Ecological Modelling 220 (2009) 3353–3361

Contents lists available at ScienceDirect

Ecological Modelling

journal homepage: www.elsevier.com/locate/ecolmodel

Epidemiological risk assessment using linked network and grid based modelling: *Phytophthora ramorum* and *Phytophthora kernoviae* in the UK

Thomas D. Harwood[a,*], Xiangming Xu[b], Marco Pautasso[c], Mike J. Jeger[c], Michael W. Shaw[a]

[a] *School of Biological Sciences, University of Reading, Lyle Tower, Whiteknights, Reading RG6 6AS, UK*
[b] *East Malling Research, New Road, East Malling, ME19 6BJ Kent, UK*
[c] *Division of Biology, Imperial College London, Silwood Park, Ascot SL5 7PY, UK*

ARTICLE INFO

Article history:
Received 23 April 2009
Received in revised form 11 August 2009
Accepted 17 August 2009
Available online 8 September 2009

Keywords:
Invasion biology
Landscape pathology
Network theory
Phytosanitary regulation
Plant health
Sudden Oak Death
Rhododendron ponticum
Vaccinium myrtillus

ABSTRACT

We developed a stochastic simulation model incorporating most processes likely to be important in the spread of *Phytophthora ramorum* and similar diseases across the British landscape (covering *Rhododendron ponticum* in woodland and nurseries, and *Vaccinium myrtillus* in heathland). The simulation allows for movements of diseased plants within a realistically modelled trade network and long-distance natural dispersal. A series of simulation experiments were run with the model, representing an experiment varying the epidemic pressure and linkage between natural vegetation and horticultural trade, with or without disease spread in commercial trade, and with or without inspections-with-eradication, to give a $2 \times 2 \times 2 \times 2$ factorial started at 10 arbitrary locations spread across England. Fifty replicate simulations were made at each set of parameter values. Individual epidemics varied dramatically in size due to stochastic effects throughout the model. Across a range of epidemic pressures, the size of the epidemic was 5–13 times larger when commercial movement of plants was included. A key unknown factor in the system is the area of susceptible habitat outside the nursery system. Inspections, with a probability of detection and efficiency of infected-plant removal of 80% and made at 90-day intervals, reduced the size of epidemics by about 60% across the three sectors with a density of 1% susceptible plants in broadleaf woodland and heathland. Reducing this density to 0.1% largely isolated the trade network, so that inspections reduced the final epidemic size by over 90%, and most epidemics ended without escape into nature. Even in this case, however, major wild epidemics developed in a few percent of cases. Provided the number of new introductions remains low, the current inspection policy will control most epidemics. However, as the rate of introduction increases, it can overwhelm any reasonable inspection regime, largely due to spread prior to detection.

1. Introduction

Planning effective containment of a disease requires models which capture the essentials of all the important possible infection pathways. In some cases an abstract model of the system such as a network or supply chain analysis may be sufficient to provide the basis for analysis and decision making. Often however, the real system has spatial structure which further defines the development of the epidemic, and network models in isolation represent only part of the system. Where disease spread occurs continuously within the landscape, any network overlaid on this, such as those representing trade pathways, will act as shortcuts for the disease, essentially distorting the topology of the landscape. In this case, neither the network nor the underlying landscape can be viewed as independent systems; their linkage is a fundamental property of the epidemiological system.

In this paper, we demonstrate one way in which this can be done. We implemented standard models of epidemic development (SEIS) and pathogen dispersal within a geographically explicit grid of susceptible plants on which a trade network is superimposed. We took this approach in order to assist risk assessment for *Phytophthora ramorum* (Werres et al., 2001) and *Phytophthora kernoviae* (Brasier et al., 2005) within the nursery trade network and the semi-natural environment in Great Britain. However, the study is largely generic and would need only parameter adjustment and possibly re-assessment of the trade network to apply to another organism.

P. ramorum is a newly described oomycete plant pathogen which is affecting both the semi-natural environment and the hardy nursery stock industry of various countries (Appiah, 2007; Hansen, 2008). It was first recorded in the UK in February 2002 in a nursery (Lane et al., 2003). In the semi-natural environment, the situation

* Corresponding author. Present address: CSIRO Entomology, GPO Box 1700, Canberra, ACT 2601, Australia.
E-mail address: tom.harwood@csiro.au (T.D. Harwood).

0304-3800/$ – see front matter © 2009 Elsevier B.V. All rights reserved.
doi:10.1016/j.ecolmodel.2009.08.014

is at present currently less severe in Europe than the USA, where *P. ramorum* is causing extensive tree and shrub mortality (Sudden Oak Death) in California and in one Oregon county (Maloney et al., 2005; Frankel, 2008; Hansen et al., 2008). In Europe, outbreaks in the landscape have so far been reported mainly in the UK. The pathogen is causing *Rhododendron* dieback (together with the death of some individual trees) in some historic gardens and woodlands (Brown and Brasier, 2007; Xu et al., 2009). In plant nurseries, the pathogen is responsible both in the USA and in Europe for a wide range of symptoms on several ornamental species (Parke et al., 2004; Neubauer et al., 2006; Kaminski and Wagner, 2008; Grünwald et al., 2008a; Hüberli et al., 2008; Moralejo et al., 2008).

P. kernoviae was discovered in Cornwall, England, during inspections for *P. ramorum* in October 2003 and has a similar host range and UK distribution (CABI, 2008) despite being only distantly related. Currently *P. kernoviae* has been found only in the UK and New Zealand. However, it also can spread on horticulturally traded plants, especially *Rhododendron*. *P. kernoviae* infections were recorded in 2008 on *Vaccinium myrtillus* in both woodland and heathland locations (Beales et al., 2009), and more recently *P. ramorum* infections on *V. myrtillus* have been found in moorland in Shropshire, England (CSL, 2009).

A key host for both species in the UK is *Rhododendron ponticum*, an important invasive species (Shaw, 1984; Dehnen-Schmutz et al., 2004) in a range of habitats including the woodland understorey. It also is widely planted in ornamental gardens. Whilst both *Phytophthora* species can infect and sporulate on *R. ponticum*, the rate of leaf loss is much greater for *P. kernoviae*, for which a greater proportion of cases are found in the semi-natural environment (Webber, 2008). *Vaccinium myrtillus* is an important sporulating host (CSL, 2006) because it is, widely distributed in woodland and heathland across the UK. *V. myrtillus* and *Calluna vulgaris* are key species in upland and lowland heath ecosystems, which are of particular conservation importance in the UK (Pakeman and Nolan, 2009). The problem is of particular concern since *Calluna vulgaris* also has shown some susceptibility to both pathogens in laboratory tests (CSL, 2006) and has been infected naturally in a nursery (Orlikowski and Szkuta, 2004). The heathland habitat therefore was included in the study both for its intrinsic interest and because large areas of potentially susceptible material are likely to affect the spread of both pathogens.

Models of the spread of *P. ramorum* in a landscape have been developed for California using various approaches, from risk assessment maps based on host presence (Meentemeyer et al., 2004) to environmental niche models (Kelly et al., 2007). There is a need for models which predict the spatial rate of spread across the landscape. Such models must include the spread of the pathogen through human activities such as the movement of infected plants in the horticultural trade (Jeger et al., 2007).

