Landscape Dynamics of the Spread of Sudden Oak Death

Maggi Kelly and Ross K. Meentemeyer

Abstract

*Sudden Oak Death is caused by a newly discovered virulent pathogen (*Phytophthora ramorum*) that is killing thousands of native oak trees in California. We present a landscape-scale study on the spatio-temporal dynamics of oak mortality. Second-order spatial point-pattern analysis techniques (Ripley's K) were applied to the distribution of dead tree crowns (derived from high-resolution imagery) in Marin County, California to determine the existence and scale of mortality clustering in two years (2000 and 2001). Both years showed clustering patterns between 100 and 300 m. A classification tree model was developed to predict spatial patterns of risk for oak mortality based on several landscape-scale variables. Proximity to forest edge was the most important explanatory factor, followed by topographic moisture index, proximity to trails, abundance of* Umbellularia californica*, and potential summer solar radiation. This research demonstrates the utility of integrating remotely sensed imagery analysis with geographic information systems and spatial modeling for understanding the dynamics of exotic species invasions.*

Introduction

Biological invasions are spreading at such a rapid pace that they are considered a major component of global and landscape-level environmental change (Vitousek *et al.*, 1996). Invasions are devastating ecological systems by reducing biodiversity, altering ecosystem processes, and functioning as vectors of disease (Parker *et al.*, 1999). The spread of non-native plant pathogens can have particularly strong influences on ecosystem dynamics due to their ability to directly kill host species, permanently changing genetic diversity and community structure. As such, the mechanisms underlying the dispersal of pathogens are critical issues being pursued from a variety of approaches and spatial scales (Thrall and Burdon, 1999).

Spatial pattern is one of the most fundamental properties of disease dynamics because it reflects the environmental forces acting on the dispersal and life cycles of a pathogen (Ristaino and Gumpertz, 2000). For this reason, researchers of plant disease epidemics are increasingly using landscape approaches to quantify and model spatial patterns of disease spread in order to understand the basic factors that influence pathogen dispersal and infection processes. Despite the strengths of a landscape approach, relatively few studies have developed spatial models of disease patterns in natural systems due to the challenge of integrating numerous, spatially referenced samples of disease incidence with digital maps describing spatial variations in environmental factors and plant community structure (Ristaino and Gumpertz, 2000; Lundquist and Klopfenstein, 2001).

We present a landscape-scale study of the spatio-temporal dynamics of Sudden Oak Death (SOD), an alarming disease that is killing thousands of native oak trees and tanoaks in the Coast Ranges of California and southwestern Oregon (McPherson *et al.*, 2000; Garbelotto *et al.*, 2001). Considerable progress has been made in the past year identifying host species and understanding how this disease is related to other tree-killing diseases (Davidson *et al.*, 2001; Davidson *et al.*, 2002; Rizzo *et al.*, 2002). However, we still do not know how the disease extends its range across California's landscape, and we have no ability to predict which stands of trees are at high risk of infection. The objectives of this research are to

- Determine the distribution and rate of oak mortality in the study area;
- Determine the scale and extent to which the oak mortality is spatially clustered;
- Determine the relative importance and combined effects of critical landscape factors governing the spatial pattern and spread of oak mortality; and
- Develop a risk model for Sudden Oak Death in Marin County, California.

Sudden Oak Death is caused by a newly discovered virulent pathogen (*Phytophthora ramorum*) that was most likely introduced to California from abroad (Rizzo *et al.*, 2001). The pathogen kills several keystone tree species in California ecosystems including tanoak (*Lithocarpus densiflorus*), coast live oak (*Quercus agrifolia*), and black oak (*Q. kelloggii*). The pathogen also colonizes the foliage of several other overstory and understory hosts without killing them (Rizzo *et al.*, 2002). These species include California bay laurel (*Umbellularia californica*), California rhododendron (*Rhododendron macrophyllum*), huckleberry (*Vaccinium ovatum*), madrone (*Arbutus menziesii*), California buckeye (*Aesculus californica*), bigleaf maple (*Acer macrophyllum*), and manzanita (*Arctostaphylos manzanita*).

The disease has been officially confirmed in ten coastal counties of California (Figure 1), and known hosts for the disease exist in many more counties across the state. It has also been detected on 16.2 ha in southern Oregon and is known to cause leafspots and twig dieback on rhododendron in Germany and The Netherlands. Marin County, California is one of the

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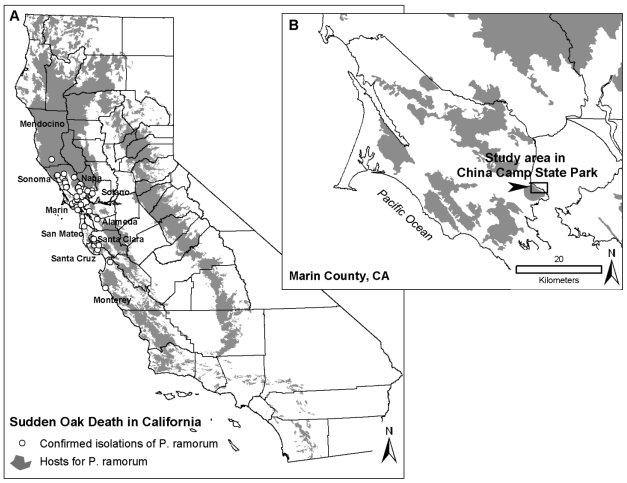

Figure 1. Sudden Oak Death in California. (A) distribution of susceptible host species and locations of positive isolations of *P. ramorum*. (B) Marin County, showing the study area and the possible range of host species within the County.

