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Predicting potential and actual distribution of sudden oak death in Oregon: Prioritizing landscape contexts for early detection and eradication of disease outbreaks

Tomáš Václavík[a,*], Alan Kanaskie[b], Everett M. Hansen[c], Janet L. Ohmann[d], Ross K. Meentemeyer[a]

[a] Center for Applied GIScience, Department of Geography and Earth Sciences, University of North Carolina at Charlotte, 9201 University City Blvd, Charlotte, NC 28223, United States
[b] Oregon Department of Forestry, 2600 State Street, Salem, OR 97310, United States
[c] Department of Botany and Plant Pathology, Oregon State University, Cordley Hall 2082, Corvallis, OR 97331-2902, United States
[d] Pacific Northwest Research Station, USDA Forest Service, 3200 SW Jefferson Way, Corvallis, OR 97331, United States

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ABSTRACT

An isolated outbreak of the emerging forest disease sudden oak death was discovered in Oregon forests in 2001. Despite considerable control efforts, disease continues to spread from the introduction site due to slow and incomplete detection and eradication. Annual field surveys and laboratory tests between 2001 and 2009 confirmed a total of 802 infested locations. Here, we apply two invasive species distribution models (iSDMs) of sudden oak death establishment and spread risk to target early detection and control further disease spread in Oregon forests. The goal was to develop (1) a model of *potential distribution* that estimates the level and spatial variability of disease establishment and spread risk for western Oregon, and (2) a model of *actual distribution* that quantifies the relative likelihood of current invasion in the quarantine area. Our predictions were based on four groups of primary parameters that vary in space and time: climate conditions, topographical factors, abundance and susceptibility of host vegetation, and dispersal pressure. First, we used multi-criteria evaluation to identify large-scale areas at potential risk of infection. We mapped and ranked host abundance and susceptibility using geospatial vegetation data developed with gradient nearest neighbor imputation. The host vegetation and climate variables were parameterized in accordance to their epidemiological importance and the final appraisal scores were summarized by month to represent a cumulative spread risk index, standardized as five categories from very low to very high risk. Second, using the field data for calibration we applied the machine-learning method, maximum entropy, to predict the actual distribution of the sudden oak death epidemic. The dispersal pressure incorporated in the statistical model estimates the force of invasion at all susceptible locations, allowing us to quantify the relative likelihood of current disease incidence rather than its potential distribution. Our predictions show that 65 km^2 of forested land was invaded by 2009, but further disease spread threatens more than 2100 km^2 of forests across the western region of Oregon (very high and high risk). Areas at greatest risk of disease spread are concentrated in the southwest region of Oregon where the highest densities of susceptible host species exist. This research identifies high priority locations for early detection and invasion control and illustrates how iSDMs can be used to analyze the actual versus potential distribution of emerging infectious disease in a complex, heterogeneous ecosystem.
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1. Introduction

The rapid spread of invasive organisms and emerging infectious diseases is one of the most important ecological outcomes from the drastic alteration of natural environments by human activities (Vitousek et al., 1996; Foley et al., 2005). In our highly globalized world, only few habitats have been spared from the detrimental impacts of biological invasions on biodiversity, community structure, nutrient cycling, or ecosystem productivity (Mack et al., 2000; Hoffmeister et al., 2005). In managed landscapes, human-induced invasions of exotic organisms and pathogens cause enormous economic losses by threatening our efforts to sustain agricultural production and maintain healthy forest ecosystems (Daszak et al., 2000; Pimentel et al., 2000). Despite intense preventive actions, invaders manage to affect extensive landscapes often due to slow and incomplete discovery of invasion outbreaks. As early detection

* Corresponding author. Tel.: +1 704 232 0073.
E-mail address: tvaclavi@uncc.edu (T. Václavík).

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crucially enhances the efficacy of invasion control and eradication treatments (Simberloff, 2003), there is an increasing need for predictive tools that identify the current geographic extent of invasion spread and the habitats at potential risk of invasion (Thuiller et al., 2005; Franklin, 2010).

