

# Effects of landscape heterogeneity on the emerging forest disease sudden oak death

T. EMIKO CONDESO and ROSS K. MEENTEMEYER\*

Department of Biology, Sonoma State University, Rohnert Park, CA 94928, USA, and \*Department of Geography & Earth Sciences, University of North Carolina at Charlotte, Charlotte, NC 28223, USA

## Summary

**1** Sudden oak death is an emerging forest disease caused by the pathogen *Phytophthora ramorum* that is invading the west coast of the United States and semi-natural areas in Europe. This disease causes lethal stem infections in oaks (*Quercus* spp.) and tanoak (*Lithocarpus densiflorus*), as well as non-lethal foliar infections in a range of other species.

**2** We investigated two questions to evaluate the effect of landscape structure on the spread of *P. ramorum*: (i) does the spatial pattern of forested habitat predict *P. ramorum* disease severity, and is this relationship scale-dependent; and (ii) what influence does spatial pattern have on the optimal microclimate conditions for *P. ramorum* reproduction?

**3** We mapped the spatial distribution of suitable forest habitat for *P. ramorum* and established 86 randomly located field plots within a 20-km<sup>2</sup> region of northern California. For each plot, we quantified *P. ramorum* disease severity and measured the abundance of woody species. Disease severity in each plot was examined in relation to the surrounding landscape structure measured for nested landscapes of increasing scale.

**4** *P. ramorum* disease severity was greatest in plots surrounded by a high proportion of contiguous forest, after accounting for plot-level variables of host abundance, elevation, canopy cover and microclimate. The explanatory power of the model increased with increasing scale up to 200 m, but was not significant at scales less than 50 m.

**5** High disease severity was associated with lower temperatures in the field than the laboratory-determined optimal range for pathogen reproduction. Variation in microclimate conditions was explained by elevation, not the pattern of host vegetation, indicating that spatially varying disease severity was not a function of microclimate-related edge effects on pathogen growth and survival.

**6** Both landscape-scale configuration and local composition of host habitat are related to the severity of this destructive forest disease. Increased disease severity within contiguous woodlands may have a considerable impact on the composition of such woodlands, with cascading effects on the population dynamics of both host and pathogen.

*Key-words*: connectivity, fragmentation, emerging infectious disease, landscape pathology, invasive species, *Phytophthora ramorum*, sudden oak death

*Journal of Ecology* (2007) **95**, 364–375  
doi: 10.1111/j.1365-2745.2006.01206.x

## Introduction

A major consequence of human expansion and global movement is the number of emerging and re-emerging infectious diseases (Mayer 2000; Weiss & McMichael 2004; Ehrenfeld 2005). This increase in impact of infectious diseases is not unique to human populations. The

decline of biodiversity due to emerging pathogens of wildlife and plants may soon rival that due to habitat loss (Daszak *et al.* 2000; Dobson & Foufopoulos 2001; Harvell *et al.* 2002; Anderson *et al.* 2004). Emerging diseases of plants are typically non-native, as they are often introduced through the commercial trade of exotic plant products (Anderson *et al.* 2004). Port Orford cedar root disease (causal agent *Phytophthora lateralis*), chestnut blight (*Cryphonectria parasitica*) and jarrah dieback (*Phytophthora cinnamomi*) are examples of forest

diseases that have caused devastating population declines of their hosts, resulting in substantial ecological and economic impacts (Zobel *et al.* 1985; Anagnostakis 1987; Weste & Marks 1987). New reports of introduced plant diseases and their effects emerge continually, and are likely to become more frequent as global trade increases (Gilbert 2002; Orwig 2002).

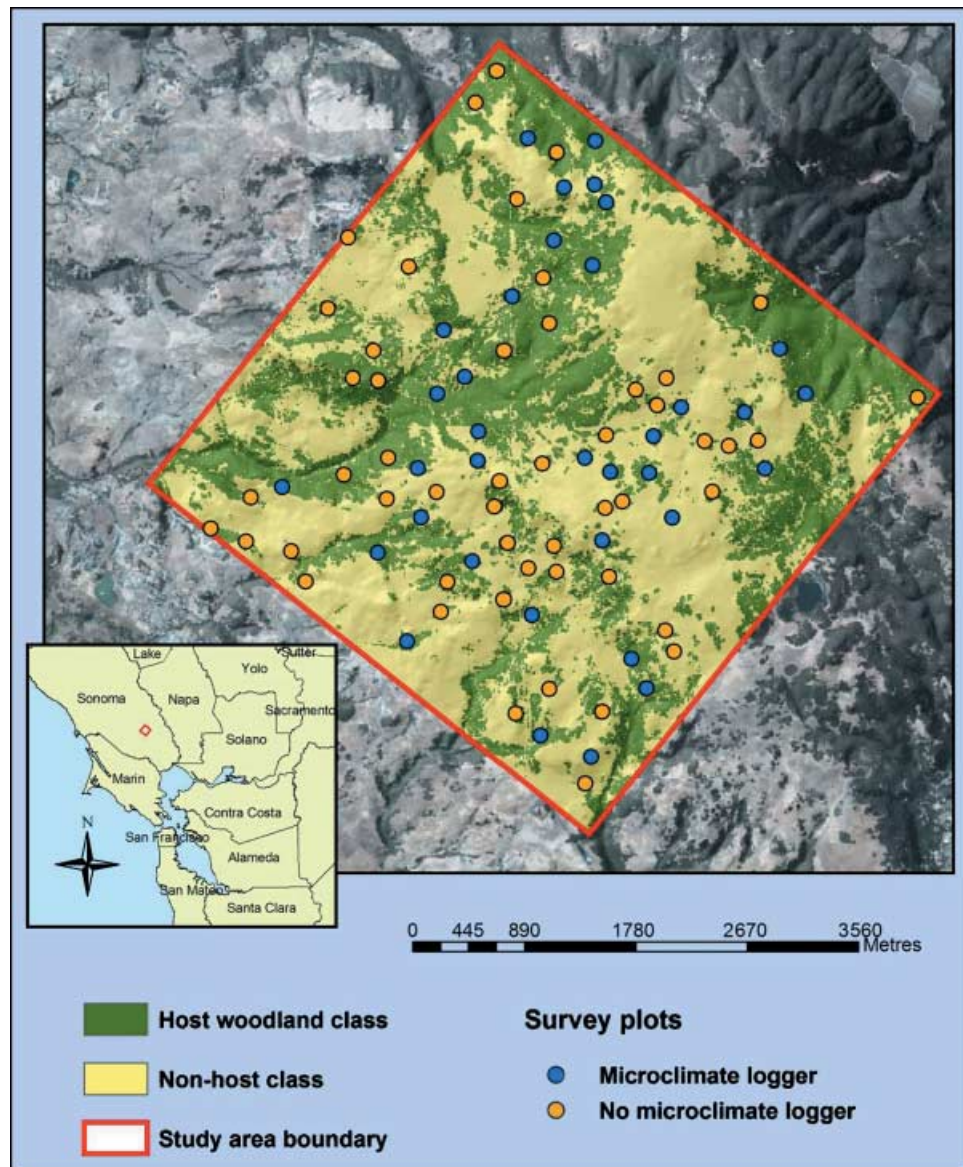
As the emergence of invasive pathogens and their impacts on ecological communities increases, so has the interest in understanding how landscape pattern (i.e. the configuration and composition of suitable habitat) affects their establishment and spread (Holdenrieder *et al.* 2004; Ostfeld *et al.* 2005). The process of invasion is inherently spatial, and each stage, from introduction to dispersal into new areas, is likely to be influenced by this aspect of landscape heterogeneity (Andow *et al.* 1990; Jerger 1999; With 2002). For the sake of simplicity, many empirical studies of plant pathogens have ignored landscape pattern as a factor influencing their establishment and spread (Ostfeld *et al.* 2005). Research has instead focused on relatively homogeneous host populations, or on the influence of local 'within-plot' host densities (Jules *et al.* 2002). However, landscapes are fragmented or patchy to varying degrees, whether naturally or by human modification (Hanski & Simberloff 1997; Tilman & Kareiva 1997; Haila 2002). The spread of forest pathogens, in particular, is likely to be influenced by heterogeneous spatial patterns of habitat because the habitat is itself composed of host, reservoir host and non-host species, and because outbreaks often occur at regional scales (Castello *et al.* 1995). Landscape pattern may also influence critical microclimate conditions that drive pathogen reproduction and survival (Davidson *et al.* 2005). Temperature in particular has been shown to respond to structural elements of habitat such as overstorey canopy (Chen *et al.* 1999). Plant pathogens that cause foliar diseases are especially sensitive to fluctuations in temperature and moisture (Woods *et al.* 2005). In addition, the effects of disease caused by forest pathogens can play a large role in shaping landscape pattern, which in turn creates the potential for feedback effects on spread rates (Chen *et al.* 1999; Holdenrieder *et al.* 2004).