"hot-spots" for SOD. Throughout the County (and central coastal area of the state) dramatic dieback of tanoaks, coast live oaks, and black oaks is occurring, presenting serious threats to alter the ecology, wildlife habitat, soil erosion properties, fire regime, and aesthetic value of thousands of hectares of coastal forest (McPherson *et al.*, 2000; Garbelotto *et al.*, 2001).

Little is still known about mechanisms driving the dispersal of the disease, but preliminary evidence suggests an aerial phase (Davidson *et al.*, 2001; Davidson *et al.*, 2002) in possible combination with efficient long-range vectors, high levels of inoculum production, and high levels of virulence (Swiecki, 2001). It is currently believed that the oaks are terminal hosts (meaning the pathogen will not emerge from the tree to infect other trees) with spread occurring primarily from one or more of the foliar hosts (i.e., *Umbellularia californica*) (Davidson *et al.*, 2001; Davidson *et al.*, 2002). Foliar hosts are plants that contract a form of the disease that remains for the most part on the leaves, and does not infect the main stem of the plant. These foliar hosts are suspected to be the most durable and persistent source of the inoculum, and during wind and rain events the fungus can be dispersed to infect new trees (Davidson *et al.*, 2002). Low tree water stress and the amount of tree canopy cover may also contribute to disease occurrence (Swiecki and Bernhardt, 2002).

This work builds on previous efforts that have mapped dead and dying oaks in a study area in Marin County, California using high-resolution imagery (Kelly, 2002). We integrate cases of oak mortality derived from remote sensing with spatially distributed environmental variables to study spatial clusters of oak mortality and model interacting factors of disease presence and spread over the landscape. It is important to note here that we are assuming that, in the study area, the overstory mortality we see from remotely sensed imagery is due to the pathogen *P. ramorum*.

Study Area

The study area for this project is within the area defined by China Camp State Park (38.0°N, 122.5°W), a forested peninsula on the east side of Marin County (Figure 1). The study area is the north-western portion of the Park, covering 370 ha (of which 210 ha are forested). It generally faces north, and ranges from sea level at San Francisco Bay to about 260 m. This mixed hardwood forest contains near even-age stands; these hillsides were cleared for lumber in the early to mid-1800s. All of the woody plants can serve as host for *Phytophthora ramorum* in these forests with the exception of valley oak (Rizzo *et al.*, 2002). Forest overstory is primarily coast live oak but can also include black oak, bay laurel, and madrone. Buckeye and manzanita occur at the margins of closed canopy (Rizzo *et al.*, 2002).

Method

Database Development

Remote Sensing of Oak Mortality

The use of remotely sensed data for monitoring forest health and forest inventories has a strong history in conifer stands, but less work has been done in hardwood forests (Boyer *et al.*, 1988; Everitt *et al.*, 1999; Gong *et al.*, 1999). The pathology of this new disease affords opportunities for continued development of techniques for remote sensing in hardwood forests, as well as characterization of the disease at a landscape scale. The disease has three characteristics that make a monitoring approach that uses remote sensing useful. First, as the trees with the disease die, in most cases the entire crown changes dramatically from healthy green to brown, and over a short time period (Rizzo *et al.*, 2002). Second, after canopy change has occurred, the leaves can stay adhered to the branches for months, giving trees a characteristic "freeze-dried" appearance. Third, the affected *Quercus* species make good targets for high-resolution imagery because the coast live oaks have a broad multi-stem canopy (Pavlik *et al.*, 1991), and the disease seems to disproportionately affect overstory trees (Swiecki and Bernhardt, 2002). Classification of dead and dying trees can be used in spatial pattern analysis to understand landscape structure of oak mortality.

Digital imagery (ADAR5500) was acquired for the China Camp study area on 30 March 2000 and 05 May 2001 with an ADAR5500 imaging system that was comprised of an SN4 20-mm lens with four mounted cameras (Spectral Bands: Blue: 450 to 550 nm; Green: 520 to 610 nm; Red: 610 to 700 nm; Near-Infrared (NIR): 780 to 920 nm), flown at an average aircraft altitude of 2,205 m. Imagery was acquired near noon on both dates, in clear-sky conditions, with comparable solar elevation and zenith angles (solar elevation for 2000 = 53.53°, and solar elevation for 2001 = 58.65°). A contractor performed imagery acquisition, image mosaicking and registration. The average ground instantaneous field of view of the images is 1 meter. Near anniversary dates were chosen to maximize the springtime canopy cover changes associated with SOD, and to minimize misclassification caused by early color change of California buckeye, a summer drought deciduous tree that can appear similar to SOD-affected trees when seen from a distance.