A multi-scale study in California has shown that *P. ramorum* outbreaks are more severe in regions of higher human population density, and also that locally tree mortality is more severe along woodland trails (Cushman and Meentemeyer, 2008). *P. ramorum* has been detected in several plant nurseries and retail outlets, and shipments of ornamental plants have spread the pathogen over long distances (Holdenrieder et al., 2004; Stokstad, 2004; Grünwald et al., 2008b; Mascheretti et al., 2008; Turner et al., 2008). However, the relative importance of different methods of spread and the consequences of different types of intervention are hard to judge intuitively. In this study, we aim to fill this knowledge gap by developing a simulation model incorporating important processes affecting the spread of *P. ramorum* and similar diseases across the UK. The simulation allows for both movement of diseased plants in a realistically modelled trade network and long-distance dispersal in the natural environment via other mechanisms such as airborne or water-borne inoculum movement.

2. Methods

2.1. Model architecture

The simulation model was developed and implemented as a standalone Windows® software application (Epidemic Simulator), using Borland® C++ Builder 5.

A realistic representation of the trade of susceptible plants in Great Britain, with simulated inspection and eradication regimes was set in a geographically explicit landscape of susceptible semi-natural material. Dispersal consequently could occur through the movement of whole infected plants through trade or through dispersal of infectious propagules by other means. Habitat was represented on a grid of 1-km grid squares covering mainland Great Britain. Within each grid square, separate nodes representing the area of coverage of woodland, heathland and horticulture summarised the spatial structure at smaller scales. All the nodes in the trade network where plants are physically present exist within the grid. Linkages between these sites via trade therefore provide dispersal shortcuts across the grid.

Interface between the grid-based landscape and trade network models was achieved by a definition of a common node type, where nodes could be either a grid cell of a particular habitat or a non-spatial node to represent junctions in the trade network (Fig. 1). The individual grower holdings (on the trade layer) then were linked to non-spatial marketing/distribution centres, which in turn were linked to non-spatial multiple retailers and then back to grid cells for the location of retail outlets. Non-spatial nodes do not have a physical spatial location, and cannot actually contain plants; instead they act as junctions in the network.

Accurate information on the spatial distribution of different susceptible species, the degree of pathogenicity and dispersal characteristics of *P. ramorum* and *P. kernoviae* still are not available for the UK. The simulation model was developed as a flexible generic framework to describe disease spread in Great Britain in systems with concurrent spread through trade and natural dispersal, both to allow adaptation to new information, and on a more fundamental level because specificity is better represented in parameterisation than the underlying model structure. A particular benefit of a generic model in this context is that it could easily be extended to simulate other *Phytophthora* species (e.g. Donahoo et al., 2006; Jung and Nechwatal, 2008; Jung and Burgess, 2009) of which an increasing number are being detected, and some of which pose new threats. The behaviour of the system then was examined over a range of parameterisations representing the current understanding of *P. ramorum* epidemiology, the spatial distribution of susceptible material, and the nursery-inspection process.

The key host species for both trade and local dispersal was assumed to be *R. ponticum*. Other *Rhododendron* species and sporulating ornamental genera such as *Viburnum* and *Pieris* were assumed to behave in the same way as *R. ponticum* and were added to the area of *R. ponticum*. Additionally, spread through heathland species was modelled. Both within and between these species, there undoubtedly will be variations in size, and consequently in the spore-producing capacity of an individual plant. Although individual species will differ in susceptibility, spore production, and other features, it was not considered feasible to make or parameterise a model representing individually all species which might act as hosts. Variation between species could be captured within the two notions of the susceptible area and standard plant size: a more susceptible plant would represent a relatively larger contribution than its area justifies, and vice versa.

The epidemiological model used was based on units representing plants with a ground area of 0.1 m^2 (maximum density $\Delta = 100{,}000\ ha^{-1}$). This represents a single pot plant as moved between nurseries. Larger plants and consignments were treated

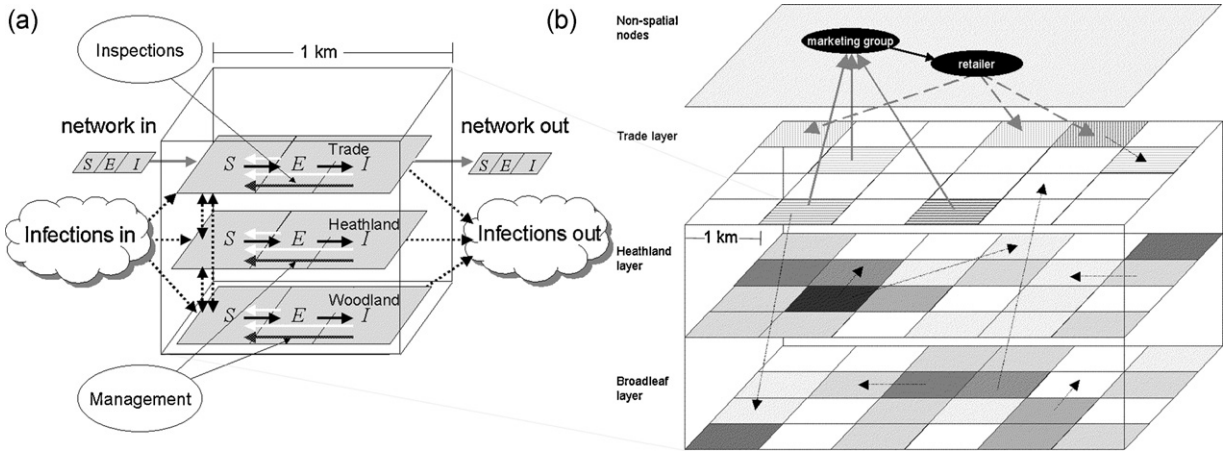

Fig. 1. Conceptual diagram of the model. (a) Structure of a single 1-km grid cell, showing the three GIS layers of population nodes, each with an SEIS model running inside. Movements of individuals between compartments are shown, with lines for ongoing infection (black), mortality and recruitment (white), and management replacement of infected plants (patterned). Trade network movements are shown in grey, and infection between layers and other cells with dotted arrows. (b) A 6 by 4-km cross section through the spatial grid, showing the three grid layers with a superimposed trade network represented by explicit directional trade links (grey) between the supplier nodes (horizontal hatching), marketing groups, multiple retailers and retail sites (vertical hatching). Dispersal of infected spores between nodes both within and between layers is shown by dotted arrows.

as assemblages of individuals. Assuming that spore production is proportional to leaf mass, which is itself proportional to leaf volume, and that leaf volume can be represented by a hemisphere, a 2-m-high *Rhododendron* plant is equivalent to 1409 individuals of ground area 0.1 m^2 (height 178 mm.). The 2-m-high plant would have 11.2 times as much leaf material per unit ground area as the standard individual. This could be expressed as an increase in the effective area occupied; however, areal estimate of species occupancy (see Data below) were not accurate enough to warrant detailed species-specific scaling.