A forest pathogen of recent concern is *Phytophthora ramorum* (Oomycota), the causal agent of 'sudden oak death', which has reached epidemic levels in coastal California (see review by Rizzo & Garbelotto 2003) and is also affecting managed landscapes in Europe (Brasier *et al.* 2004; Denman *et al.* 2005). Described in 2000 (Werres *et al.* 2001), the pathogen has caused extensive mortality of tanoak (*Lithocarpus densiflorus*), coast live oak (*Quercus agrifolia*), California black oak (*Quercus kelloggii*) and Shreve's oak (*Quercus parvula* vs. *shrevei*) in California and Oregon (Goheen *et al.* 2002; Rizzo *et al.* 2002). Although the pathogen's geographical origin remains unknown, its aggressive infection, limited geographical range and clonal population structure suggest that *P. ramorum* was probably introduced to North America (Rizzo & Garbelotto 2003; Ivors *et al.* 2006).

The symptoms caused by *P. ramorum* are expressed in two distinct forms: a lethal canker disease and a non-lethal foliar and twig infection. Whereas the canker disease appears limited to the previously mentioned *Quercus* and *Lithocarpus* species, the foliar blight affects a variety of woody species native to California (see Rizzo *et al.* 2005). The foliar hosts, especially the evergreen tree bay laurel (*Umbellularia californica*) are believed to play the major role in disease transmission, as dispersal spores (in the form of sporangia, zoospores and chlamydospores) are produced only from their leaf tissues (Davidson *et al.* 2002). Such dispersal spores have never been found or cultured from the tissues of canker hosts with the exception of tanoak, which is both a canker and a foliar host (Davidson *et al.* 2002).

In North America, *P. ramorum* occurs in the coastal forests of northern and central California with one infested area identified in south-western Oregon. However, given the high diversity and broad distribution of host species, this disease threatens thousands of hectares of woodlands (Rizzo & Garbelotto 2003; Meentemeyer *et al.* 2004; Barrett *et al.* 2006). There are a variety of potential avenues for *P. ramorum* dispersal. It is hypothesized that infective material may spread to nearby individuals by movement via rain splash (Davidson *et al.* 2002, 2005). Longer distance dispersal into uninfected areas may occur via wind-blown rain, stream water, movement of nursery stock and timber products, and through soil tracked by the movements of humans and other vertebrates (Cushman & Meentemeyer 2005; Davidson *et al.* 2005).

Although much research has focused on the biology of *P. ramorum* and mechanisms for its dispersal (Davidson *et al.* 2002, 2005; Rizzo *et al.* 2002, 2005), little is known about how the pattern of host habitat may influence *P. ramorum* disease severity, despite its potential influence (Holdenrieder *et al.* 2004; Ostfeld *et al.* 2005). The spatial arrangement and composition of host vegetation may play a crucial role in mediating spread of invasive organisms in general and infectious forest diseases in particular (Andow *et al.* 1990; Jules *et al.* 2002; Holdenrieder *et al.* 2004). In this paper, we investigate two questions to evaluate the effect of landscape pattern on the establishment and spread of the invasive forest pathogen *P. ramorum*: (i) does the composition and configuration of host habitat predict *P. ramorum* disease severity, and is this relationship scale-dependent; and (ii) what influence does the composition and configuration of host habitat have on the optimal microclimate conditions for *P. ramorum* growth and reproduction? We hypothesize that small isolated forest fragments have lower levels of *P. ramorum* infection, owing to an associated larger grassland dispersal barrier and less suitable microclimate conditions. Studies such as this, which examine the effects of landscape heterogeneity and the scale of a species' response to habitat, represent an integration of pathology and landscape ecology that will be critical to understanding *P. ramorum* disease establishment and spread.



**Fig. 1** Study area map based on aerial photography. The land-cover classes (host and non-host) and locations of survey plots are shown.

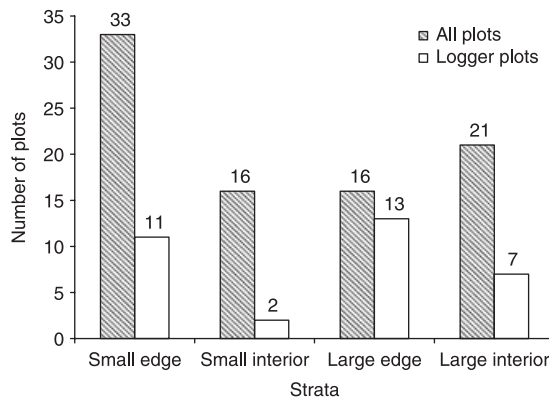
## Methods

### STUDY SYSTEM AND VEGETATION MAPPING

This study was conducted in a 20.25-km<sup>2</sup> region on Sonoma Mountain, with elevation ranging from 170 to 748 m (Sonoma County, California, USA; Fig. 1). The size and position of the study area was chosen to minimize the effect of potentially confounding variables such as solar aspect, climate, infection history and land-use history. This predominantly west-facing section of Sonoma Mountain has a Mediterranean-type climate characterized by cool, rainy winters and warm, dry and often foggy summers. Vegetation is primarily composed of distinct patches of mixed evergreen forest (dominated by bay laurel and oak species) in a matrix of exotic annual grassland (Fig. 1). This simple binary study system of host habitat (bay laurel/oak forest) and non-host habitat (annual grassland) was chosen to minimize the

effect of multiple dispersal barriers (non-host land cover types). The impact of *Phytophthora ramorum* on the vegetation of Sonoma Mountain was first observed in 2000, and ongoing studies show that the pathogen is widely distributed across the region (Cushman & Meentemeyer 2005; Davidson *et al.* 2005). Although detailed land-use history is not known, the study area has experienced a common history of grazing and some logging over the past century.

To examine the effects of landscape heterogeneity, a map of woodland habitat was derived from Airborne Data Acquisition and Registration (ADAR) multispectral aircraft imagery (August 2001, Positive Systems, Inc., Whitefish, MT, USA). ADAR has a spatial resolution of 1 × 1 m, and collects data from four spectral bands (red, green, blue and near-infrared). Pixels were classified into land cover categories using a supervised approach, where field data were used to develop spectral signatures for pixels of known vegetation composition. These



**Fig. 2** The distribution of all survey plots and plots containing microclimate loggers among the selection strata. Strata were defined by patch size and distance to edge. Large patches were larger than 10 ha, interior woodland was defined as greater than 15 m from a large canopy break (larger than 225 m<sup>2</sup>). Because small patches are usually 'all edge', many more plots fell into this category.

signatures were then used to place the remaining pixels in the image into the appropriate land cover classes using a maximum-likelihood classifier (Erdas Imagine 8.7, Leica Geosystems, Norcross, GA, USA). The map was re-sampled using a nearest-neighbour algorithm to 5-m resolution to increase the computational efficiency of landscape pattern metric calculation (ArcInfo 8, ESRI, Redlands, CA, USA). The final study area map contained approximately 3.6 million cells and was composed of two land cover classes: a host-woodland class and a non-host class (Fig. 1). The woodland class was dominated by bay laurel and coast-live oak, but also contained California black oak, Oregon white oak (*Quercus garryana*), big leaf maple (*Acer macrophyllum*), California buckeye (*Aesculus californica*), toyon (*Heteromeles arbutifolia*) and a small amount of Douglas fir (*Pseudotsuga menziesii*). The non-host class was primarily composed of exotic annual grassland (75%), but also included a small percentage of other non-host classes such as farm ponds and seeps, agricultural land (vineyards), exposed land (bare ground) and residential developments. The host-woodland and non-host classes comprised 43% and 57% of the total study area,

respectively. Overall pixel classification accuracy was 98% based on field plot data described below.