The 2000 imagery was classified for dead and dying crowns using a combination of unsupervised classifications (ISODATA clustering), spectral enhancements (NDVI and PCA), spatial search, and manual methods (Kelly, 2002). The accuracy of this classification was 92 percent (Kelly, 2002). A normalized difference vegetation index (NDVI) was then calculated for each date using the standard formula (NIR − Red)/(NIR + Red). No corrections were applied to the raw imagery before NDVI calculation, because the images were collected at near anniversary dates, with clear sky conditions on both days. The 2001 imagery was used with 2000 imagery in an NDVI image subtraction and thresholding procedure that yielded dead and dying tree crowns for the study area for the year beginning April 2000. Centroids of dead and dying crowns were retained for use in point-pattern analysis. In the study area, 1,086 dead crowns (about 5 trees per forested ha) were found in spring

2000, and 1,394 dead crowns (about 7 trees per forested ha) were found in 2001.

A combination of supervised classification (minimum-distance classifier) and manual determination was also performed to locate centers of overstory bay trees. The accuracy of this map was not assessed. A forest land-cover class was derived from an unsupervised classification routine. This image was smoothed using several iterations of mean filters to remove individual pixels classified as bare, and the result is used to show the general forested area in Figure 2. This area was used to compare the distribution of dead and dying trees in 2000 and 2001 with like numbers of randomly located live trees ($n_{2000} = 1086$ and $n_{2001} = 1394$). Finally, park trails were manually digitized from a 1:6,000-scale map of trails used for orienteering.

Derivation of Landscape Predictor Variables

Eight landscape variables were derived for modeling relationships between the environment and spatial patterns in oak mortality incidence. Five of the variables were derived from a high-resolution (5-m) digital elevation model (DEM) produced for Marin County using photogrammetric methods. These include elevation, slope gradient, topographic moisture index (TMI), and potential solar insolation over the winter (PSIw) and summer (PSIs). The remaining three variables include the density of *Umbellularia californica* per ha (BAY), distance to the forest edge (EDGE), and distance to the nearest trail (TRAIL).

The topographic moisture index (TMI) (Beven and Kirkby, 1979) was computed to characterize topographic effects on potential soil moisture distribution. TMI is the natural log of the ratio between upslope drainage area, a (m^2) and the slope gradient of a given grid cell, b (Moore *et al.*, 1991): i.e.,

$$\text{TMI} = \ln(a/\tan b).$$

The equation in this form assumes uniform soil transmissivity across the study area. Locations with small upslope drainage areas (e.g., ridges) have lower TMI values than do sites with large upslope areas (e.g., toeslopes and drainages). Given constant upslope area, steep slopes have lower TMI values than do gentle slopes. Potential direct beam solar insolation (PSI) was calculated for each grid cell in the DEM using the cosine of the illumination angle on slope (Dubayah, 1994): i.e.,

$$\text{PSI} = \cos\theta\cos S + \sin\theta\sin S\cos(\Phi - A)$$

where θ is the solar zenith angle, Φ is the solar azimuth, S is the slope of the terrain, and A is the aspect of the slope. Both S and A are derived from the digital elevation model. The PSIw was calculated as the average daily PSI for winter (21 December to 21 March), and PSIs was calculated as the average daily PSI for summer (21 June to 21 September 21). The index is rescaled from 0 to 255 (low to high potential solar insolation). Examples of six of these variables are found in Plate 1.

Analysis

Spatial Pattern of Oak Mortality

The rates of oak mortality spread were examined by comparing the numbers of dead crowns in 2000 with the number of dead crowns in 2001. We also examined and tested the significance of the pattern (distance and direction) of oak mortality spread between the two years. The distribution of distances from each dead tree in 2001 to its nearest dead neighbor in 2000 was compared to the distribution of distances from each dead tree in 2001 to its nearest random live neighbor using an unpaired t-test. The bearings associated with these shortest distance were also examined, and Raleigh's (Batschelet, 1981) statistic (R_{dead} for bearings from 2001 to 2000, and R_{random} for bearings from