Predicting the spatial distribution of invaders and pathogens is enormously challenging in heterogeneous environments. However, species distribution models (SDMs) that characterize the ecological niche of organisms and relate it to known environmental factors have provided an effective analytical framework for predicting the spread of biological invasions (e.g., Lippitt et al., 2008; Chytry et al., 2009; Strubbe and Matthysen, 2009). To develop invasive species distribution models (iSDMs), two approaches have been generally adopted, although their distinction has often been unclear in the literature. First, researchers predict the *potential distribution* of a biological invasion by identifying locations with environmental conditions potentially suitable for growth and reproduction, in which the invader could exist (Hirzel and Le Lay, 2008; Jeschke and Strayer, 2008). Second, researchers estimate the *actual distribution* of a biological invasion by identifying areas where the invader currently exists, constrained not only by environmental factors but also by colonization time lag and dispersal limitations (Soberon, 2007; Jimenez-Valverde et al., 2008). While the first approach has been used to target various ecosystems potentially threatened by invasive organisms and diseases (Meentemeyer et al., 2004; Lippitt et al., 2008), or to understand the behavior of invaders in novel landscapes (Peterson et al., 2003; Sutherst and Bourne, 2009), the second approach is essential for quantifying the actual range of invasions and predicting their extant consequences in specific environments (Meentemeyer et al., 2008a; Václavík and Meentemeyer, 2009). Although knowledge from both types of spatial models can be extremely useful for guiding the management of biological invasions, no studies to date have used both approaches simultaneously to prioritize landscape contexts for early detection surveillance and invasion control.

In this study, we model and map the potential and actual distribution of sudden oak death (SOD) disease in western Oregon. An isolated outbreak of this emerging forest disease, caused by the invasive plant pathogen *Phytophthora ramorum*, was discovered in Oregon forests in 2001 (Hansen et al., 2008), more than 200 km from the closest documented infection in Humboldt County, California. *P. ramorum* causes significant mortality of tanoak (*Notholithocarpus densiflorus*) and oak (*Quercus* spp.) trees and infects a wide range of other plant species, such as Oregon myrtle (*Umbellularia californica*), Pacific rhododendron (*Rhododendron macrophyllum*), and evergreen huckleberry (*Vaccinium ovatum*), considerably altering the composition and structure of forest communities and changing ecosystem processes (Meentemeyer et al., 2008b; Cobb et al., 2010; Davis et al., 2010). The disease symptoms are expressed in two distinct forms, either as lethal infections in canker hosts that serve as epidemiological dead-ends or as non-lethal infections in foliar hosts that produce large amounts of infectious spores on necrotic leaves (Garbelotto et al., 2003; Rizzo and Garbelotto, 2003). These spores are passively transmitted among individual trees and forest patches via rain-splash and wind-driven rain (Davidson et al., 2005), affecting considerable forest area with susceptible host species and favorable environmental conditions.

In contrast to relatively wide distribution throughout California, the pathogen occurs in Oregon only in one small area in Curry County near the town of Brookings (Kanaskie et al., 2009a). Despite substantial control efforts consisting of cutting and burning infected and potentially exposed host plants, and applying herbicide to prevent tanoak sprouting, *P. ramorum* continues to spread from the initial infested sites (Hansen et al., 2008; Kanaskie et al., 2009b). In 2007, SOD quarantine area was extended to current 420 km^2 due to the emergence of six new outbreaks found outside the original 65 km^2 quarantine boundary. The abrupt disease expansion is attributed to several consecutive years of unusually wet and warm weather that promotes long distance dispersal of the pathogen (Davidson et al., 2005; Rizzo et al., 2005). However, it is believed that the major reason why control activities have been only partially successful is the late discovery of disease outbreaks, which propagated across forested landscapes before typical disease symptoms were recognized and infected sites treated (Goheen et al., 2009; Kanaskie et al., 2009c).

Successful containment of SOD depends heavily on early detection, so the pathogen can be destroyed before it can intensify and spread. Aerial surveys searching for dead and dying trees are good detection tools but their effectiveness largely depends on the degree of latency of disease symptoms. Field surveys and stream baiting with subsequent laboratory analyses can detect an infestation in a very early stage but represent labor intensive and costly methods. Predictive risk models thus offer important alternatives for prioritizing areas for early detection and eradication treatments. Although predictive models of *P. ramorum* establishment and spread have been developed and used in California (Meentemeyer et al., 2004, 2008a), similar modeling has been limited in Oregon due to unavailable vegetation data. This situation now has been remedied by new spatial vegetation data (Ohmann and Gregory, 2002; Ohmann et al., 2007) that allow us to map host susceptibility characteristics across Oregon forests.