#### DISEASE SEVERITY ASSESSMENT

To assess variation in disease severity across the study area, we established 86 field plots (225 m<sup>2</sup>, 15 × 15 m) within host woodland patches following a stratified-random criterion defined by woodland patch size and distance to a forest–grassland boundary (Figs 1 & 2). Plot locations within each stratum were chosen by randomly selecting pixels from the digital map of woodland habitat produced for the study area. Field crews navigated to each location using GPS (global positioning satellite) receivers and centred each plot at the selected coordinates with a horizontal accuracy within 1 m using differential correction (Trimble Navigation Limited, Sunnyvale, CA, USA).

*Phytophthora ramorum* causes a foliar blight on bay laurel and symptomatic leaves feature characteristic spotting and necrosis, usually at the tip (Fig. 3; and see Rizzo *et al.* 2005). Assessment of disease severity focused on bay laurel as it is the primary producer of inoculum in this forest type (Davidson *et al.* 2005), typically the first species in an area to show infection by *P. ramorum* (Rizzo *et al.* 2005) and is abundant within the study area (bay laurel was present in 81 of 86 plots, and represented 69% of all trees observed in plots). Laboratory and field evidence from this region also suggests that inoculum from bay laurel plays a crucial role in vectoring *P. ramorum* to oaks (Davidson *et al.* 2002; Kelly & Meentemeyer 2002; Swiecki & Bernhardt 2002).

Given that most bays in the study area exhibited some level of *P. ramorum* infection, assessing an individual as only infected or uninfected was deemed too coarse to examine inter-plot variation in disease severity. Therefore, we quantified disease severity in a plot by counting the number of symptomatic leaves on each bay laurel stem greater than 2 cm in diameter at breast height (d.b.h., defined as 1.4 m) for 90 s. Fieldwork was completed by six observers (each tree was counted once) between 28 March and 29 April 2005, the general period of peak disease expression (Davidson *et al.* 2005). Prior to field sampling, we conducted a preliminary study to



**Fig. 3** Typical symptoms of *Phytophthora ramorum* infection on bay laurel (*Umbellularia californica*) leaves.

**Table 1** Landscape pattern metrics used in analyses. Source: McGarigal *et al.* (2002) Fragstats 3.3 Documentation

| Pixel-based metrics calculated for nested landscapes                                                                                                        |                                                                                                                                                                                                                                                                          |
|-------------------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Area (percentage of the landscape that is woodland)                                                                                                         | Sum of all cells within the extent that are woodland, divided by the total area of the extent.                                                                                                                                                                           |
| Mean perimeter : area ratio                                                                                                                                 | Average perimeter : area ratios of all patches within the extent.                                                                                                                                                                                                        |
| $\text{Patch cohesion} = \left[ 1 - \frac{\sum_{j=1}^n p_{ij}}{\sum_{j=1}^n p_{ij} \sqrt{a_{ij}}} \right] \left[ 1 - \frac{1}{\sqrt{A}} \right]^{-1} * 100$ | One minus the sum of patch perimeter divided by the sum of patch perimeter times the square root of patch area for woodland patches, divided by 1 minus 1 over the square root of the total number of cells in the extent, multiplied by 100 to convert to a percentage. |
| $p_{ij}$ = perimeter of patch $ij$<br>$a_{ij}$ = area of patch $ij$<br>$A$ = total area of the extent                                                       |                                                                                                                                                                                                                                                                          |

calibrate leaf symptom observations among observers and determine repeatability of the sampling method. There were no significant differences in the number of symptomatic leaves counted among observers ( $F_{5,153} = 1.10$ ,  $P = 0.36$ ), no significant increases in leaf counts across three time intervals of 60 s (mean =  $15.43 \pm 1.538$ ), 90 s (mean =  $18.04 \pm 1.543$ ) and 120 s ( $19.5 \pm 1.538$ ) ( $F_{2,476} = 1.84$ ,  $P = 0.16$ ), and there was no significant relationship between leaf count and d.b.h. of an individual tree ( $F_{1,1149} = 0.19$ ,  $P = 0.66$ ).

We confirmed the presence of *P. ramorum* in the symptomatic leaves by collecting up to 36 symptomatic bay laurel leaves from each of three randomly selected trees in each plot. Leaf material was cultured according to standard methods, by plating on standard pimaricin–ampicillin–rifampicin–pentachloronitrobenzene-selective agar (PARP) and incubating at 15 °C for 1–2 weeks (Erwin & Ribeiro 1996). Cultures were then assessed under magnification for diagnostic characteristics and verification of *P. ramorum* (Davidson *et al.* 2002). Other *Phytophthora* species which can cause similar symptoms were not detected in the study area.

#### LANDSCAPE PATTERN ASSESSMENT

Analysis of landscape patterns requires quantification of the arrangement and composition of cover types. Although a large number of landscape metrics have been developed for this purpose, no single metric captures all information about landscape pattern, and strong correlations between many make choosing an appropriate suite difficult (Li & Reynolds 1995; McGarigal & McComb 1995; Riitters *et al.* 1995; Gustafson 1998; Hargis *et al.* 1998; Li & Wu 2004; Wagner & Fortin 2005). In addition, some metrics provide a linear index only over particular levels of fragmentation (Hargis *et al.* 1998). With these caveats, we derived three simple metrics that described the size, shape and connectivity of host woodland using Fragstats 3.3; these were woodland area, perimeter : area ratio and patch cohesion (McGarigal *et al.* 2002) (Table 1).

To examine the spatial scale at which *P. ramorum* responds to landscape pattern, each landscape metric was quantified using a multiscale nested approach (e.g. Chust

*et al.* 2004; Holland *et al.* 2004). The metrics of area, shape and connectivity were calculated for nested landscapes of increasing size surrounding each sample plot. Each nested landscape was delineated by a circular boundary centred on the plot, with increasing radii of 50-m increments from 50 to 500 m. To capture smaller-scale effects of landscape pattern, we also included a 30-m radius.

#### PLOT-LEVEL VARIABLES

To examine the influence of local conditions in addition to landscape pattern, we also measured the following variables within each of the 86 field plots: abundance of bay laurel, canopy cover, distance to forest edge and elevation. Bay abundance was estimated by measuring the cumulative d.b.h. of all bay stems that exceeded 2 cm in d.b.h. within the plot (min. – max. = 27–397, mean = 313, SD = 222). Canopy cover was estimated by standing at the plot centre and visually approximating the percentage of the plot covered by canopy vegetation (four plots had < 25% cover, 10 plots had 26–50%, 24 had 51–75% and 48 plots had > 75%). Distance to forest edge was measured from aerial imagery in the GIS and defined as the shortest linear distance between the plot edge and a canopy break larger than 225 m<sup>2</sup> (min. – max. = 1–247, mean = 35, SD = 53). Elevation of each plot was also calculated in the GIS based on a USGS 10-m digital elevation model (min. – max. = 239–717, mean = 526, SD = 118).

#### MICROCLIMATE ASSESSMENT

To examine the influence of landscape pattern on microclimate conditions, we installed microclimate loggers (Onset Corp., Bourne, MA, USA), in a subset of 35 plots distributed across the strata used for plot placement (Figs 1 & 2). Each logger was positioned in the centre of its plot on a mounting pole 1 m off the ground and was housed inside a protective solar radiation shield. Temperature and relative humidity were recorded every hour during the pathogen's reproductive season (Davidson *et al.* 2005) from the beginning of the winter rainy season (1 December 2004) until rain ceased at the beginning of the summer, dry season (21 June 2005).