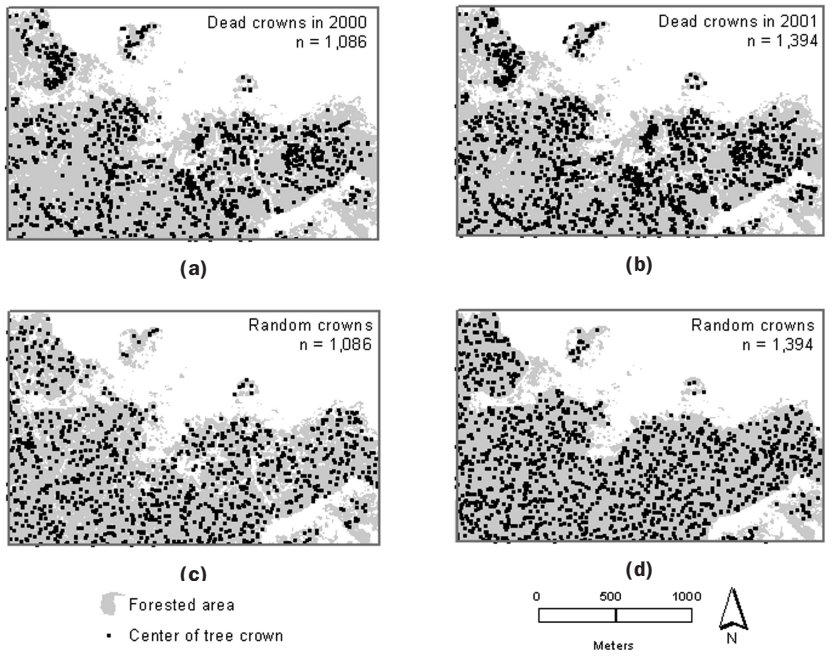

Figure 2. Distribution of dead and dying trees and randomly located live trees used to test spatial pattern significance. (a) Dead crowns in 2000. (b) Dead crowns in 2001. (c) 1,086 randomly located live trees. (d) 1,394 randomly located live trees.

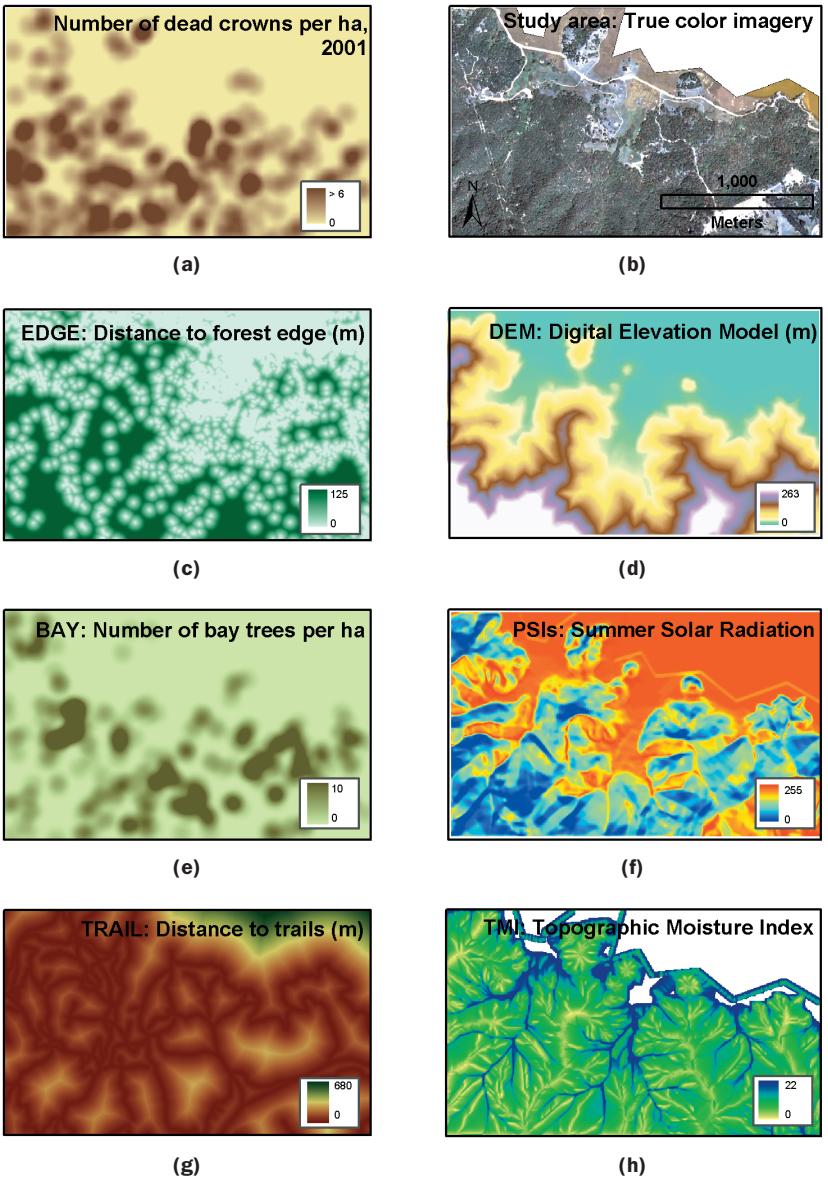

Plate 1. Derived environmental variables used in classification tree analysis. The clustering of dead crowns is shown in (a), and the study area from high-resolution imagery shown in true color (b) is presented for comparison. In (b) dark green areas are mixed oak woodlands affected with SOD; light areas indicate roads, paths, and gaps in forest cover. (c) and (e) are derived from high-resolution imagery (b). Potential Solar Insolation in the summer (f) and topographic moisture index (h) are derived from Digital Elevation Model (d). In (h) yellows indicate ridges, and blues indicate drainages.