2.2. Data

Geographical information was used at a 1-km^2 resolution for the whole of Great Britain, based on the National Grid. Habitat data was taken from the CS2000 survey describing the distribution of Broad Habitat Types at this resolution (Broadleaved, mixed and yew woodland stock 1998, Dwarf shrub heath stock 1998, units ha km^{-2}). Whilst better quality information on the distribution of susceptible plants is available on a local scale, the national coverage provides a consistent background.

The distribution of hardy nursery stock was estimated from the 2003 AgCensus data for England and for Wales, which subsequently was modified using data from the 2006 HortWeek Suppliers Guide to represent the distribution of sites growing susceptible stock. The 2003 AgCensus data for England and Wales provided 2-km resolution areal-estimates of Hardy Nursery Stock. For Wales, the class was a broader Horticulture definition which must be expected to contain other sectors. For Scotland, no direct spatial data was available, so the baseline spatial arrangement was generated by assuming that the spatial distribution of sites was similar to that of England and overlaying a subset of the English AgCensus data over Scotland. Subsequent sampling as described below ensured that the total area was appropriate. For each 2-km grid square, a single 1-km grid square was allocated randomly to contain the site.

The locations of marketing groups for growers specialising in susceptible stock (*Rhododendron*, Heathers) were recorded from the 2006 HortWeek Suppliers Guide, and sizes either directly recorded (59%) or estimated as the mean size of recorded susceptible holdings (41%). The sizes of all holdings were recorded as areas. Where only plant numbers were available, the growing area was estimated from the mean ratio of area to plant numbers from those sites for which both were available. For each marketing group, individual nursery holdings (1-km^2) were allocated randomly according to a radial search algorithm giving preference to sites near the marketing group. Each marketing group took turns selecting nearby occupied grid squares until it had the specified total size. For each marketing group, the starting square was randomly selected within the 8-cell Moore neighbourhood, and the direction (clockwise or anticlockwise) similarly randomly allocated. At each step, searching continued until an occupied grid square which had not been allocated to another group was located. The search continued in the specified direction, moving radially out into the next ring of squares when a circuit had been completed. All holdings not allocated were assumed not to trade in susceptible stock and were not included in the model.

The locations of retailers were derived from the inspection records of Plant Health and Seeds Inspectorate (PHSI). All were allocated the same ground area, by equally dividing the total output of the nurseries. Nodes representing these sites were then added to the grid.

2.3. Spatial SEIS model

A spatial implementation of an SEIS model (Madden et al., 2007) was developed to model pathogen dynamics and spatial dispersal. SEIS models have three categories of plants: susceptible (S), Exposed (E), and Infectious (I), where Exposed individuals are those which have been infected but are not yet producing infectious propagules. The total constant population size N is defined as $S+E+I$. In both trade and natural settings, dead plants will be replaced rapidly by new ones. The removal rate of plants $\mu(S+I+E)$ therefore was assumed to be matched by an equal input of susceptible plants so as to keep population density constant. This means that the model cannot capture long-term species replacement processes in trade or the landscape, but this was not our aim. Within each population (each layer in each grid cell) the SEIS model was run as a deterministic model. A rectangular integration of the model on a daily time-step was used. The numbers of individuals in each category is represented by S, E and I respectively. The rate of infection of new individuals is given by βSI, where β is the number of infections that a single infectious individual would make per susceptible individual per day in ideal conditions. Exposed individuals become infectious at a rate kE, where k is the reciprocal of the latent

period in days. Recovery or immunity is not documented. Increased mortality due to infection was considered negligible over the short time spans of the simulations, except where destruction following a positive inspection was simulated. A blanket mortality rate μ (representing replacement by new susceptible individuals) was applied to all individuals. Thus:

$$\frac{dS}{dt} = -\beta SI + \mu E + \mu I \quad (1)$$

$$\frac{dE}{dt} = \beta SI - kE - \mu E \quad (2)$$

$$\frac{dI}{dt} = kE - \mu I \quad (3)$$

This basic model was implemented on a 1-km spatial grid of dimensions 700 × 1300-km. The dispersal kernel was an inverse-square power-law with median dispersal of 2-m. Dispersal bearing was assumed to be uniform. To save calculation time whilst retaining accuracy, the resulting dispersal across the grid was pre-calculated as follows. The function was integrated from an array of point sources on a 20-m sub-grid within the source cell to an equivalent array of point sinks within all 1-km grid cells within an arena of radius 50-km from the source. No viable dispersal beyond 50-km was assumed. The probability of inoculation within the source cell (ψ) relative to the rest of the arena ($1-\psi$) was stored, and the remaining results comprising a cell-specific probabilities (p^{cell}) normalised such that $\Sigma p^{cell} = 1$ for all cells outside the source, sorted into descending order. For a single dispersal event outside the source cell, a destination cell then was selected from the normalised p^{cell} values using a single uniform random number. If R represents the daily number of infections per infectious individual that would occur within the entire 50-km radius arena entirely populated with susceptible individuals at a density Δ, the maximum daily number of infections within the source cell is $R\psi$. Since this also may be defined as $\beta\Delta$, we can derive β for a given dispersal kernel as

$$\beta = \frac{R\psi}{\Delta} \quad (4)$$

Three habitat layers (broadleaved woodland, heathland and the horticultural trade) were defined, with an area A^{cell}_{layer} in each grid cell (where $\sum_{layer} A^{cell}_{layer} \leq 1\,\mathrm{km}^2$). Of this area, only a habitat-specific proportion δ_{layer} is susceptible, giving an initial number of susceptible plants per layer in each cell of

$$S^{cell}_{layer} = \Delta \delta_{layer} A^{cell}_{layer} \quad (5)$$

Outside the source cell, infection was assumed to be independent of the source habitat; so, the expected number of new infections in a given cell and layer is

$$e^{cell}_{layer} = \left(1-\psi\right) p^{cell} \beta S^{cell}_{layer} I \quad (6)$$

Within the source cell, the probability of habitat of the same type being in proximity to the source was assumed to be greater than that of other habitat layers. A weighting w was applied to allow for this by modifying the effective areas of habitat, with the number of other layers $n_{other\ layers}$ set at 2 for the 3 layer model.

$$S^{w}_{source\ layer} = \frac{wS_{source\ layer}}{w + n_{other\ layers}} \quad (7)$$

$$S^{w}_{other\ layer} = \frac{S_{other\ layer}}{w + n_{other\ layers}}. \quad (8)$$

The expected number of new infections within a given layer in the source cell then is

$$e^{cell}_{layer} = \beta S^{wcell}_{layer} I \quad (9)$$

This model then was implemented in a stochastic form to represent discrete dispersal events and the formation of new disease foci, which are random events due to spore movement through a chaotic medium. The expected number of new infections for a given destination was used to generate an actual whole number from a Poisson distribution. Whilst there are computational costs to such an approach, the time cost was greatly reduced in comparison with conventional methods by the dispersal algorithm described above. Efficient computation was achieved by generating explicit events for all cells within 10-km of the source, but generating a single event for the remaining area. When this was non-zero it was allocated to a specific cell in the 10–50-km zone according to the distribution kernel.