Co-kriging was used to interpolate spatially the amount of rainfall that occurred each day, at each plot location, based on a network of 16 precipitation gauges that we established across the Sonoma Mountain area (Goovaerts 2000). Using the rain data with the temperature and humidity data, we calculated five microclimate variables for this period based on optimal conditions for *P. ramorum* reproduction (sporangia and zoospore production) identified in the laboratory by Davidson *et al.* (2005). The five variables included: (i) average daily number of hours in the optimal temperature range (10–25 °C) for *P. ramorum* reproduction (Hours Optimal Temperature); (ii) average daily number of hours at high relative humidity (> 95%, Hours Optimal Relative Humidity); (iii) number of hours in the optimal temperature range (10–25 °C) during and up to 24 h after a rain event of at least two consecutive days (Hours Optimal Temperature: rain event); (iv) number of hours at lower than optimal temperatures (0–10 °C) during and up to 24 h after a rain event of at least two consecutive days (Hours Non-optimal Temperature: rain event); and (v) the average daily number of hours at high temperatures (outside of the optimal range for *P. ramorum* reproduction or > 25 °C, Hours High Temperature).

#### STATISTICAL ANALYSIS

The response of cumulative symptomatic leaf count per plot to the pattern of host woodland (area, perimeter : area ratio and patch cohesion) was examined using multiple regression, testing all possible subsets of predictors and identifying the best model using the Akaike Information Criterion (AIC) (*sensu* Quinn & Keough 2002). Standard beta coefficients were used to assess the relative importance of each of the significant predictors in the regression model as the predictors were measured in different units (Quinn & Keough 2002). Potentially influential plot-level variables were controlled for by including the aforementioned measures of the cumulative d.b.h. of bay laurel, plot canopy cover, distance to forest edge and elevation. Microclimate variables were analysed separately to avoid problems due to multicollinearity with the landscape pattern metrics (Quinn & Keough 2002; Graham 2003).

To identify the spatial scale at which *P. ramorum* responds to landscape pattern, we fitted a separate regression model for the plot data (only plot-level variables) and for each of the 11 nested scales (which additionally included the metrics of area, shape and connectivity), while controlling for the plot-level covariates. Variables that were not significant at the  $P < 0.05$  level in the full models were omitted and the models were re-run. All landscape pattern variables and the leaf count response variable were log transformed, and cumulative d.b.h. of bay laurel was root transformed to compensate for non-linearity and the influence of observations with high leverage (Quinn & Keough 2002). We also computed Geary's C to test for spatial autocorrelation of the response variable among plots (Geary 1954; Cliff & Ord 1981).

Geary's C equaled 1.00001, indicating that symptomatic leaf counts in neighbouring plots were unrelated and assumptions of independence were not violated.

Once it was determined which landscape variables, plot-level covariates and microclimate predictors were highly related, the effects of the microclimate variables on disease severity were tested on the subset of plots containing microclimate loggers ( $n = 32$ ) at the scale at which the landscape variables had the greatest effect on disease severity. This final model was selected by testing all possible subsets of the predictor variables including the microclimate variables but excluding other covariates with which they were highly correlated. The final model contained the best predictors in the presence of no highly correlated variables.

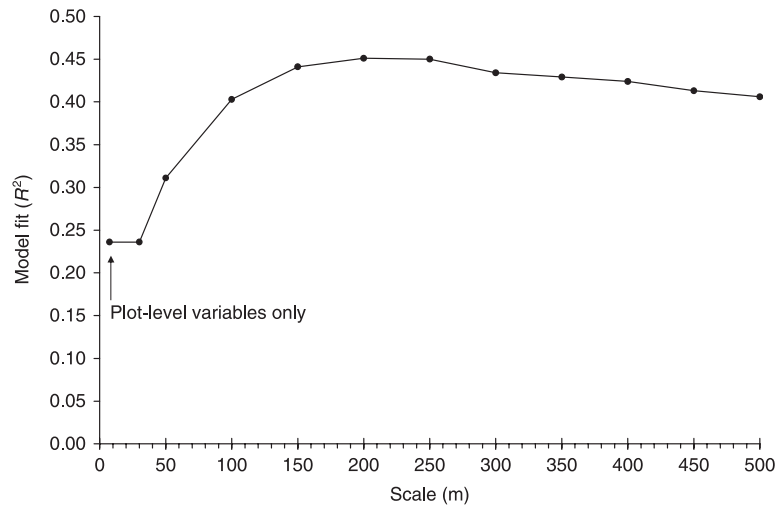
## Results

#### EFFECTS OF LANDSCAPE PATTERN ON DISEASE SEVERITY

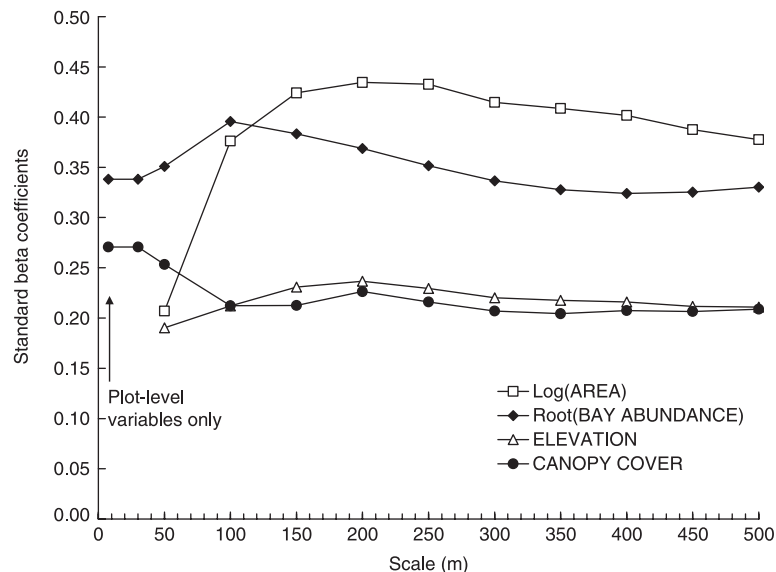
The results of the multiscale analysis revealed a scale-dependent effect of landscape pattern on disease severity (Fig. 4). At the plot scale, with no landscape pattern variables included, model fit was lowest, and of the plot-level variables only bay abundance ( $F_{1,78} = 10.8$ ,  $P = 0.002$ ) and canopy cover ( $F_{1,78} = 6.9$ ,  $P = 0.01$ ) significantly predicted leaf count (Fig. 5). With landscape context added at the smallest spatial scale (30 m), there were no significant landscape pattern variables, making the final model identical to the plot-scale model (Figs 4 & 5). At larger scales, model fit steadily increased from 50 m ( $r^2 = 0.31$ ,  $P < 0.0001$ ) to a peak at 200 m ( $r^2 = 0.45$ ,  $P < 0.0001$ ) and then declined slightly with increasing scale to 500 m (Fig. 4). At all scales greater than 30 m, woodland area, bay abundance, elevation and canopy cover significantly predicted leaf count at the  $P < 0.05$  level (Fig. 5). The effect of woodland area increased from 50- to 200-m scales, with the strongest effect at scales greater than 100 m (Fig. 5). The effect of bay abundance was fairly consistent across all scales (Fig. 5). The effect of canopy cover was highest at small scales, then decreased between 30- and 100-m scales to reach a low of  $\beta_{100} = 0.21$  (Fig. 5). The effect of elevation became significant at the 50-m scale and remained fairly consistent (Fig. 5). Mean perimeter : area ratio of host woodland within each nested landscape did not predict leaf count at any scale. Patch cohesion was highly correlated with woodland area at all scales (Pearson's  $r$  ranging from 0.77 to 0.92,  $P < 0.0001$ ) and was omitted from these models because when evaluated as an alternative variable, woodland area consistently explained more variance in leaf count across all spatial scales.