Here, we present spatial predictions of *P. ramorum* establishment and spread risk that are being actively used to target early detection and control further disease spread in Oregon forests. The goal of this study was to develop two predictive models: (1) a model of *potential distribution* that estimates the level and spatial variability of *P. ramorum* establishment and spread risk in six ecoregions in Oregon, and (2) a model of *actual distribution* that quantifies the relative likelihood of *P. ramorum* current invasion in the SOD quarantine area. Our predictions are based on GIS analysis of four groups of primary parameters that vary in space and time: climate conditions, topographical factors, abundance and susceptibility of host vegetation, and dispersal pressure. First, we built a heuristic model using multi-criteria evaluation (MCE) method to identify large-scale areas at potential risk of disease infection. Second, using extensive field data for model calibration and calculation of dispersal pressure we applied the machine-learning method, maximum entropy (MAXENT), to predict the actual distribution of the sudden oak death epidemic. Spatially explicit models of potential and actual distribution of *P. ramorum* invasion in Oregon are urgently needed to provide a better picture of forest resources threatened by this destructive pathogen.

2. Methods

2.1. Field data collection

To examine factors influencing the spatial distribution of invasion probability of *P. ramorum*, we collected field data over the span of nine years throughout heterogeneous habitat conditions in southwest Oregon. The early detection program was coordinated by the Oregon Department of Forestry and the USDA Forest Service year-round since 2001, using a combination of fixed-wing and helicopter surveys and ground-based checks (Goheen et al., 2006; Kanaskie et al., 2009c). Each year, the forest landscape in southwest Oregon was systematically scanned from a helicopter to look for signs of dead or dying trees, covering the majority of the tanoak host type. Cases, in which apparent crown mortality was discovered, were recorded and mapped using sketch maps and the Global Positioning System (GPS), and followed by thorough field inspections. All mapped sites with tree mortality were visited and

evaluated on the ground, although the difficulty in accessing some areas due to rugged terrain and other accessibility obstacles occasionally delayed field visits. Additional ground-based surveys were conducted in areas with known host vegetation because detecting *P. ramorum* from the air is impossible when the symptoms are restricted to necrotic lesions on leaves and twigs or external bleeding on the trunks of infected live trees with healthy-appearing foliage. Transect surveys were used to check for symptomatic vegetation in potential timber sale areas, along roadsides, popular hiking trails, and high-use campgrounds. Extensive watershed-level monitoring was done both inside and outside the quarantine area using stream baiting with tanoak and rhododendron leaves, followed by surveys to locate infected plants when stream baits detected *P. ramorum* (Sutton et al., 2009). Symptomatic host plants were checked for infection by: (1) isolating and transferring symptomatic tissue directly onto plates with a selective media for *Phytophthora* species, and (2) analyzing samples in Oregon State University and Oregon Department of Agriculture laboratories via traditional culturing and a polymerase chain reaction (PCR)-based molecular assay, using primers designed to amplify *P. ramorum* DNA (Ivors et al., 2004; Goheen et al., 2006). Through these procedures, we obtained a reliable set ($n = 802$) of confirmed locations for plants infected by *P. ramorum* between 2001 and 2009.

2.2. Host species mapping

We restricted our modeling area to six ecoregions in western and central Oregon that have susceptible host species and environmental conditions that can potentially harbor *P. ramorum*: Coast range, Willamette valley, Klamath mountains, Western Cascades, East Cascades-north, and East Cascades-south (Fig. 1). To map and rank susceptibility and distribution of *P. ramorum* hosts, we used geospatial vegetation data developed using gradient nearest neighbor (GNN) imputation (Ohmann and Gregory, 2002; Ohmann et al., 2007; Pierce et al., 2009). The GNN method applies direct gradient analysis (canonical correspondence analysis) and nearest neighbor imputation to ascribe detailed ground attributes of vegetation to each pixel in a regional landscape. We developed GNN species models for each of the six ecoregions in western and central Oregon in which *P. ramorum* hosts occur. Field plot data consisted of canopy cover of plant species recorded on several thousand field plots installed in regional inventory, ecology, and fuel mapping programs. Spatial explanatory variables were measures of climate, topography, parent material, and geographic location. The resulting GNN models are 30-m-resolution GIS rasters, in which each cell value is associated with codes of individual species and their abundances (percent cover). We extracted abundance data for 14 host species present in the study area (Table 1).