2.4. Realistic trade network model

A simulated trade network was used to connect nursery and retailer nodes and to allow the movement of plant units between nodes to simulate trade. The positions and links of the individual grower holdings, and their umbrella groups, multiple and single retailers were determined as described in Section 2.2. Fig. 1 shows how this network was defined relative to the spatial grid. Trade occurred between grower holdings and retailers/gardens according to specific links. Fixed links were established from the data to connect individual holdings with distribution hubs and to connect multiple retailers to their individual outlets. The nature of the links between suppliers and retailers could not be determined due to commercial sensitivity of the information and was generated stochastically for each model run (e.g. black arrow showing the link between non-spatial nodes in Fig. 1) rather than arbitrarily define a structure. Annual turnover of produce was assumed to be constant for all holdings and was set at 0.6 of the total population size. The turnover for each holding (0.6N) was used to generate the total throughput of the system, which was balanced by the sales from the retailers. Retailers then randomly selected suppliers (including distribution hubs) until their annual quota was reached. This ensured balanced trading. In practice, the fixed elements of the network resulted in a consistent network type, which was scale-free and small-world (Pautasso and Jeger, 2008). Whilst the network structure allowed for large-scale marketing and retail activities, it also allowed a single-grower holding to supply directly to a retailer or any other combination of links.

Trading was carried out explicitly by the movement of a randomly selected subset of the plants on site. Trading occurs every day throughout a trading period specific to each source site. The daily number of plants traded was given as 0.6N/number of days trading. The trading period for each site was set by its distribution centre, to which a randomly generated period of 1–60 days between 1st March and 31st August was allocated. Since population size was constant for all sites, a node exporting plants was topped up with fresh susceptible individuals, and plants arriving at a node replaced a random sample of the existing population. Plants travelling through a non-spatial node representing the transport process were held in transit for one day for each non-spatial node they pass through. During this period they were not infectious and could not develop new infections.

2.5. Inspections and management

Management strategies for control of the epidemic in the nursery trade and in the semi-natural environment were considered separately.

All trade premises were added to an inspection list, and a random start day for inspections allocated to each. Inspections then occurred at a fixed basic rate (e.g. one every 180 days) until any infection was detected. At this point, re-inspection was scheduled

Table 1
Parameter values used for simulations.

Parameter	Range of values
Rate at which new infections produced by single infected individual in healthy population (R)	0.5, 1/day
Latent period (k)	10 day
Intrinsic replacement rate (μ)	0.0005/day
Equivalent host density (Δ)	100,000/ha
Habitat transfer coefficient (w)	3
Proportion of broadleaf area occupied by susceptibles ($\delta^{broadleaf}$)	0.001 or 0.01
Proportion of heath area occupied by susceptibles ($\delta^{heathland}$)	0.001 or 0.01
Proportion of nursery area occupied by susceptibles (δ^{trade})	0.1
Initial infection size	10 plants
Trade turnover	0.6/year
Interval between inspections	180 day
Re-inspection interval for positives in days	90 day
Efficiency of removal on positive inspection (p_d)	80%

at a shorter interval (e.g. 90 days), eradication measures applied at a certain efficiency, and trace-forward and/or trace-back measures (inspection of all connected nodes) applied where specified. This accurately represented the official inspection policy within the PHSI in 2007/2008. Inspection efficiency was modelled both at inspections and during the eradication process. At an infected site, there was a probability p_d that the infection was detected, and a Boolean throw was made to determine the results of an inspection. If the infection was detected, it was assumed that a thorough inspection of all plants was made, and p_d of infected plants were removed.

Semi-natural environment surveys and subsequent management were controlled by a habitat-specific parameter specifying annual probability of detection and efficiency of control. Positive inspections resulted in the node being added to a regular inspection list (using the standard nursery-inspection rate) and control at the specified efficiency. Practical field management of these diseases was variable, but this is a reasonable summary model. In large-scale epidemics, the cost of management of the semi-natural environment would become prohibitive, and this model becomes overly optimistic. This option was not used for the main simulations, allowing the effects of the nursery inspections to be assessed in a simpler environment.

2.6. Software and model testing

Extensive testing of the software was carried out on Windows 98SE, 2000 and XP on four separate PCs prior to the simulation study. Robustness of the model to input parameters was tested by examining model behaviour over a range of extreme values for all parameters and functions. Subsequently, the sensitivity of the model in the region of the values given in Table 1 was examined for reasonable behaviour. For each parameterisation 5 simulations were conducted. No unexpected responses were observed. This did not represent a full sensitivity analysis.

Direct validation of the model against historical data was not possible. We have only one historical record, with unknown introduction points, unquantified management intervention, and an incomplete record of its spatial distribution. In addition to this, stochastic processes, especially those of dispersal can result in a wide range (log normally distributed) of potential outcomes for a given set of parameters. Since this is true for both the real and model worlds, it is only possible to say that observed behaviour falls within the range of model outcomes. To circumvent this problem, results were obtained over a range of parameter values to derive qualitative and general conclusions for *Phytophthora* like systems with this spatial structure.

For a given set of model parameters and dispersal functions, the lognormal distribution of results was reasonably consistently described by a sample of 30 or more simulations. Consequently a standard replicate number of 50 was used for the simulation experiments.

2.7. Simulation experiments: effects of trade, linkage of commerce and wild vegetation, and inspections

Simulations were carried out to examine the behaviour of the model system under a range of different scenarios. Due to uncertainty as to the nature of infection by *P. ramorum* and *P. kernoviae* under British conditions the aim was to explore the model under settings appropriate for a range of plant pathogens, rather than attempt to simulate species-specific predictions. Table 1 gives parameter values used to run the simulations.

Series 1: Effect of trade, inspections and linkage to natural environment. A series of simulations was run, representing an experiment varying the epidemic pressure with or without disease spread in commercial trade, and with or without inspections-with-eradication at a high and low contact rate and with high and low linkage between natural vegetation and horticultural trade, to give a $2 \times 2 \times 2 \times 2$ factorial. Each simulation was run for 10 years. Fifty replicate simulations were made at each set of parameter values at each of 10 locations spread over England (8000 simulations), allowing formal statistical tests to be made and, where these were significant, the relative importance of different influences to be judged. The resultant epidemic sizes were transformed to logarithms before analysis. The residuals were skewed because of the distribution of epidemic sizes described below, but the results are easiest to interpret on this scale as effects correspond to proportionate changes.

Series 2: repeated introductions and enhanced inspection regimes. The effect of regular randomly located introductions into the trade network was investigated to simulate the effect of failure to detect infection at the time of import. Introductions once, twice, and four times a year were used in combination with normal (180:90) and double (90:45) inspection: re-inspection intervals at the higher infection rate ($R = 1$). The epidemic in the semi-natural environment in terms of woodland and heathland was not modelled, allowing the nursery inspection system to be assessed in isolation. Five hundred 10-year simulations (3000 simulations in total) were carried out for each combination of inspection parameters, using default values in Table 1.

3. Results

3.1. Distribution of epidemic size

Because of the stochastic nature of the simulation, replicate epidemics started at the same location and with the same parameters evolved differently. The probability distribution of log epidemic size had three peaks: cases where the epidemic failed to establish; moderate sizes, locally often saturated but failing to spread; and very large epidemics, where infection reached a large hub (Fig. 2).