#### EFFECTS OF LANDSCAPE PATTERN ON MICROCLIMATE

Regression analysis revealed that two of the five reproductive-season microclimate variables were related



**Fig. 4** Multiple regression results for the multiscale analysis. The figure shows model fit ( $r^2$ ) across varying spatial scales. The model at the smallest scale (7.5 m) represents the plot level, with no landscape context variables. All models are significant at the  $P < 0.0001$  level. Single variables are significant at the  $P < 0.05$  level.



**Fig. 5** Multiple regression results for the multiscale analysis. The figure shows standardized beta coefficients for significant ( $P < 0.05$ ) predictors of leaf count across scales. The model at the smallest scale (7.5 m) represents the plot level, with no landscape context variables.

to landscape pattern and four were associated with plot-level variables (Table 2). The average daily number of hours at  $> 95\%$  relative humidity was positively related to woodland area and elevation ( $r^2 = 0.69$ ,  $P < 0.0001$ ,  $n = 35$ ), although elevation alone explained most of the variation ( $r^2 = 0.58$ ,  $P < 0.0001$ ,  $n = 35$ ) (Table 2). The number of hours within the optimal temperature range ( $10\text{--}25\text{ }^\circ\text{C}$ ) during and 24 h after two consecutive days of rain was negatively associated with elevation ( $r^2 = 0.75$ ,  $P < 0.0001$ ,  $n = 35$ ) (Table 2). Conversely, the number of hours at lower than optimal temperatures ( $< 10\text{ }^\circ\text{C}$ ) during and 24 h after two consecutive days of rain was positively associated with elevation ( $r^2 = 0.68$ ,  $P < 0.0001$ ,  $n = 35$ ) (Table 2). This variable was also positively related to woodland shape measured at the 200-m scale

(Table 2). The average daily number of hours at high temperatures ( $> 25\text{ }^\circ\text{C}$ ) was negatively associated with elevation ( $r^2 = 0.17$ ,  $P < 0.01$ ,  $n = 35$ ), but during the reproductive season only 63% of plots (22/35) reached temperatures this high.

As the optimal microclimate conditions of *P. ramorum* were more closely associated with elevation than landscape pattern, an additional model for predicting symptomatic leaf count was developed using the microclimate variables as a substitute for elevation for the subset of plots with both logger and leaf count data available ( $n = 32$ , Table 3). The number of hours within the optimal temperature range for *P. ramorum* associated with a rain event, the number of hours at lower than optimal temperatures associated with a rain event, and

**Table 2** Multiple regression results for the effects of landscape pattern on each microclimate summary variable ( $n = 35$ ). Microclimate variables were summarized for the reproductive season of *Phytophthora ramorum*, defined as winter and spring (December 2004 to 21 June 2005). Models were significant at the  $P < 0.001$  level.  $\beta$  = the standardized beta coefficient for each effect. Woodland area and shape were measured at the 200-m scale. The effect of woody species abundance was not significant

| Summary                                    | Effect $\beta$     |      |       |          |           |      |                  |              |       |
|--------------------------------------------|--------------------|------|-------|----------|-----------|------|------------------|--------------|-------|
|                                            | Dependent variable | $F$  | $r^2$ | AIC      | Elevation | Area | Distance to edge | Canopy cover | Shape |
| Hours† Optimal Temperature                 | NS                 | NS   | NS    | NS       | NS        | NS   | NS               | NS           | NS    |
| Hours‡ Optimal Relative Humidity           | 36.1               | 0.69 | -22.1 | 0.89***  | 0.36**    | NS   | NS               | NS           | NS    |
| Hours‡ Optimal Temperature: rain event     | 97.9               | 0.75 | 263.1 | -0.86*** | NS        | NS   | NS               | NS           | NS    |
| Hours‡ Non-optimal Temperature: rain event | 34.3               | 0.68 | 277.0 | 0.85***  | NS        | NS   | NS               | NS           | 0.26* |
| Hours† High Temperature                    | 7.0                | 0.17 | 0.52  | -0.42**  | NS        | NS   | NS               | NS           | NS    |

\* $P < 0.05$ ; \*\* $P < 0.01$ ; \*\*\* $P < 0.001$ ; NS, not significant.

†Average daily number of hours; ‡number of hours during and up to 24 h after two consecutive days of rain.

**Table 3** Multiple regression results for the effects of microclimate variables on symptomatic leaf count ( $n = 32$ ). Models were significant at the  $P < 0.0001$  level.  $\beta$  = the standardized beta coefficient for each effect. Woodland area and shape were measured at the 200-m scale; effect shape was not significant. Other microclimate variables, distance to edge and canopy cover did not significantly predict symptomatic leaf count for this subset

| Summary                                                           | Effect $\beta$     |      |       |         |         |               |           |          |                          |
|-------------------------------------------------------------------|--------------------|------|-------|---------|---------|---------------|-----------|----------|--------------------------|
|                                                                   | Dependent variable | $F$  | $r^2$ | AIC     | Area    | Bay abundance | Elevation | Hours OT | Hours non-OT: rain event |
| Area, bay abundance, hours OT, hours non-OT: rain event, hours HT | 13.6               | 0.77 | -53.6 | 0.51*** | 0.64*** | -             | 0.27*     | 0.45***  | 0.44**                   |
| Area, bay abundance, elevation                                    | 15.5               | 0.62 | -55.4 | 0.61*** | 0.51*** | 0.27*         | -         | -        | -                        |

\* $P < 0.05$ ; \*\* $P < 0.01$ ; \*\*\* $P < 0.001$ ; -, omitted.

the average daily number of hours at optimal relative humidity were examined as alternatives because of the degree to which they covaried. Variables that significantly predicted leaf count were the number of hours logged at lower than optimal temperatures during and after rain, the average daily number of hours at optimal temperatures, the average daily number of hours at high temperatures, bay abundance and woodland area measured at 200 m (Table 3). The number of hours at lower than optimal temperatures during and after rain was positively related to symptomatic leaf counts (Table 3). In comparison, substitution of microclimate (whole model  $r^2 = 0.77$ ,  $P < 0.0001$ ,  $n = 32$ ) for elevation (whole model  $r^2 = 0.62$ ,  $P < 0.0001$ ,  $n = 32$ ) (Table 3) marginally increased the explanatory power of the model for this subset of the data.

## Discussion

Predicting spatial dynamics of disease establishment and spread in forest ecosystems requires knowledge of not only the spatial arrangement and composition of host vegetation, but also the appropriate scales at which to examine host-pathogen interactions. In this study, we determined that: (i) *Phytophthora ramorum* disease severity is greater in plots surrounded by a high percentage of woodland habitat; (ii) the effect of landscape

pattern on disease severity is scale-dependent; and (iii) in this system, optimal microclimate conditions for *P. ramorum* reproduction and growth are influenced more by topography than by landscape pattern.

## EFFECTS OF LANDSCAPE PATTERN ON DISEASE SEVERITY

Multiscale analyses allow landscape pattern to be examined across space as it may be perceived by an organism, with no a priori assumptions about the perceptions of patch boundaries or the scale that the organism responds to its environment (e.g. Holland *et al.* 2004; Wagner & Fortin 2005). Results of our analysis demonstrated that the response of *P. ramorum* to habitat conditions depended on landscape pattern and the spatial scale of the observation. The result that *P. ramorum* had no response to landscape pattern at small scales (less than 50 m), but that the effect of area increased up to the 200-m scale, indicates that *P. ramorum* disease severity at a particular plot is most influenced by the amount of surrounding forest within a radius of 200 m (12.5 ha). Species often respond to landscape pattern at different scales (e.g. Roland & Taylor 1997) and it is typically hypothesized that the scale at which they respond most strongly to pattern is related to the movement range of the organism (e.g. Holland *et al.*

2004). A radius of 200 m, the scale at which fragmentation effects are most meaningful, may correspond to a dispersal limitation for *P. ramorum* in this study area. However, because the proportion of woodland surrounding each plot did not markedly change at scales greater than 200 m, model fit and effect  $\beta$  of woodland area also did not decrease considerably. Future studies focusing on the frequency of successful dispersal across larger distances are needed.