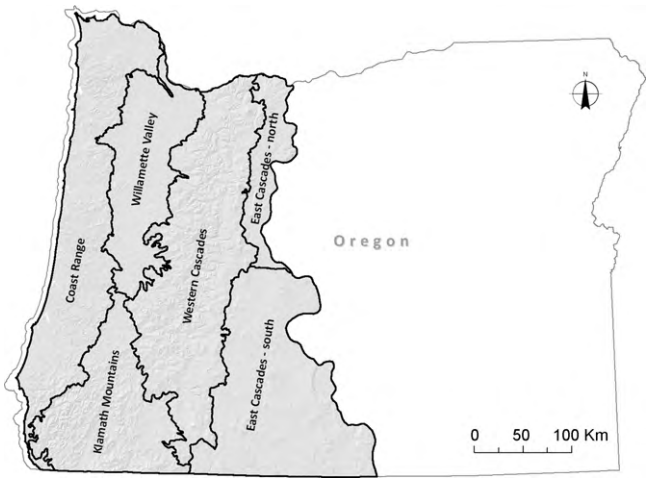

Fig. 1. Study area: six ecoregions in western Oregon that have susceptible host species and climate conditions potentially suitable for establishment and spread of *P. ramorum*.

Table 1
Spread scores of host species based on their potential to spread inoculum of *P. ramorum*.

Hosts	Score
Arbutus menzeisii—Pacific madrone	1
Arctostaphylos spp.—Manzanita	1
Frangula californica—California buckthorn	1
Frangula purshiana—Pursh's buckthorn	1
Notholithocarpus densiflorus—Tanoak	10
Lonicera hispidula—Pink honeysuckle	1
Pseudotsuga menziesii—Douglas-fir	1
Quercus chrysolepis—Canyon live oak	0
Quercus kelloggii—Black oak	0
Rhododendron spp.—Rhododendron	5
Rubus spectabilis—Salmonberry	1
Sequoia sempervirens—Redwood	3
Umbellularia californica—Oregon myrtle	5
Vaccinium ovatum—Evergreen huckleberry	1

2.3. Climate and topography surfaces

We quantified a set of three climate and three topographical variables that play an important role in the establishment and spread of sudden oak death disease. To map weather conditions known to affect foliar plant pathogens (Woods et al., 2005), we derived 30-year monthly averages (1971–2001) of maximum temperature, minimum temperature, and precipitation characteristics from the parameter elevation regression on independent slopes model (PRISM; Daly et al., 2001). PRISM uses point measurements from a large sample of weather base stations and combines them with digital terrain data, coastal proximity, vertical mass layering, and other factors to spatially interpolate climate variability across large landscapes. We used 800 m resolution grids for each month in the rainy season (December to May) that represents the reproductive period for *P. ramorum* in California and Oregon (Davidson et al., 2005). We also derived three topographic variables: elevation, topographic moisture index (TMI), and potential solar irradiation (PSI) from the U.S. Geological Survey 30-m digital terrain model. The TMI describes the effect of topography on local moisture availability and was calculated as the natural log of the ratio between the upslope contributing drainage area and the slope gradient of a grid cell (Moore et al., 1991). The PSI characterizes the potential mean solar irradiation and was calculated for the rainy season using the cosine of illumination angle on slope equation (Dubayah, 1994).

2.4. Model of potential distribution

We developed a heuristic (rule-based) iSDM model using multi-criteria evaluation (MCE) method (Malczewski, 1999; Jiang and Eastman, 2000; Mendoza and Martins, 2006) to identify the areas at potential risk of *P. ramorum* establishment and spread in western Oregon. Following methods described in Meentemeyer et al. (2004) for California, expert input was used to assign a weight of relative importance to each predictor variable and rank the criterion range to standardize the data and determine the magnitude and direction of their effect on potential disease spread.