3.2. Series 1: effect of trade, inspections, and linkage to natural environment

The most important factor determining total final epidemic size was R (Table 2), although the two values compared are of course arbitrary. The next most influential factor was the strength of the linkage between the natural environment and trade infections; without infections arising in nature, the final population size was smaller. Removing the commercial trade in plants also greatly

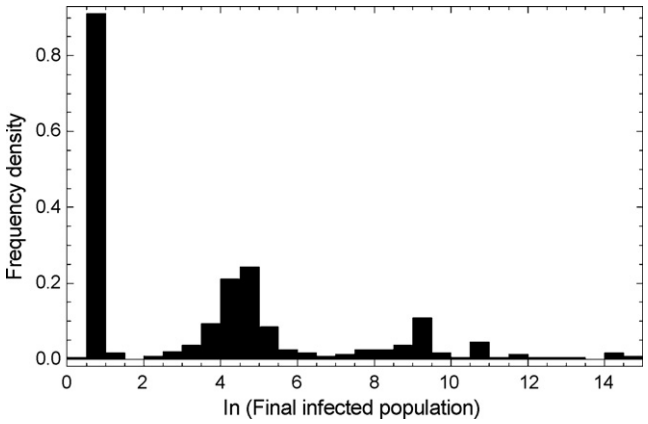

Fig. 2. Experiments, Series 1. Histogram of the natural logarithm of the infected host population size after 10 years; 500 replicates; $R = 1$/day; commercial movement enabled; inspections with removal enabled; natural spread parameter 0.001 (low); epidemic start at UK grid ref SJ326768. Other parameters at default (Table 1).

reduced the final epidemic size. The effect of the introduction location differed substantially, depending on how they were connected to the trade network and susceptible vegetation. Inspections-with-eradication had the next largest effect, but this interacted strongly with the strength of linkage to the natural environment: with strong links the effect of inspections with commercial trade enabled was weakened substantially, from −0.98 to −0.23 (averaged over R), equivalent to 63% and 22% reductions, respectively. Without commercial trade in susceptible species, the effect of inspections was somewhat greater with strong natural linkages and much greater with weak natural linkages.

3.3. Series 2: repeated introductions and enhanced inspection regimes

Repeated introductions at random locations led to larger final infected population sizes after 10 years, as expected (Fig. 3). The increase was closely linear with the number of introductions (linear contrast SS 1022.6, $F_{1,2994} = 580$, $P < 0.001$, quadratic deviation SS 3.3, $F_{1,2994} = 24$, $P > 0.001$) at about 60% per extra introduction (0.47 ln units). Increasing the stringency of the inspection regime reduced the severity by 0.23 ln units, equivalent to about a 21% improvement in control ($F_{1,2994} = 23.6$, $P > 0.001$) with a small but significant interaction with the frequency of introduction ($F_{1,2994} = 9.0$, $P > 0.001$), because the increase due to extra introductions was reduced slightly with the more stringent inspection regime.

4. Discussion

The model demonstrates a useful framework in which to incorporate epidemic processes which are naturally conceptualised as a network - trade, with ones conceptualised as a continuous spatial model – the natural environment. The model is reasonably fast, taking a few days to run the example experiments presented, but captures most of the features controlling epidemics of this type. The use of a network and spatial structure which are realistic allows us to ask questions which are relevant to policy.

However, the extent to which a model can represent a real system is always constrained by the available data. In this case, the structure of the trade network in England and Wales may be regarded as adequately representing the spatial locations and size structure of the industry. In Scotland, where no data was available on the locations of actual holdings the spatial distribution of trade sites will be less representative of the real situation; however, this is compensated by the relatively few susceptible trade sites in Scotland. Since these were allowed to select holdings in close proximity, a reasonable degree of confidence in the large-scale spatial distribution is appropriate. Key links between marketing groups and distribution hubs had to be generated randomly, once per run. This was found to have little effect on the properties of the network in isolation, but did affect the spatial organisation of the network in relation to the underlying semi-natural habitat. The results of replicate simulations ensure that this uncertainty is represented in the statistical analysis of results. Consultation with working inspectors suggests that the processes and parameter values used in the model runs represent the inspection process effectively.

The actual distribution of susceptible species is poorly understood. By assuming an even distribution of susceptible species within the habitats in which they occur, it is likely that the modelled species distributions are less heterogeneous than in the real world. Greater heterogeneity might be expected to reduce the degree of contact between susceptible elements of the landscape; this is incorporated at present in the parameter w. However, higher local concentrations of infection could build up, resulting in a greater

Table 2
Analysis of variance of ln(epidemic size) after 10 years from a single introduction.

Source of variation	d.f.	Total size		Heath		Woodland	
		m.s.	Effect or mean	m.s.	Effect or mean	m.s.	Effect or mean
Grand mean			6.02		1.48		3.14
Start location	9	2691.5	+2.59	405.9	+1.01	922.3	+1.51
R high	1	26503.7	+3.64 ± 0.08	9519.3	+2.18 ± 0.06	22101.2	+3.32 ± 0.06
Commerce off	1	5125.2	−1.60	2516.6	−1.12	2216.0	−1.05
Inspections off	1	2104.4	+1.03	300.7	+0.39	757.0	+0.62
Nature linkage low	1	6896.2	−1.86	7609.6	−1.95	22986.9	−3.39
R.commerce	1	389.3	−0.88 ± 0.16	1009.2	−1.42 ± 0.14	482.0	−0.98 ± 0.14
R.inspections	1	147.2	+0.54	206.3	+0.64	320.6	+0.80
Commerce.insp	1	350.2	−0.84	99.7	−0.45	143.1	−0.54
R.nat	1	803.9	−1.27	3451.9	−2.63	3027.0	−2.46
Commerce.nat	1	48.7	−0.31	502.2	1.00	72.7	+0.38
Insp.nat	1	1296.2	+1.61	67.7	0.37	391.7	+0.88
R.commerce.insp	1	$18.3^{0.05}$	−0.38 ± 0.32	57.3	−0.68 ± 0.28	43.7	−0.59 ± 0.28
R.commerce.nat	1	$14.9^{0.05}$	−0.34	71.8	+0.76	6.2	-0.22^{ns}
R.insp.nat	1	76.2	+0.78	57.2	+0.68	263.4	1.45
Commerce.insp.nat	1	342.1	−1.65	67.4	−0.73	157.6	−1.12
R.commerce.insp.nat	1	$14.4^{0.05}$	−0.68 ± 0.65	55.0	−1.33 ± 0.54	61.6	−1.40 ± 0.55
Residual		3.28		2.30		2.37	

Fifty replicate epidemics were simulated for each combination of parameters and starting location. Unless specified, all effects are significant at $P < 0.001$; the mean square (m.s.) shows the relative contribution to variation in the results. ^{ns} Not significant at $P = 0.05$. $^{0.05}$ Significant only at $P = 0.05$.