As the amount of woodland surrounding a plot increases, it is likely that the abundance of inoculum-producing hosts (e.g. bay laurel) also increases. Therefore, high disease severity in areas surrounded by continuous forest is probably due to a greater available inoculum reservoir. In this analysis, plot surroundings were measured at multiple scales, as opposed to only considering the characteristics within the field plot or the forest patch it occupied. The positive association between woodland area and disease severity is consistent with the general predictions of island biogeography and metapopulation theory (MacArthur & Wilson 1967; Levins 1969) and is consistent with other studies of the effect on host population size on disease incidence and severity (Burdon *et al.* 1995; Ericson *et al.* 1999; Thrall *et al.* 2003). Large, contiguous swathes of host woodland may provide more surface area to intercept incoming propagules compared with smaller ones, and thus increased disease severity may be partially due to increased colonization rates (Burdon *et al.* 1989). Once invaded, large areas of woodland are likely to support higher disease levels because they are likely to contain a greater number of susceptible hosts (Burdon *et al.* 1989), resulting in the potential for higher inoculum production within contiguous woodland and a lower probability of disease extinction (Burdon *et al.* 1989). Over time, large areas may be more effective pathogen reservoirs, maintaining more resilient levels of disease than their smaller neighbours.

Fragmentation of woodlands is likely to reduce the spread rate of pathogens that are less mobile and have vulnerable dispersal stages, with the probability of transmission declining with distance from an infected host (Ostfeld *et al.* 2005). Highly mobile species tend to respond to landscape pattern at broader scales than less mobile species (Wiens 1989). Although dispersal of *P. ramorum* via wind-blown rain rarely reached 15 m in studies conducted by Davidson *et al.* (2005), they hypothesize that high winds associated with relatively rare storm events could move spores over far greater distances. Davidson *et al.* (2005) did find that inoculum may be highly mobile when dispersed through stream water, and detected dispersal 1 km from an inoculum source. In this study system, even the most isolated patches surrounded by as much as 50 m of grassland had moderately high disease severity.

In all regressions, bay abundance, elevation and canopy cover were consistently significant plot-level covariates. The positive influence of bay abundance on disease severity corresponds to the explanation that more

susceptible hosts lead to higher general infection levels. The consistent positive association between disease severity and elevation at scales greater than 30 m is probably due to topographically driven differences in optimal temperature and moisture conditions for *P. ramorum*. It is also possible that greater wind velocities at high elevations increase the rate of leaf-to-leaf and/or tree-to-tree spread. The positive association between disease severity and plot canopy cover is probably due to cooler and moister microclimate conditions in woodlands with denser canopy cover. However, the local effect of canopy cover seemed to diminish after accounting for the large-scale effects of woodland area.

#### MICROCLIMATE ANALYSIS

Analysis of microclimate logger data indicated that most of the variation in the optimal microclimate conditions of *P. ramorum* was due to elevation not landscape pattern. This result indicates that the effect of landscape pattern on disease severity is independent of the effect of microclimate. Only the number of hours at lower than optimal temperatures during and 24 h after a 2-day rain event varied significantly with forest shape, and the average daily number of hours at optimal relative humidity with forest area. In both cases, however, elevation explained most of the variation in this measurement. Edge effects, or spatially varying differences in microclimate conditions, are often called upon to explain spatially varying differences in habitat quality, and thus species abundances (Chen *et al.* 1999; Ostfeld *et al.* 2005). However, in this oak woodland system, none of the microclimate variables measured varied significantly with distance to woodland edge after accounting for the effect of topography. This may be due to the open canopy structure and mix of deciduous and evergreen species in oak woodlands, as microclimate variables are known to be sensitive to changes in overstorey canopy, and variability within these forest types is high (Chen *et al.* 1999). Temperature and moisture may vary more dramatically as a function of landscape pattern across areas with more extreme climatic gradients, and may be predictive of disease severity in such environments.

The subset analysis to determine directly the strength and direction of the effect of microclimate on symptomatic leaf count unexpectedly showed that more hours at lower than optimal temperatures (0–10 °C) during and 24 h after 2 days of continuous rain was associated with greater disease severity. This result suggests that in the field, *P. ramorum* disease severity may reach higher levels at lower temperatures than would be expected based on laboratory studies of zoospore production (Davidson *et al.* 2005). It is possible that in the field, temperature and relative humidity may affect the susceptibility of bay laurel, with lower temperatures and moist conditions promoting leaf stomata to be open for longer periods and providing more opportunity for infection. It is also likely that water remains on leaf surfaces for longer periods at lower temperatures. Once

rainwater on the leaves evaporates, infection and pathogen reproduction may cease and zoospore survival may decline, even at optimal temperatures (10–25 °C).

The emergence and rapid spread of *P. ramorum* has increased awareness of the impact of invasive pathogens on natural communities. Regulatory officials, land managers and landowners are in need of information that will help them understand the potential ecological impacts of this disease and guide management decisions. These data suggest that forest fragmentation may slow the spread and reduce the overall abundance of pathogens such as *P. ramorum*, although it is not known at this time how it will impact the overall mortality rate. However, it is possible that fragmentation will keep inoculum levels low, reducing the likelihood of oak infection. The effect of fragmentation may be achieved by means other than thinning or clearing. Forests could be managed for decreased connectivity of reservoir hosts (such as bay laurel) by increasing diversity of non-host species within a forest stand (Perry 1988; Pautasso *et al.* 2005; Waring & O'Hara 2005; Keesing *et al.* 2006). It is also important to note that fragmentation at the scale of this study did not completely isolate woodland patches from disease, so thinning or removal of forest is not a guaranteed preventative measure. Rizzo *et al.* (2005) speculated that thinning of woodlands could increase air flow through stands and thus actually facilitate tree-to-tree dispersal of *P. ramorum* via wind, although in this study, increased density of woody species within a plot resulted in higher counts of symptomatic leaves on bay laurel. Intentional removal of forest habitat may also have other unwanted and unexpected ecological effects and impacts on associated species (Soule & Simberloff 1986; Wilcove *et al.* 1986). The causes, and not simply the effects, of the spatial pattern of vegetation in an area should be considered when making management decisions. For example, fire suppression may have increased the area and density of woodlands in Sonoma County relative to historic levels (R.K. Meentemeyer & J.H. Cushman, unpublished data) and created conditions which favour disease (Rizzo & Garbelotto 2003; Moritz & Odion 2005). In addition, although it is useful to examine differences in disease severity across a woodland landscape at the relevant scale, drawing conclusions about the spatial dynamics of *P. ramorum* from just one landscape is cautioned owing to potential pseudoreplication, which may be associated with large-scale studies (Hurlbert 1984; Li & Wu 2004). Although the effects of landscape pattern and plot-level covariates account for a significant amount of variation in disease severity (as much as 45%), additional explanations for unaccounted for variation should also be explored. One potential source of unexplained variance may be landscape variability in host and/or pathogen genotype (Lundquist & Klopfenstein 2001; Dodd *et al.* 2005). Future research is also needed on the relationship between fragmentation and disease incidence across additional landscapes, including those with more complex mosaics of vegetation and human land uses.

Collectively, these analyses suggest that both landscape configuration and local characteristics of host habitat influence the invasion of a destructive pathogen. Determining the spatial scale of a species' response to habitat is critical for understanding movement ranges and dispersal of invasive organisms such as *P. ramorum*. Future studies should examine the effects of landscape heterogeneity on disease dynamics over time and utilize these data, along with other biotic and abiotic predictors, to analyse disease risk across susceptible areas and inform management decisions. Landscape pathology is a growing field and in this period of continuing human expansion, will be increasingly needed to predict and manage the spread of emerging infectious diseases worldwide.