2001 to random) were tested for randomness (Batschelet, 1981; Kelly, 2000). Raleigh's test for circular distribution involves using the sum of the cosine of each bearing and the sum of the sine of each bearing to derive a mean angle, theta, and a normalized vector length that can be compared to a normal circular distribution function. Random distributions of bearings yield very small normalized vector lengths (Batschelet, 1981).

Second-order spatial point-pattern analysis techniques (Ripley's K) were applied to the distribution of dead and dying trees in 2000 and to the total dead trees in 2001 to determine the effective distance of dispersal mechanisms for this disease. Ripley's K statistic (Ripley, 1976; Ripley, 1981) allows for testing of complete spatial randomness and is defined as the expected number of individuals within a distance t of a randomly chosen individual in a population (Ripley, 1976; Kenkel, 1988; Cressie, 1993; Kenkel, 1994;). The method has been used to study mortality patterns and tree interactions in numerous systems (Andersen, 1992; Szwagrzyk and Czerwczak, 1993; Vacek and Leps, 1996; Cole and Syms, 1999; Eccles *et al.*, 1999; Goreaud and Pelissier, 1999), and can be followed by analyzing environmental factors possibly controlling the existence and spread of disease (Szwagrzyk and Czerwczak, 1993).

We calculated Ripley's K and Lhat (transformed K) for distances between 10 and 1000 m at 10-m intervals. Upper and lower limits for significance were determined by Monte Carlo simulations (100 binomial simulations to determine upper and lower bounds of significance). Because Ripley's K can be sensitive to edges (Haase, 1995; Goreaud and Pelissier, 1999), we did not analyze the outer 20 m of the study area. This reduced the bias that could be introduced by the many clusters of trees at the north end of the area.

Risk Model Development and Validation

Relationships between the occurrence of biological organisms and landscape factors are often non-monotonic and involve complex interactions (Austin *et al.*, 1990; Franklin, 1995). As an alternative to logistic regression, we used classification trees (De'ath and Fabricius, 2000) to model relationships between oak mortality incidence and landscape variables due to the method's ability to capture hierarchical and nonlinear relationships and expose interactions among predictor variables (Clark and Pregibon, 1993; Michaelsen *et al.*, 1994; De'ath and Fabricius, 2000). The response variable included 1,075 remotely sensed observations of SOD presence at time period 2000 and 1,075 randomly distributed observations of disease absence. The observations of oak mortality incidence were then merged with the corresponding cell values of each landscape variable for model development. We did not space the samples at distances greater than the clustering distance indicated by the Ripley's K statistic because the study area is not large enough to allow this spacing of samples and obtain a sufficient number of samples for model development and validation. We are currently extending this work to larger areas, which will enable us to better examine the effect of spatial autocorrelation on model development.

Classification tree models are developed by recursively partitioning the response variable into increasingly homogeneous subsets based on critical thresholds in continuous or categorical variables. The dataset is partitioned on the predictor variable, which upon splitting at some break point yields the greatest reduction in the error sum of squares for the response variable. Tree-based models are typically graphically displayed so that one can follow the tree node (*root*), through a series of binary splits on the predictor variables (*branches*), to an end node (*leaf*) (e.g., see Figure 4). The predicted value at each end node is the mean value of all observations that flow through the tree to that node. The estimate for all observations that follow the same lineage of branchings from root to a given leaf is given by the mean x-value for that set of observations.

To avoid over-fitting the tree model, an iterative cross-validation procedure was used that identifies an optimal tree size beyond which validation performance drops as additional branches "grow" in response to peculiarities in the development data, but fail to account for variance in the test data. The point at which this occurs suggested the appropriate number of terminal nodes for developing the tree from the entire dataset, and the classification tree is "pruned" to this optimum size (Davis *et al.*, 1990; Clark and Pregibon, 1993).

The resulting model was then applied to the landscape variables in the GIS to predict spatial variations in the probability (or risk) of oak mortality incidence. Model performance was evaluated by comparing the predictive risk map to an independent dataset that describes where the oak mortality had (n = 687) and had not (n = 687) spread to in year 2001. Finally, we calculated the mean error (MER) in order to identify tendencies of under- versus over-prediction: i.e.,

$$\text{MER} = 1/n \sum_{i=1}^{n} (P_i - O_i)$$

where n is the number of samples, P_i is the predicted probability of presence rounded to 0 (absent) or 1 (present), and O_i is observed incidence (0 or 1). Positive and negative MER values indicate over- and under-prediction, respectively.

Results

Spatial Pattern of Oak Mortality

We calculated an annual rate of mortality of about 216 per year or just over one tree per ha using the mortality derived from remotely sensed imagery.