2.4.1. Ranking host vegetation

We compiled vegetation data to create a host index variable calculated in the GIS by summing the products of the species abundance score and spread score in each 30 m cell. To generate the species abundance score, the percent canopy cover of each species

Table 2
Range of values and assigned scores (*R*), ranked 0–5 from least to most suitable for establishment and spread of *P. ramorum*.

Rank	Precipitation (mm)	Average maximum temperature (°C)	Average minimum temperature (°C)
5	>125	18–22	–
4	100–125	17–18; 22–23	–
3	75–100	16–17; 23–24	–
2	50–75	15–16; 24–25	–
1	25–50	14–15; 25–26	>0
0	<25	<14; >26	<0

was linearly reclassified into ten abundance classes using equal interval classification scheme. To generate the species spread score, individual host species were scored from 0 to 10 according to their potential to produce inoculum and spread the disease to other hosts (Table 1). With minor changes to account for specific disease behavior in Oregon (Hansen et al., 2008), we followed the scoring scheme previously developed by Meentemeyer et al. (2004) for SOD risk model for California. Tanoak was assigned the highest score of 10 as it is the most affected species (Goheen et al., 2006; Kanaskie et al., 2009c) and predominant sporulating host in Oregon forests (Hansen et al., 2008). Tanoak is susceptible to both foliar and stem infection and is associated with high severity infections in mixed redwood-tanoak and evergreen forest associations (Maloney et al., 2005). Oregon myrtle was scored moderately high (5) because the foliar infection on this host produces significant amounts of inoculum that spreads to other host vegetation in the form of zoospores and sporangia (Davidson et al., 2005; Rizzo et al., 2005). Several landscape epidemiological studies in California consistently observed positive correlation between the presence of Oregon myrtle and *P. ramorum* infection (Kelly and Meentemeyer, 2002; Condeso and Meentemeyer, 2007; Meentemeyer et al., 2008a), but this host appears to play a less important role in the epidemiological system in Oregon (Hansen et al., 2008). *Rhododendron* species were also scored moderately high (5) as they are susceptible to both foliar and branch infection, and are widely distributed in the understory of mixed evergreen and coniferous forests in Oregon (Goheen et al., 2006). Redwood (*Sequoia sempervirens*) was given a score of 3 because the production of sporangia from its foliar infestation is limited but the species is often present in association with more susceptible tanoak (Maloney et al., 2005). The remaining species that are susceptible to foliar infection and provide transmission pathways for the pathogen were assigned a value of 1. Both species of oaks were scored 0, as they represent terminal-hosts in the epidemiological system and their potential to spread inoculum is minimal (Davidson et al., 2005). The final host index values were linearly rescaled into five standard ranks (0–5).

2.4.2. Ranking climate factors

We ranked precipitation and temperature conditions using threshold values from Meentemeyer et al. (2004) based on published knowledge of *P. ramorum* biophysical properties gained from laboratory tests and field studies (Table 2). Since water must be available on plant surfaces for a substantial period of time (6–12 consecutive hours) before infection is initiated (Garbelotto et al., 2003; Tooley et al., 2009), precipitation represents a significant limiting factor for *P. ramorum*. We assigned the highest score (5) to areas with an average monthly precipitation greater than 125 mm, being the most suitable for disease establishment and inoculum production. Lower scores (4–1) were given to progressively lower rainfall amounts, while areas receiving less than 25 mm of rainfall were given a score of 0. Laboratory experiments demonstrated that *P. ramorum* thrives best at mild temperatures between 18 and 22 °C, while infection rates decrease to less than 50% at temperatures below 12 °C and above 30 °C (Werres et al., 2001; Garbelotto et al., 2003; Englander et al., 2006; Tooley et al., 2009). Therefore, we assigned the areas with an average maximum temperature between 18 and 22 °C the highest rank of 5. Areas with maximum temperatures outside the most suitable range were given progressively lower scores. Although little is known about the effect of minimum temperature on infection rates, *P. ramorum* is intolerant to temperatures below freezing (Rizzo and Garbelotto, 2003; Browning et al., 2008). Areas with average minimum temperatures above freezing (0 °C) were assigned a score of 1 and areas with average minimum temperatures below freezing a score of 0.

Table 3
Importance weights (*W*) assigned to predictor variables, ranked 1–6 from lowest to highest importance for *P. ramorum* (according to Meentemeyer et al., 2004).