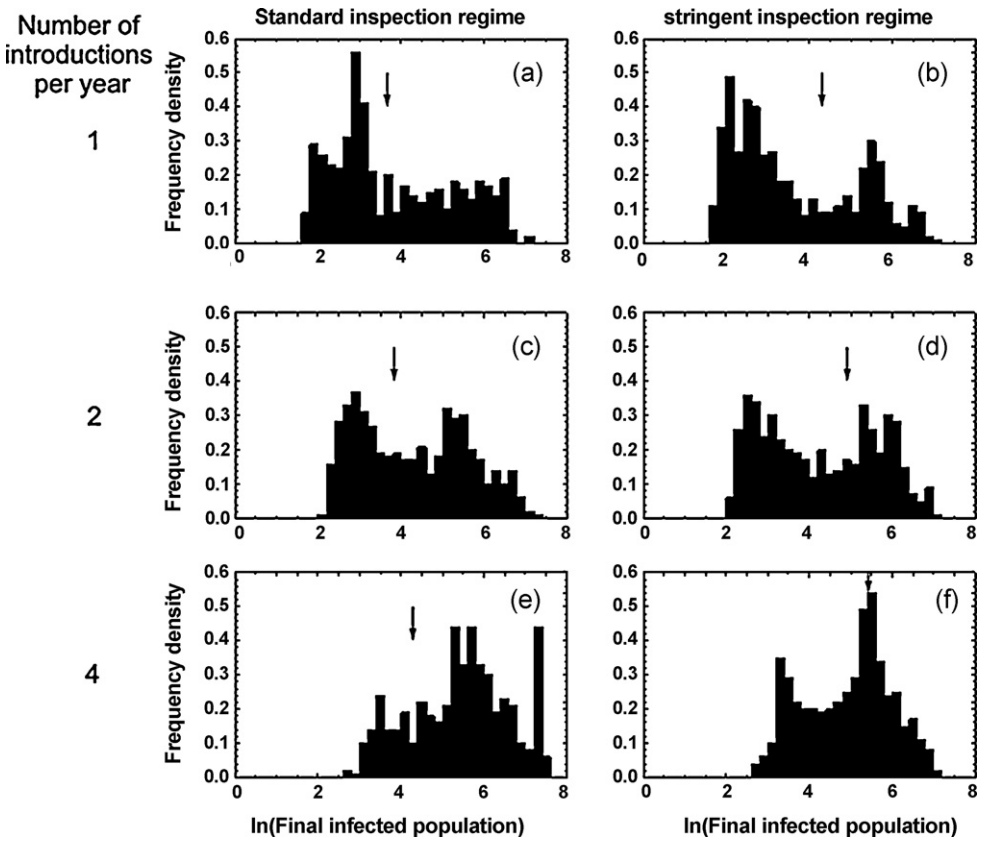

Fig. 3. Experiments, Series 2. Effect of standard and stringent inspection rates with 1, 2 or 4 introductions per year to random locations. The arrows indicate the mean in each panel. (a, c and e) Standard inspection regime; (b, d and f) stringent inspection regime; (a and b) 1 introduction per year; (c and d) 2 introductions per year; (e and f) 4 introductions per year. Histogram of the natural logarithm of the infected host population size after 10 years; 500 replicates; $R = 1$/day; commercial movement enabled; inspections with removal enabled; natural spread parameter 0.001 (low). Other parameters at default (Table 1).

probability of dispersal to any distance, but with the effect of decreasing w as epidemics intensify. The model therefore is somewhat conservative in estimating the process of spread to the natural environment. Previous studies have shown the importance of landscape heterogeneity in the spread and establishment of *P. ramorum* in California (Condeso and Meentemeyer, 2007; Kelly et al., 2008; Meentemeyer et al., 2008), and our model confirms this.

By combining two model families, the model is able to treat all the relevant aspects of the system. *P. ramorum* and *P. kernoviae* are not unique in their dispersal through both natural and human mediated means, which is common to many important pests and diseases. In these cases, modelling approaches ignoring either route of transmission are likely to have limited scale or practical application.

The modelling experiments in Series 1 demonstrate the importance of the semi-natural environment in sustaining *P. ramorum*/*P. kernoviae*-like epidemics. It is inappropriate to view the trade network as a truly independent system. Once infections become established in the semi-natural environment, management becomes reactive rather than proactive due to the size of the areas involved. Whilst the extent of infection between the two environments is unclear, there is some evidence for cross infection (Xu et al., 2009). Given the capacity of the semi-natural environment to sustain an epidemic in the face of efficient management, a holistic approach is thus required. However it should be noted that molecular work has shown that the landscape and nursery epidemics remain largely independent in the California epidemic (Mascheretti et al., 2008).

The trade epidemic seems to be controlled at the present level of intervention (Xu et al., 2009), and the modelling supports this, provided the semi-natural environment does not become infected. However, both series show that the effect of control declines with increasing disease pressure. Series 2 show that at a high level of new introductions, it is not possible to control epidemic spread, even with the most stringent of reasonable inspection regimes. This is largely due to the onward movement and dispersal of plants in the period between inspections.

Long-distance movement of plants will result inevitably in some long-distance transfer of diseased material, either via the trade or via amenity horticulture. If the epidemic becomes established in countries with strong trade links to the UK, any movement is likely to confound local attempts to control the epidemic.

Choice of dispersal kernels is critical to the behaviour of models of this type. Here we used negative-square power-law model of median dispersal distance 2 m. The dispersal kernel of either pathogen has not been measured empirically, although it is generally considered to be concentrated close to the source due to splash dispersal (Davidson et al., 2005; Garbelotto, 2008) but with occasional long-distance aerial dispersal (Rizzo et al., 2005). This would indicate fat-tailed distribution, and a median distance of 2-m is reasonable for point source of small aerial spores or pollen (Shaw et al., 2006). No specific treatment is made of the transport of infected litter or of mud on boots. These and similar unquantified dispersal pathways were assumed to be summarised by the dispersal kernel. Clearly this will fail to represent a visitor carrying mud on boots further than the modelled maximum natural-dispersal distance of 50-km.

Linking a network model to the underlying landscape has implications for the nature of connectivity between nodes in the network. Geographically close nodes may well infect one another, even if not explicitly connected via the network (in this case no trade), via disease spread across the landscape. It may be argued

that all the nodes in the network are connected to some degree. If explicitly modelling both the network and the landscape, this is not an issue; however, if one is attempting to describe a network which can be characterised in this way using conventional network theory, it may be difficult to choose an appropriate cut-off point.

Where a pathogen has the potential to become established in both the network and the wider environment, the appropriateness of model assumptions can be expected to change according to the relative levels of infection in the different sub-systems. Thus, if infections largely are restricted to the nursery trade, a strict network approach could be justified. Once a disease becomes established outside the network, both systems become important. At a later stage, when the disease is firmly established, low-level long-distance spread via networks may become irrelevant.

5. Conclusions

Our model suggests that, provided the *Phytophthora* epidemics are limited to the nursery trade, inspections usually can control single epidemics. Restricting trade in particularly susceptible species would reduce spread of the disease greatly, but would be unpalatable to the industry. With continued trade there is a continuing likelihood of escape to the natural environment. Once an epidemic becomes established in the semi-natural environment, the impact of inspections is greatly reduced. The difficulty of total eradication of the disease from known outbreak sites (e.g. Prospero et al., 2007; Hansen, 2008) and the possibility of re-infection gradually will reduce the number of nursery premises available for the cultivation of *P. ramorum*/*P. kernoviae* susceptible-plants. The effect on the semi-natural environment depends on factors such as the implementation of effective *R. ponticum* control.