The results of the unpaired t-test between the distribution of distances from each tree in 2001 to its nearest dead neighbor in 2000, and from each tree in 2001 to its nearest neighbor from the population of 1,086 randomly located trees indicated that the distributions are not significantly different at $\alpha = 0.05$ ($t = 1.65$).

The bearings of the shortest distance vector between a dead crown in 2001 and its closest dead neighbor in 2000, and those between a dead crown in 2001 and a random tree, were also found to be randomly distributed. The mean angle, theta, and normalized vector length, R, were smaller than the test statistic for $\alpha = 0.05$ in both cases (bearings from 2001 to 2000, and from 2001 to random), and the null hypothesis for randomness was accepted for both cases ($R_{dead} = 0.006$, $R_{random} = 0.002$).

Ripley's K analysis was performed on the distribution of dead and dying trees in 2000, and the dead and dying crowns through 2001. Monte Carlo simulations were used to compare observed distributions to those randomly selected from the underlying tree pattern. Both years showed clustering patterns across similar spatial scales. The distribution of dead crowns in 2000 shows clear evidence of clumping between 100 to 300 m (Figure 3a). From 500 to 1000 m, the pattern is closer to random, but still significantly clustered. The distribution of dead trees through 2001 shows significant clustering between 100 and 300 m, and from 700 m to 900 m. From 300 to 700 m, the pattern is closer to random but still clustered (Figure 3b).

Classification Tree Model of Oak Mortality Risk

The classification tree model (Figure 4) was developed using mortality information from 2000, and tested with tree mortality data from 2001. The model explains 63 percent of the variability in the location of oak mortality when evaluated with the data used for model development (2000). Using the test data (from 2001), the risk model corresponded to 58 percent of the spread that occurred in 2001 with a slight tendency to overpredict (MER = 0.20).

The model is structured such that there are five levels and 11 end node predictions of risk (Figure 4). Every site has some degree of infection risk, ranging from 0.16 to 0.87. Distance to forest edge, on average, explains the most variance (37 percent) (deviance explained by each variable divided by the total deviance of the model) in oak mortality incidence, followed by topographic moisture index (22 percent), distance to the nearest trail (17 percent), density of *U. californica* (12 percent), and potential summer solar radiation (12 percent). Trees within 6.1 m of forest edges are at considerably higher risk (0.82) than trees further than 6.1 m (0.45) (Figure 4). Of all combinations of landscape conditions, SOD risk is greatest for trees that are relatively close to forest edges (EDGE $<$ 6.1 m), occur in association with *U. californica* (BAY $>$ 1), and are within 76 m of a trail (TRAIL $<$ 76 m).

The proximity of a tree to the edge of forest (EDGE $<$ 6 m) was the most important factor explaining oak mortality presence, followed by density of bay trees (BAY $>$ 1), and proximity to trails (TRAIL $<$ 76 m). The lowest risk for oak mortality occurs for trees away from forest edges (EDGE $>$ 9.8 m), on topographically moist slopes (TMI $>$ 2.56), and with high summer solar insolation levels (PSIs $>$ 202). Intermediate levels of oak

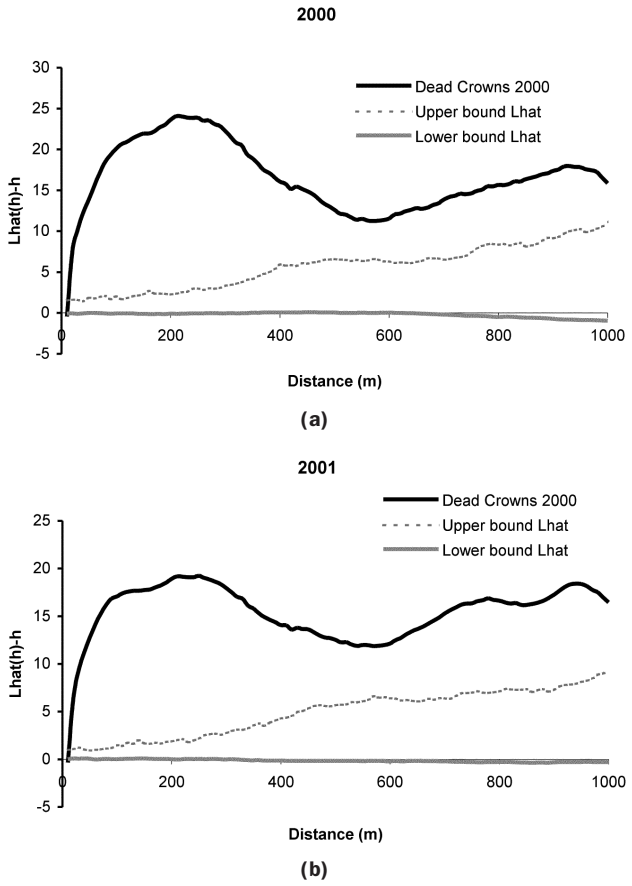

Figure 3. Diagram of the Lhat(h)-h transformation for dead crowns in 2000 (a) and 2001 (b) showing upper and lower bounds of the function. Results above the upper limit display clustering; between the upper and lower limits display random pattern; below the lower limit results display regularity.