Variable	Weight
Host species index	6
Precipitation	2
Maximum temperature	2
Minimum temperature	1

2.4.3. Developing heuristic model

We summarized the final appraisal scores for western Oregon to represent a cumulative spread risk index that was subsequently standardized into five risk categories from very low risk to very high risk. Each predictor variable (criterion), ranked between 0 and 5 to encode the suitability for disease establishment and spread, was assigned a weight according to the estimated relative importance of the variable in the epidemiological system (Table 3). Using the weights and scores of vegetation and climate parameters, the final spread risk was computed for each grid cell by finding the sum of the product of each ranked variable and its weight, divided by the sum of the weights:

$$\bar{S} = \frac{\sum_i^n W_i R_{ij}}{\sum_j^n W_i} \quad (1)$$

where $\bar{S}$ is the appraisal score (spread risk) for a grid cell, W_i is the weight of the ith predictor variable, and R_{ij} is the rank, or score, of the jth value of the ith variable. We computed the equation for each month in the pathogen's reproductive season (December–May) and averaged the six monthly maps into one cumulative spread risk index. This risk model represents a potential distribution of *P. ramorum* in western Oregon based on site suitability for disease establishment and inoculum production, without considering pathogen's dispersal pressure or human-mediated forms of spread.

2.5. Model of actual distribution

We developed a statistical iSDM model using maximum entropy (MAXENT) to estimate the actual distribution of *P. ramorum* infections within the 2008 quarantine area in southwest Oregon. MAXENT is a machine-learning method that predicts the distribution of an organism by finding the probability distribution of maximum entropy (i.e., the closest to uniform) that respects a set of constraints derived from sample locations. The constraints are represented by simple functions of environmental predictor variables, with their means required to be close to the empirical average of occurrence sites (Phillips et al., 2006; Phillips and Dudik, 2008). This method has been shown to perform well in comparison with other algorithms that utilize presence-only data to predict species distributions (Elith et al., 2006; Elith and Graham, 2009; Václavík and Meentemeyer, 2009).

2.5.1. Developing statistical model

We split the total of 802 field samples of confirmed *P. ramorum* infection chronologically into three datasets. The 2005–2008 dataset ($n = 482$) was used to calibrate the relative likelihood of current invasion based on the relationship between the field observations of disease occurrence and 21 predictor variables including three climate factors (maximum temperature, minimum temperature, precipitation), three topographical factors (elevation, TMI, PSI), abundance of 14 host species (listed in Table 1), and a dispersal pressure variable. The dispersal pressure term was computed with the 2001–2004 dataset ($n = 218$) to quantify the relative force of invasion at all locations in the study area (Hastings et al., 2005; Meentemeyer et al., 2008a) and thus force the MAXENT model to predict the actual or current distribution of the pathogen rather than its potential distribution (Václavík and Meentemeyer, 2009). We used a cumulative distance metric that incorporates dispersal limitations in iSDMs without explicitly estimating the dispersal characteristics of the organism (Allouche et al., 2008). The cumulative distance (D_i) summed the inverse of the squared Euclidean distances d_{ik} between each potential source of invasion k (confirmed between 2001 and 2004) and target plot i (sampled between 2005 and 2008):

$$D_i = \sum_{k=1}^{N} \left(\frac{1}{(d_{ik})^2} \right) \quad (2)$$

Such a distance-constraining factor is crucial for discriminating the actual distribution from the potential distribution of biological invasions, as it accounts for restrictive forces that prevent invasive species from colonizing habitats environmentally favorable but remote from already invaded locations (Václavík and Meentemeyer, 2009; Lobo et al., 2010). If dispersal pressure was omitted, then all sites that are environmentally similar to those already invaded, would be modeled as actual distribution, yielding considerable over-predictions.

Utilizing the MAXENT software version 3.2.1, we iteratively weighted each predictor variable to maximize the likelihood to reach the optimum probability distribution, and used the logistic output to ensure a predicted range between 0 and 1 (Elith and Burgman, 2003; Phillips and Dudik, 2008). We selected 500 iterations for model convergence and employed the regularization procedure that prevents overfitting better than variable-selection methods commonly used in traditional statistical models (Phillips and Dudik, 2008). In addition, we used a jackknife test of the relative contribution to model gain to get insight into the relative importance of individual explanatory variables (Phillips et al., 2006).