The structure of national and international horticultural trade networks still is poorly documented. Models of disease spread in complex networks suggest that in the case of heterogeneity in the contact structure of trade players, control should be specifically targeted towards super-connected nodes (Jeger et al., 2007; Pautasso and Jeger, 2008). Again, this is commercially unpalatable, because it often is economically more efficient to use major concentration depots to receive and distribute plants (and other commodities); these are automatically super-connected nodes. Unfortunately, the implications of network theory for diminishing the threat of undesired introductions of plant pathogens still are not considered in the context of the bio-security threat posed by the increasing international horticultural trade (Brasier, 2008).

The software developed can be used also for predicting the spread of other emerging plant diseases and, with some adjustments, of invasive plant species introduced through the ornamental trade (Levine and D'Antonio, 2003; Baker et al., 2005; Dehnen-Schmutz et al., 2007a,b). Further research should study the spread of *P. ramorum* and other emerging plant diseases over the long-term so as to assess, among other things, the importance of climate change for such processes (Coakley, 1995; Chakraborty et al., 2000; Jeger and Pautasso, 2008).

Acknowledgements

Many thanks to R. Baker, C. Brasier, O. Holdenrieder, A. Inman, J. Turner, and J. Webber for data, insights and discussions. This project was funded by the Department for Environment, Food and Rural Affairs, UK.

References

Appiah, A.A., 2007. A global review of the threat of *Phytophthora ramorum* infections and the prospects for effective control strategies. CAB Rev. 2 (No. 037).

Baker, R.H.A., Cannon, R., Bartlett, P., Barker, I., 2005. Novel strategies for assessing and managing the risks posed by invasive alien species to global crop production and biodiversity. Ann. Appl. Biol. 146, 177–191.

Beales, P.A., Giltrap, P.G., Payne, A., Ingram, N., 2009. A new threat to UK heathland from *Phytophthora kernoviae* on *Vaccinium myrtillus* in the wild. Plant Pathol. 58, 393–1393.

Brasier, C.M., 2008. The biosecurity threat to the UK and global environment from international trade in plants. Plant Pathol. 57, 792–808.

Brasier, C.M., Beales, P.A., Kirk, S.A., Denman, S., Rose, J., 2005. *Phytophthora kernoviae* sp.nov., and invasive pathogen causing bleeding stem lesions on forest trees and foliar necrosis of ornamentals in the UK. Mycol. Res. 109 (8), 853–859.

Brown, A.V., Brasier, C.M., 2007. Colonization of tree xylem by *Phytophthora ramorum*, *P. kernoviae* and other *Phytophthora* species. Plant Pathol. 56 (2), 227–241.

Centre for Agriculture Bioscience International, 2008. *Phytophthora kernoviae* (Distribution map). Distrib. Maps Plant Dis. 1023.

Chakraborty, S., Tiedemann, A.V., Teng, P.S., 2000. Climate change: potential impact on plant diseases. Environ. Pollut. 108, 317–326.

Coakley, S.M., 1995. Biospheric change—will it matter in plant pathology. Can. J. Plant Pathol. 17 (2), 147–153.

Condeso, T.E., Meentemeyer, R.K., 2007. Effects of landscape heterogeneity on the emerging forest disease sudden oak death. J. Ecol. 95 (2), 364–375.

Central Science Laboratory, 2006. Final Summary Report: Determining the Susceptibility of Key/Dominant UK Heathland Species to *Phytophthora kernoviae*, available online at: http://www.defra.gov.uk/planth/kernovii/kernrep.pdf. Accessed 5th April 2009.

Central Science Laboratory, 2009. CSL list of natural hosts for *Phytophthora ramorum* with symptom and location. Updated 26/02/2009, available online at http://www.defra.gov.uk/planth/newsitems/suscept.pdf. Accessed 2nd April 2009.

Cushman, J.H., Meentemeyer, R.K., 2008. Multi-scale patterns of human activity and the incidence of an exotic forest pathogen. J. Ecol. 96 (4), 766–776.

Davidson, J.M., Wickland, A.C., Patterson, H.A., Falk, K.R., Rizzo, D.M., 2005. Transmission of *Phytophthora ramorum* in mixed-evergreen forest in California. Phytopathology 95, 587–596.

Dehnen-Schmutz, K., Perrings, C., Williamson, M., 2004. Controlling *Rhododendron ponticum* in the British Isles: an economic analysis. J. Environ. Man. 70, 323–332.

Dehnen-Schmutz, K., Touza, J., Perrings, C., Williamson, M., 2007a. The horticultural trade and ornamental plant invasions in Britain. Conserv. Biol. 21 (1), 224–231.

Dehnen-Schmutz, K., Touza, J., Perrings, C., Williamson, M., 2007b. A century of the ornamental plant trade and its impact on invasion success. Divers. Distrib. 13 (5), 527–534.

Donahoo, R., Blomquist, C.L., Thomas, S.L., Moulton, J.K., Cooke, D.E.L., Lamour, K.H., 2006. *Phytophthora foliorum* sp nov., a new species causing leaf blight of azalea. Mycol. Res. 110, 1309–1322.

Frankel, S.J., 2008. Sudden oak death and *Phytophthora ramorum* in the USA: a management challenge. Aust. Plant Pathol. 37 (1), 19–25.

Garbelotto, M., 2008. Molecular analysis to study invasions by forest pathogens: examples from Mediterranean ecosystems. Phytopathol. Mediterr. 47, 183–203.

Grünwald, N.J., Goss, E.M., Press, C.M., 2008a. *Phytophthora ramorum*: a pathogen with a remarkably wide host range causing sudden oak death on oaks and ramorum blight on woody ornamentals. Mol. Plant Pathol. 9, 729–740.

Grünwald, N.J., Goss, E.M., Larsen, M.M., Press, C.M., McDonald, V.T., Blomquist, C.L., Thomas, S.L., 2008b. First report of the European lineage of *Phytophthora ramorum* on *Viburnum* and *Osmanthus* spp. in a California nursery. Plant Dis. 92 (2), 314.

Hansen, E.M., 2008. Alien forest pathogens: *Phytophthora* species are changing world forests. Boreal Environ. Res. 13, 33–41.

Hansen, E.M., Kanaskie, A., Prospero, S., McWilliams, M., Goheen, E.M., Osterbauer, N., Reeser, P., Sutton, W., 2008. Epidemiology of *Phytophthora ramorum* in Oregon tanoak forests. Can. J. For. Res. 38 (5), 1133–1143.

Holdenrieder, O., Pautasso, M., Weisberg, P.J., Lonsdale, D., 2004. Tree diseases and landscape processes: the challenge of landscape pathology. Trends Ecol. Evol. 19, 446–452.

Hüberli, D., Lutzy, B., Voss, B., Calver, M., Ormsby, M., Garbelotto, M., 2008. Susceptibility of New Zealand flora to *Phytophthora ramorum* and pathogen sporulation potential: an approach based on the precautionary principle. Aust. Plant Pathol. 37, 615–625.