mortality risk generally occur on drier slopes (TMI $<$ 2.56), increasing with increasing density of *U. californica* (BAY $>$ 1.7 bay per ha), decreasing distance to forest edge (EDGE $<$ 9.6 m), and increasing summer solar radiation (PSIs $>$ 59). The split on TMI (at 2.56) generally distinguishes toeslopes and ephemeral drainages from upper hillslopes in this environment, and indicates that oak mortality risk is lower on the topographically wetter toeslopes and drainages. With the exception of proximity to trails (TRAIL), the direction and the effect of each variable on oak mortality risk is the same at all levels of the tree (Figure 4). For example, greater risk is associated with higher densities of *U. californica* regardless of how a far a tree is from forest edges. The variable PSIw was dropped from the classification tree in the tree "pruning" process, indicating that this variable was not significant in explaining the distribution of oak mortality.

Discussion

Spatial Pattern of Oak Mortality

It is clear that mortality occurs throughout the study area. Determining the rates of disease spread in the area requires assumptions. We assumed that the disease appeared there in 1995 and that, at that time, background mortality was zero. We also used two simple functions to estimate rates. Neither of these functions, linear or polynomial, are likely adequate because pathogen dispersal may occur in waves during the most suitable weather conditions (Rizzo *et al.*, 2002) and it is also likely that mortality will decrease in years to come due to some genetic resistance.

The analysis of oak mortality spread from 2000 to 2001 indicates that dead trees in 2001 are not more likely to be closer to existing dead trees than to randomly located crowns. New mortality occurs from 0.5 to over 100 m from existing dead trees. The pattern analysis presented here suggests that the clustering of mortality, while pronounced, cannot be accounted for by a directional control, as might be expected with a dominant aerial phase to disease spread that incorporates wind. More clues to the possible controls or risk factors for the disease come from the classification tree analysis.

The pattern and scale of mortality in a forested system can lend insight into the processes controlling associated diseases, and the scales and at which they operate (Cole and Syms, 1999). It has been shown that small scale ($<$ 15-m) pattern of healthy trees in forests is regular to random (Szwagrzyk and Czerwczak, 1993) and that, absent epidemics, mortality across scales is often random in pattern (Eccles *et al.*, 1999). In our study area at very small scales (below 10 m), tree mortality appears random. However, second-order pattern analysis indicates strong clumping of tree mortality in both years across multiple scales. In both years, mortality clusters (100 to 300 m) correspond to the small clusters of mortality evident across the study area visible in Figures 2a and 2b. In 2001, clustering appears stronger than in 2000 at larger scales (700 to 900 m), which is possibly related to the fact that dead trees in 2001 are not statistically closer to dead trees in 2000 than to random trees, and that, as the disease spreads, larger clusters are being formed. The overall spatial clustering is commensurate with the understanding that this is an epidemic and so does not conform to the random mortality hypothesis, but rather, the disease presence is controlled by other environmental factors. The spatial scale of clustering found in this research has important implications for determining plot sizes and sampling designs for future epidemiological research on Sudden Oak Death. Individual plots much smaller than 1 ha in size may not capture spatial patterning occurring at broader scales.

Classification Tree Model of Disease Risk

Classification tree modeling provided a useful approach to examine interrelationships among environmental factors controlling oak mortality spread. The analysis reveals that trees within or near the forest edge, near bay trees, and near trails have a high risk of mortality. The forest edge is often where understory foliar hosts such as buckeye and manzanita are common, so these results further strengthen the current hypothesis that foliar hosts are the most important risk factor in disease spread (Davidson *et al.*, 2002). Forest edges might be a useful surrogate for presence of understory foliar hosts. These understory shrubs are not visible using remote sensing, and so a surrogate for their presence would be very useful in disease modeling.

The topographic moisture result deviates from Swiecki and Bernhardt (2002) who found higher risk for disease on moister slopes in a study area in another part of Marin County. These discrepancies might be the result of analysis in differing forest types, or it could indicate that our study area should be broadened to include more examples of disease over a variety of slopes and soil moisture conditions.

Much concern has been expressed among state agencies about the possible role of humans in spreading the disease. The first locations of disease confirmation were in parks throughout the coastal area of the state that have extremely high numbers of hiking and biking visitors (i.e., Pfieffer Big Sur in the south, Sugarloaf and Jack London State Park in the north). Because the pathogen has been isolated from soil material (Davidson *et*

