in heterogeneous landscapes: spread of sudden oak death in California (1990–2030). – *Ecosphere* 2: art17.
- Neri, F. M., Cook, A. R., Gibson, G. J., Gottwald, T. R. and Gilligan, C. A. 2014. Bayesian analysis for inference of an emerging epidemic: citrus canker in urban landscapes. – *PLoS Comput. Biol.* 10: e1003587.
- Neyman, J. and Scott, E. L. 1958. Statistical approach to problems of cosmology. – *J. R. Stat. Soc. B* 20: 1–29.
- North, A. and Ovaskainen, O. 2007. Interactions between dispersal, competition, and landscape heterogeneity. – *Oikos* 116: 1106–1119.
- Rieux, A., Soubeyrand, S., Bonnot, F., Klein, E. K., Ngando, J. E., Mehl, A., Ravigne, V., Carlier, J. and Bellaire, L. D. L. D. 2014. Long-distance wind-dispersal of spores in a fungal plant pathogen: estimation of anisotropic dispersal kernels from an extensive field experiment. – *PLoS One* 9: e103225.
- Ripley, B. D. 1976. The second-order analysis of stationary point processes. – *J. Appl. Probab.* 13: 255–266.
- Ripley, B. D. 1977. Modeling spatial patterns. – *J. R. Stat. Soc. B* 39: 172–212.
- Suprunenko, Y. F., Cornell, S. J. and Gilligan, C. A. 2021. Analytical approximation for invasion and endemic thresholds, and the optimal control of epidemics in spatially explicit individual-based models. – *J. R. Soc. Interface* 18: 20200966.

- te Beest, D. E., Hagenaars, T. J., Stegeman, J. A., Koopmans, M. P. and van Boven, M. 2011. Risk based culling for highly infectious diseases of livestock. – *Vet. Res.* 42: 81.
- Tildesley, M. J., Savill, N. J., Shaw, D. J., Deardon, R., Brooks, S. P., Woolhouse, M. E. J., Grenfell, B. T. and Keeling, M. J. 2006. Optimal reactive vaccination strategies for a foot-and-mouth outbreak in the UK. – *Nature* 440: 83–86.
- van den Bosch, F., McRoberts, N., van den Berg, F. and Madden, L. 2008. The basic reproduction number of plant pathogens: matrix approaches to complex dynamics. – *Phytopathology* 98: 239–249.
- van den Bosch, F., Helps, J. and Cunliffe, N. J. 2024. Data from: The basic-reproduction number of infectious diseases in spatially structured host populations. – Dryad Digital Repository, <https://doi.org/10.5061/dryad.kh18932fw>.
- Wadkin, L. E., Holden, J., Ettelaie, R., Holmes, M. J., Smith, J., Golightly, A., Parker, N. G. and Baggaley, A. W. 2024. Estimating the reproduction number, R_0 , from individual-based models of tree disease spread. – *Ecol. Modell.* 489: 110630.
- Wiegand, T. and Moloney, K. A. 2004. Rings, circles, and null-models for point pattern analysis in ecology. – *Oikos* 104: 209–229.