Jeger, M.J., Pautasso, M., 2008. Plant disease and global change—the importance of long-term data sets. New Phytol. 177, 8–11.

Jeger, M.J., Pautasso, M., Holdenrieder, O., Shaw, M.W., 2007. Modelling disease spread and control in networks: implications for plant sciences. New Phytol. 174, 179–197.

Jung, T., Burgess, T.I., 2009. Re-evaluation of *Phytophthora citricola* isolates from multiple woody hosts in Europe and North America reveals a new species, *Phytophthora plurivora* sp. nov. Persoonia 22, 95–110.

Jung, T., Nechwatal, J., 2008. *Phytophthora gallica* sp nov., a new species from rhizosphere soil of declining oak and reed stands in France and Germany. Mycol. Res. 112, 1195–1205.

Kaminski, K., Wagner, S., 2008. In vitro inoculation studies for estimating the susceptibility of ornamental plants to *Phytophthora ramorum*. J. Phytopathol. 156, 480–486.

Kelly, M., Guo, Q., Liu, D., Shaari, D., 2007. Modeling the risk for a new invasive forest disease in the United States: an evaluation of five environmental niche models. Comput. Environ. Urban 31 (6), 689–710.

Kelly, M., Liu, D.S., McPherson, B., Wood, D., Standiford, R., 2008. Spatial pattern dynamics of oak mortality and associated disease symptoms in a Califor-

nia hardwood forest affected by sudden oak death. J. For. Res. 13 (5), 312–319.
Lane, C.R., Beales, P.A., Hughes, K.J.D., Griffin, R.L., Munro, D., Brasier, C.M., Webber, J.F., 2003. First outbreak of *Phytophthora ramorum* in England on *Viburnum tinus*. Plant Pathol. 52 (3), 414–1414.
Levine, J.M., D'Antonio, C.M., 2003. Forecasting biological invasions with increasing international trade. Conserv. Biol. 17, 322–326.
Madden, L.V., Hughes, G., Van Den Bosch, F., 2007. The Study of Plant Disease Epidemics. APS Press, St. Paul, MN.
Maloney, P.E., Lynch, S.C., Kane, S.F., Jensen, C.E., Rizzo, D.M., 2005. Establishment of an emerging generalist pathogen in redwood forest communities. J. Ecol. 93 (5), 899–905.
Mascheretti, S., Croucher, P.J.P., Vettraino, A., Prospero, S., Garbelotto, M., 2008. Reconstruction of the Sudden Oak Death epidemic in California through microsatellite analysis of the pathogen *Phytophthora ramorum*. Mol. Ecol. 17 (11), 2755–2768.
Meentemeyer, R., Rizzo, D., Mark, W., Lotz, E., 2004. Mapping the risk of establishment and spread of sudden oak death in California. For. Ecol. Man. 200, 195–214.
Meentemeyer, R.K., Rank, N.E., Anacker, B.L., Rizzo, D.M., Cushman, J.H., 2008. Influence of land-cover change on the spread of an invasive forest pathogen. Ecol. Appl. 18 (1), 159–171.
Moralejo, E., Pérez-Sierra, A.M., Alvarez, L.A., Belbahri, L., Lefort, F., Descals, E., 2008. Multiple alien *Phytophthora* taxa discovered on diseased ornamental plants in Spain. Plant Pathol. 58, 100–110.
Neubauer, C., Gliessmann, J., Beltz, H., 2006. Zu den Ausbreitungswegen von *Phytophthora ramorum* an *Rhododendron* und *Viburnum* auf Container-Stellflächen. Gesunde Pflanzen 58, 185–191.
Orlikowski, L.B., Szkuta, G., 2004. First notice of *Phytophthora ramorum* on *Calluna vulgaris*, *Photina frasei* and *Pieris japonica* in Polish container-ornamental nurseries. Phytopathol. Pol. 34, 87–92.
Pakeman, R.J., Nolan, A.J., 2009. Setting sustainable grazing levels for heather moorland: a multi-site analysis. J. Appl. Ecol. 46, 363–368.
Parke, J.L., Linderman, R.G., Osterbauer, N.K., Griesbach, J.A., 2004. Detection of *Phytophthora ramorum* blight in Oregon nurseries and completion of Koch's postulates on *Pieris*, *Rhododendron*, *Viburnum* and *Camellia*. Plant Dis. 88, 87–187.
Pautasso, M., Jeger, M.J., 2008. Epidemic threshold and network structure: the interplay of probability of transmission and of persistence in directed networks. Ecol. Complex. 5, 1–8.
Prospero, S., Hansen, E.M., Grunwald, N.J., Winton, L.M., 2007. Population dynamics of the sudden oak death pathogen *Phytophthora ramorum* in Oregon from 2001 to 2004. Mol. Ecol. 16 (14), 2958–2973.
Rizzo, D.M., Garbelotto, M., Hansen, E.A., 2005. *Phytophthora ramorum*: integrative research and management of an emerging pathogen in California and Oregon forests. Ann. Rev. Phytopathol. 43, 309–335.
Shaw, M.W., 1984. *Rhododendron ponticum*—ecological reasons for the success of an alien species in Britain and features that may exist in its control. Asp. Appl. Biol. 5, 231–242.
Shaw, M.W., Harwood, T.D., Wilkinson, M.J., Elliott, L., 2006. Assembling spatially explicit landscape models of pollen and spore dispersal by wind for risk assessment. Proc. Biol. Sci. 273 (1594), 1705–1713.
Stokstad, E., 2004. Plant pathology: nurseries may have shipped sudden oak death pathogen nationwide. Science 303, 1959.
Turner, J., Jennings, P., Humphries, G., Parker, S., McDonough, S., Stonehouse, J., Lockley, D., Slawson, D., 2007. Natural outbreaks of *Phytophthora ramorum* in the U.K.—current status and monitoring update. In: Frankel, S.J., Kliejunas, J.T., Palmieri, K.M. (Eds.), Proceedings of the Sudden Oak Death Third Science Symposium. Santa Rosa, California, USA, 5–9 March, pp. 43–48 (PSW-GTR-214).
Webber, J.F., 2007. Status of *Phytophthora ramorum* and *P. kernoviae* in Europe. In: Frankel, S.J., Kliejunas, J.T., Palmieri, K.M. (Eds.), Proceedings of the Sudden Oak Death Third Science Symposium. Santa Rosa, California, USA, 5–9 March, pp. 19–26 (PSW-GTR-214).
Werres, S., Marwitz, R., Man in't Veld, W.A., De Cock, A.W., Bonants, P.J.M., De Weerdt, M., Themann, K., Ilievea, E., Baayen, R.P., 2001. *Phytophthora ramorum* sp. Nov., a new pathogen on *Rhododendron* and *Viburnum*. Mycol. Res. 105, 1155–1165.
Xu, X.-M., Harwood, T.D., Pautasso, M., Jeger, M.J., 2009. Spatio-temporal analysis of an invasive plant pathogen (*Phytophthora ramorum*) in England and Wales. Ecography 32 (3), 504–516.