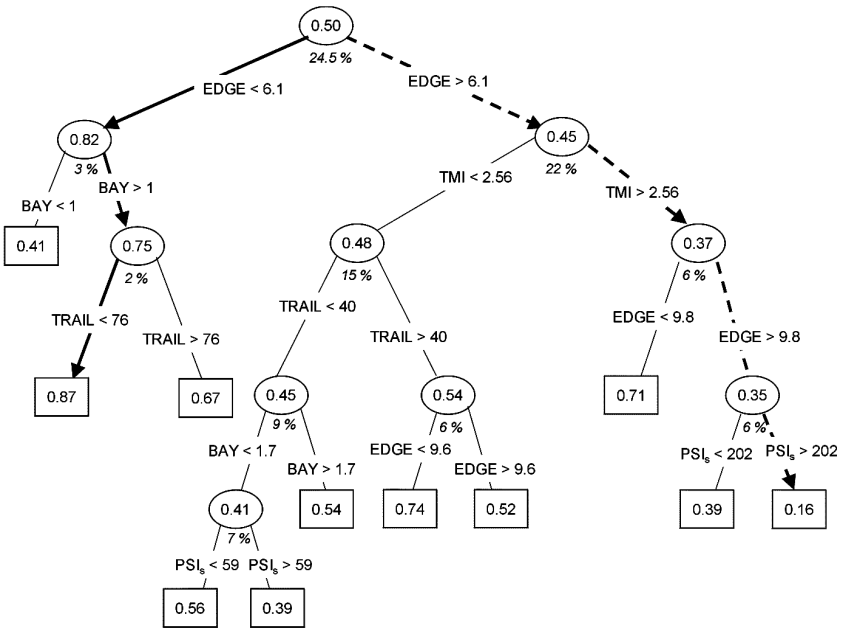

Figure 4. Classification tree model of disease risk. Ovals and squares represent non-terminal and terminal nodes, respectively. The values inside the ovals and squares are the predicted probabilities (means) of oak mortality presence. The values beneath the predictions indicate the proportion of the total deviance that each split explains. Critical thresholds are displayed between the node connections, splits that provide the basis for calculating predictions. The variable abbreviations are EDGE = distance to forest edge (m), TRAIL = distance to the nearest trail (m), BAY = density of *U. californica* per ha, TMI = topographic moisture index, and PSIS = average potential summer solar radiation. The dark solid arrows indicate the combination of environmental conditions associated with maximum risk for mortality. The dashed arrow indicates the combination of environmental conditions associated with the lowest risk of disease.

al., 2002), this recreational activity has often been proposed as a possible vector to spread the disease within parks and between parks and other areas. Some caution should be taken here, however. It is not clear that *P. ramorum* isolated from soil is viable as inoculum (Davidson *et al.*, 2002), nor is it clear from this study that proximity to trails is the most convincing explanation of spread. While trees within 76 m of trails are at higher risk, trees within 40 m of a trail are at lower risk (following the medium risk trajectory). More important risk factors do not indicate a human vector, but are associated with forest structure.

The model has a slight tendency to overpredict oak mortality occurrence. This might be a result of the stage of the disease, and it is possible that the mortality will spread to the areas identified by the model in later years. Genetic resistance must also be considered as an explanation for overprediction. While some research has begun on the possible genetic resistance of individual *Quercus* to the disease, little is known now on the subject apart from the observation that some individuals appear to be resistant. The disease has been in the County for several years, and is well established. For this reason, models developed here might be less applicable in other areas in which the disease is just beginning to spread. It is very likely that early infection patterns differ greatly from those of mature diseases. It is also possible that the spatial pattern of and controls on the disease vary with forest type.

Conclusions

China Camp State Park in Marin County is a known "hot-spot" for Sudden Oak Death (McPherson *et al.*, 2002; Rizzo *et al.*, 2002). This work quantifies the scale at which oak mortality clusters, and builds a model for understanding the landscape controls on mortality.

The current rate of mortality is 216 trees per year. Measurement of actual numbers of tree mortality per year might be facilitated through analysis of historical photography. According to simple distance measurements, the disease does not seem to spread from tree to tree (this conforms to current thinking that oaks are a terminal host for the disease), nor is there a prevailing directional component to the spread.

Clustering of mortality is pronounced in each year observed. Overstory tree mortality in the study area in both years occurs in clusters from 100 to 300 m in size, and in 2001 the mortality also exists in clusters of 700 to 900 m in size.

In the study area, the proximity of a tree to the forest edge (< 6 m) was the most important factor explaining mortality presence, followed by density to bay trees, and proximity to trails. Lowest risk for mortality was found away from forest edges, on moister slopes, and with high summer solar radiation.

This work provides a model of the risk factors associated with the disease that can be used to map areas at risk for Sudden Oak Death throughout Marin County. Further model calibration will continue over larger areas, and in different forests to evaluate these issues.

The integration of remote sensing with GIS and spatial modeling allows for mapping and understanding of disease spread across large areas. It provides a tool to "scale up" data derived from small plots, and to see spatial patterns and relationships not readily apparent in smaller plots (< 1 ha). The disease progression makes a remote sensing approach useful for mapping dead and dying overstory crowns. Remote sensing

also provides a method to locate forest edges for use as surrogates for edge-related foliar hosts. Still, there is more work needed here, including the analysis of hyperspectral imagery to identify stressed trees before the canopy changes, and risk mapping methods that incorporate understory foliar hosts, as well as genetic resistance to the disease.

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