### Acknowledgements

We thank J.H. Cushman, N.E. Rank, D. Rizzo and two anonymous referees for valuable comments on the analysis and manuscript. Research technicians and fellow students who provided invaluable technical assistance, manpower and moral support were J. Amaris, B. Anacker, S. Benson, C. Boylen, D. DiPietro, E. Gordon, R. Hunter, E. Lotz, L. Miller, S. Moyle, P. Smith and D. Zanzot. The Cypress Grove Research Center of Audubon Canyon Ranch provided additional support for the completion of this manuscript. Special thanks to the landowners on Sonoma Mountain who granted access to their properties. This work was supported by NSF grant DBI-0217064.

### References

- Anagnostakis, S. (1987) Chestnut blight: the classical problem of an introduced pathogen. *Mycologia*, **79**, 23–37.
- Anderson, P.K., Cunningham, A.A., Patel, N.G., Morales, F.J., Epstein, P.R. & Daszak, P. (2004) Emerging infectious diseases of plants: pathogen pollution, climate change and agrotechnology drivers. *Trends in Ecology and Evolution*, **19**, 535–544.
- Andow, D.A., Kareiva, P.M., Levin, S.A. & Okubo, A. (1990) Spread of invading organisms. *Landscape Ecology*, **4**, 177–188.
- Barrett, T.M., Gatzolis, D., Fried, J.S. & Waddell, K.L. (2006) Sudden oak death in California: what is the potential? *Journal of Forestry*, **104**, 61–64.
- Brasier, C.M., Denman, S., Rose, J., Kirk, S.A., Hughes, K.J.D., Griffin, R.L., Lane, C.R., Inman, A.J. & Webber, J.F. (2004) First report of ramorum bleeding canker on *Quercus falcata*, caused by *Phytophthora ramorum*. *Plant Pathology*, **53**, 804–804.
- Burdon, J.J., Ericson, L. & Muller, W.J. (1995) Temporal and spatial changes in a metapopulation of the rust pathogen *Triphragmium ulmariae* and its host, *Filipendula ulmaria*. *Journal of Ecology*, **83**, 979–989.
- Burdon, J.J., Jarosz, A.M. & Kirby, G.C. (1989) Pattern and patchiness in plant–pathogen interactions – causes and consequences. *Annual Review of Ecology and Systematics*, **20**, 119–136.
- Castello, J.D., Leopold, D.J. & Smallidge, P.J. (1995) Pathogens, patterns, and processes in forest ecosystems. *Bioscience*, **45**, 16–24.

- Chen, J., Saunders, S.C., Crow, T.R., Naiman, R.J., Brosfoske, K.D., Mroz, G.D., Brookshire, B.L. & Franklin, J.F. (1999) Microclimate in forest ecosystem and landscape ecology. *Bioscience*, **49**, 288–297.
- Chust, G., Pretus, J.L., Ducrot, D. & Ventura, D. (2004) Scale dependency of insect assemblages in response to landscape pattern. *Landscape Ecology*, **19**, 41–57.
- Cliff, A.D. & Ord, J.K. (1981) *Spatial Processes: Models and Applications*. Pion, London.
- Cushman, J.H. & Meentemeyer, R.K. (2005) The importance of humans in the dispersal and spread of *Phytophthora ramorum* at local, regional and landscape scales. *Proceedings of the 2nd Sudden Oak Death Science Symposium* (eds S.J. Frankel, P.J. Shea & M.I. Haverty), pp. 161–163. USDA FS PSW Research Station, Albany, CA.
- Daszak, P., Cunningham, A.A. & Hyatt, A.D. (2000) Emerging infectious diseases of wildlife – threats to biodiversity and human health. *Science*, **287**, 443–449.
- Davidson, J.M., Rizzo, D.M. & Garbelotto, M. (2002) *Phytophthora ramorum* and sudden Oak Death in California. II. Pathogen transmission and survival. *5th Symposium of California Oak Woodlands* (eds R. Standiford & D. McCreary), pp. 741–749. USDA Forest Service, General Technical PSW-GTR-184. USDA FS PSW Research Station, Albany, CA.
- 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**, 587–595.
- Denman, S., Kirk, S.A., Brasier, C.M., Barton, V.C., Hughes, K.J.D. & Webber, J.F. (2005) *Phytophthora ramorum* on *Quercus ilex* in the United Kingdom. *Plant Disease*, **89**, 1241.
- Dobson, A. & Foufopoulos, J. (2001) Emerging infectious pathogens of wildlife. *Philosophical Transactions of the Royal Society of London B*, **356**, 1001–1012.
- Dodd, R.S., Huberli, D., Douhovnikoff, V., Harnik, T.Y., Afzal-Rafii, Z. & Garbelotto, M. (2005) Is variation in susceptibility to *Phytophthora ramorum* correlated with population genetic structure in coast live oak (*Quercus agrifolia*)? *New Phytologist*, **165**, 203–214.
- Ehrenfeld, D. (2005) The environmental limits to globalization. *Conservation Biology*, **19**, 318–326.
- Ericson, L., Burdon, J.J. & Muller, W.J. (1999) Spatial and temporal dynamics of epidemics of the rust fungus *Uromyces valerianae* on populations of its host *Valeriana salina*. *Journal of Ecology*, **87**, 649–658.
- Erwin, D.C. & Ribeiro, O.K. (1996) *Phytophthora Diseases Worldwide*. The American Phytopathological Society, St. Paul, MN.
- Geary, R.C. (1954) The contiguity ratio and statistical mapping. *Incorporated Statistician*, **5**, 115–145.
- Gilbert, G.S. (2002) Evolutionary ecology of plant diseases in natural ecosystems. *Annual Review of Phytopathology*, **40**, 13–43.
- Goheen, E.M., Hansen, E.M., Kanaskie, A., McWilliams, M.G., Osterbauer, N. & Sutton, W. (2002) Sudden oak death caused by *Phytophthora ramorum* in Oregon. *Plant Disease*, **86**, 441.
- Goovaerts, P. (2000) Geostatistical approaches for incorporating elevation into the spatial interpolation of rainfall. *Journal of Hydrology*, **228**, 113–129.
- Graham, M.H. (2003) Confronting multicollinearity in ecological multiple regression. *Ecology*, **84**, 2809–2815.
- Gustafson, E.J. (1998) Quantifying landscape spatial pattern: what is the state of the art? *Ecosystems*, **1**, 143–156.
- Haila, Y. (2002) A conceptual genealogy of fragmentation research: from island biogeography to landscape ecology. *Ecological Applications*, **12**, 321–334.
- Hanski, I. & Simberloff, D. (1997) The metapopulation approach, its history, conceptual domain, and application to conservation. *Metapopulation Biology* (eds I.A. Hanski & M.E. Gilpin), pp. 5–26. Academic Press, New York.
- Hargis, C.D., Bissonette, J.A. & David, J.L. (1998) The behavior of landscape metrics commonly used in the study of habitat fragmentation. *Landscape Ecology*, **13**, 167–186.
- Harvell, C.D., Mitchell, C.E., Ward, J.R., Altizer, S., Dobson, A.P., Ostfeld, R.S. & Samuel, M.D. (2002) Climate warming and disease risks for marine biota. *Science*, **296**, 2158–2162.
- Holdenrieder, O., Pautasso, M., Weisberg, P.J. & Lonsdale, D. (2004) Tree diseases and landscape processes: the challenge of landscape pathology. *Trends in Ecology and Evolution*, **19**, 446–452.
- Holland, J.D., Bert, D.G. & Fahrig, L. (2004) Determining the spatial scale of species response to habitat. *Bioscience*, **54**, 227–233.
- Hurlbert, S.H. (1984) Pseudoreplication and the design of ecological field experiments. *Ecological Monographs*, **54**, 187–211.
- Ivors, K., Garbelotto, M., Vries, I.D.E., Ruyter-Spira, C., Hekkert, T.E., Rosenzweig, N. & Bonants, P. (2006) Microsatellite markers identify three lineages of *Phytophthora ramorum* in US nurseries, yet single lineages in US forest and European nursery populations. *Molecular Ecology*, **15**, 1493–1505.
- Jerger, M.J. (1999) Improved understanding of dispersal in crop pest and disease management: current status and future directions. *Agricultural and Forest Meteorology*, **97**, 331–349.
- Jules, E.S., Kauffman, M.J., Ritts, W.D. & Carroll, A.L. (2002) Spread of an invasive pathogen over a variable landscape: a nonnative root rot on Port Orford cedar. *Ecology*, **82**, 3167–3181.
- Keesing, F., Holt, R.D. & Ostfeld, R.S. (2006) Effects of species diversity on disease risk. *Ecology Letters*, **9**, 485–498.
- Kelly, M. & Meentemeyer, R.K. (2002) Landscape dynamics of the spread of Sudden Oak Death. *Photogrammetric Engineering and Remote Sensing*, **68**, 1001–1009.
- Levins, R. (1969) Some demographic and genetic consequences of environmental heterogeneity for biological control. *Bulletin of the Entomological Society of America*, **15**, 237–240.
- Li, H. & Reynolds, J.F. (1995) On definition and quantification of heterogeneity. *Oikos*, **73**, 280–284.
- Li, H. & Wu, J. (2004) Use and misuse of landscape indices. *Landscape Ecology*, **19**, 389–399.
- Lundquist, J.E. & Klopfenstein, N.B. (2001) Integrating concepts of landscape ecology with the molecular biology of forest pathogens. *Forest Ecology and Management*, **150**, 213–222.
- MacArthur, R.H. & Wilson, E.O. (1967) *The Theory of Island Biogeography*. Princeton University Press, Princeton, NJ.
- Mayer, J.D. (2000) Geography, ecology and emerging infectious diseases. *Social Science and Medicine*, **50**, 937–952.
- McGarigal, K., Cushman, S.A., Neel, M.C. & Ene, E. (2002) *FRAGSTATS: Spatial Pattern Analysis Program for Categorical Maps*. Computer Software Program Produced by the Authors at the University of Massachusetts, Amherst. Available at: [www.umass.edu/landeco/research/fragstats/fragstats.html](http://www.umass.edu/landeco/research/fragstats/fragstats.html).
- McGarigal, K. & McComb, W.C. (1995) Relationships between landscape structure and breeding birds in the Oregon Coast Range. *Ecological Monographs*, **65**, 235–260.
- Meentemeyer, R.K., Rizzo, D., Mark, W. & Lotz, E. (2004) Mapping the risk of establishment and spread of sudden oak death in California. *Forest Ecology and Management*, **200**, 195–214.
- Moritz, M.A. & Odion, D.C. (2005) Examining the strength and possible causes of the relationship between fire history and Sudden Oak Death. *Oecologia*, **144**, 106–114.
- Orwig, D.A. (2002) Ecosystem to regional impacts of introduced pests and pathogens: historical context, questions and issues. *Journal of Biogeography*, **29**, 1471–1474.
- Ostfeld, R.S., Glass, G.E. & Keesing, F. (2005) Spatial epidemiology: an emerging (or re-emerging) discipline. *Trends in Ecology and Evolution*, **20**, 328–336.

- Pautasso, M., Holdenrieder, O. & Stenlid, J. (2005) Susceptibility to fungal pathogens of forests differing in tree diversity. *Forest Diversity and Function: Temperate and Boreal Systems, Ecological Studies*, Vol. 176 (eds M. Scherer-Lorenzen, Ch. Körner & E.-D. Schulze), pp. 263–289. Springer, New York.
- Perry, D.A. (1988) Landscape pattern and forest pests. *North-west Environmental Journal*, **4**, 213–228.
- Quinn, G.P. & Keough, M.J. (2002) *Experimental Design and Data Analysis for Biologists*. Cambridge University Press, Cambridge, UK.
- Riitters, K.H., O'Neill, R.V., Hunsaker, C.T., Wickham, J.D., Yankee, D.H., Timmins, S.P., Jones, K.B. & Jackson, B.L. (1995) A factor analysis of landscape pattern and structure metrics. *Landscape Ecology*, **10**, 23–39.
- Rizzo, D.M. & Garbelotto, M. (2003) Sudden oak death: endangering California and Oregon forest ecosystems. *Frontiers in Ecology and the Environment*, **1**, 197–204.
- Rizzo, D.M., Garbelotto, M., Davidson, J.M., Slaughter, G.S. & Koike, S.T. (2002) *Phytophthora ramorum* as the cause of extensive mortality of *Quercus* spp. and *Lithocarpus densiflorus* in California. *Plant Disease*, **86**, 205–214.
- Rizzo, D.M., Garbelotto, M. & Hansen, E.M. (2005) *Phytophthora ramorum*: integrative research and management of an emerging pathogen in California and Oregon forests. *Annual Review of Phytopathology*, **43**, 309–335.
- Roland, J. & Taylor, P.D. (1997) Insect parasitoid species respond to forest structure at different spatial scales. *Nature*, **386**, 710–713.
- Soule, M.E. & Simberloff, D. (1986) What do genetics and ecology tell us about the design of nature reserves? *Biological Conservation*, **35**, 19–40.
- Swiecki, T.J. & Bernhardt, E. (2002) Evaluation of stem water potential and other tree and stand variables as risk factors for *Phytophthora ramorum* canker development in coast live oak. *5th Symposium of California Oak Woodlands* (eds R. Standiford & D. McCreary), pp. 787–798. USDA FS PSW Research Station, Albany, CA.
- Thrall, P.H., Godfree, R. & Burdon, J.J. (2003) Influence of spatial structure on pathogen colonization and extinction: a test using an experimental metapopulation. *Plant Pathology*, **52**, 350–361.
- Tilman, D. & Kareiva, P. (1997) *Spatial Ecology: the Role of Space in Population Dynamics and Interspecific Interactions*, Preface. Princeton University Press, Princeton, NY.
- Wagner, H.H. & Fortin, M. (2005) Spatial analysis of landscapes: concepts and statistics. Special Feature. *Ecology*, **86**, 1975–1987.
- Waring, K.M. & O'Hara, K.L. (2005) Silvicultural strategies in forest ecosystems affected by introduced pests. *Forest Ecology and Management*, **209**, 27–41.
- Weiss, R.A. & McMichael, A.J. (2004) Social and environmental risk factors in the emergence of infectious diseases. *Nature Medicine Supplement*, **10**, S70–S76.
- Werres, S., Marwitz, R., Man in't Veld, W.A., De Cock, A.W.A.M., Bonants, P.J.M., De Weerd, M., Themann, K., Ilieva, E. & Baayen, R.P. (2001) *Phytophthora ramorum* sp. nov., a new pathogen on *Rhododendron* and *Viburnum*. *Mycological Research*, **105**, 1155–1165.
- Weste, G. & Marks, G.C. (1987) The biology of *Phytophthora cinnamomi* in Australasian forests. *Annual Review of Phytopathology*, **25**, 207–229.
- Wiens, J.A. (1989) Spatial scaling in ecology. *Functional Ecology*, **3**, 385–397.
- Wilcove, D.S., McLellan, C.H. & Dobson, A.P. (1986) Habitat fragmentation in the temperate zone. *Conservation Biology, the Science of Scarcity and Diversity* (ed. M.E. Soule), pp. 237–256. Sinaur Associates, Sunderland, MA.
- With, K.A. (2002) The landscape ecology of invasive spread. *Conservation Biology*, **15**, 1192–1203.
- Woods, A., Coates, K.D. & Hamann, A. (2005) Is an unprecedented *Dothistroma* needle blight epidemic related to climate change? *Bioscience*, **55**, 761–769.
- Zobel, D.B., Roth, L.F. & Hawk, G.M. (1985) *Ecology, Pathology, and Management of Port-Orford-Cedar (Chamaecyparis lawsoniana)*. USDA Forest Service. General Technical Report PNW-184. USDA PNW Forest and Range Experimental Station, Portland, OR.

Received 3 May 2006

revision accepted 4 October 2006

Handling Editor: Jeremy Burdon