2.6. Evaluating the models

Since the potential distribution is a hypothetical concept that refers to locations which could be infested by the forest pathogen based on suitable environmental factors, the heuristic model cannot be rigorously assessed with the use of field presence/absence data (Chefaoui and Lobo, 2008; Václavík and Meentemeyer, 2009; Lobo et al., 2010). As an alternative, we can examine the correspondence between the predicted risk levels and infested locations and compare it to risk levels at randomly distributed points. We used the GIS to generate the same number of random points as the number of confirmed locations ($n = 802$) and ran a *T*-test to identify the degree to which predicted potential distribution differs between invaded and random sites.

The actual distribution refers to locations where the pathogen most likely exists at a specific time, as constrained by environmental and dispersal limitations. The performance of the statistical model thus can be rigorously evaluated with field data. The 2009 set of confirmed infections ($n = 102$) was set aside from the model development process to be used as an independent dataset for validation. Samples that were collected and tested in laboratory in 2009 but were negative for infection were used as absences in calculating the accuracy statistics. However, we included only those ($n = 34$) located further than 200 m from known confirmed sites to account for scale on which the disease is known to be clustered (Condeso and Meentemeyer, 2007) and thus avoid potential false negative cases. We compiled these datasets and used the area under the curve (AUC) of the receiver operating characteristics (ROC) to examine the true positive rate as a function of the false positive rate at each possible probability threshold predicted by the model (Fielding and Bell, 1997; Pontius and Schneider, 2001; Hirzel et al., 2006). We also calculated omission and commission error rates at the threshold that maximized specificity and sensitivity of the statistical model (Jimenez-Valverde and Lobo, 2007; Freeman and Moisen, 2008).

3. Results

3.1. Predicted geographic patterns of potential invasion

The model of potential distribution predicts the level and spatial variability of *P. ramorum* establishment and spread risk in six ecoregions in Oregon (Fig. 2). Nearly 252 km^2 (0.2%) of western and central Oregon's 111,694 km^2 of land area was predicted as very high risk for disease spread (Table 4). Very high risk habitats occur in the southwest portion of the study area, mostly in the Coast Range ecoregion within 50 km from the Pacific Ocean. They are patchily distributed across the valleys of Chetco River, Wheeler Creek, Pistol River, Rogue River, Elk River, Sixes River, and their tributaries. Very high risk was generally identified over relatively small areas (mean patch size = 0.9 hectares) nested within larger areas of high risk and coinciding with the highest abundances of the most important host species: tanoak, rhododendron, and Oregon myrtle. The very high risk levels occur most frequently in Curry County, in which they encompass a total of 243.3 km^2 (5.8% of county). Three other counties (Coos, Douglas, and Josephine) include very high risk habitats, although these habitats cover only 0.1% of each county area.

Nearly 1865 km^2 (1.7%) of the study area was mapped high risk. High risk habitats form slightly more continuous stretches (mean patch size = 2.1 ha) along river valleys of the Coast Range and the western part of the Klamath Mountains ecoregion. Although highly concentrated in the southwest portion of the state, high risk areas extend north in small patches along the coast to the Umpqua River and its tributaries in Douglas County. The majority of continuous areas predicted as high risk occur in Curry County, in which it encompasses a total of 1333.3 km^2 (31.8% of county area). These areas coincide with suitable climate conditions of high moisture availability and relatively warm temperatures in large continuous areas of susceptible forest vegetation. High risk habitats are typically mixed evergreen forests including redwood and Douglas fir but with tanoak as a dominant or co-dominant species. Rhododendron and evergreen huckleberry often occur in the understory of these forest communities. Larger areas of high risk were also identified in Coos County (210.1 km^2; 5.1% of county area) and Josephine County (290.9 km^2; 6.9% of county area).

Over 4216 km^2 (3.8%) of the study area was mapped moderate risk. Moderate risk habitats are scattered across southern half of the Coast Range ecoregion and western part of the Klamath Mountains but extends in smaller amounts to the Western Cascades ecoregion. In the two counties with the largest moderate risk prediction, Curry County (1084.1 km^2; 25.8% of county area) and Josephine County (1021.7 km^2; 25.8% of county area), moderate risk was mapped mostly in habitats with climatically suitable condi-

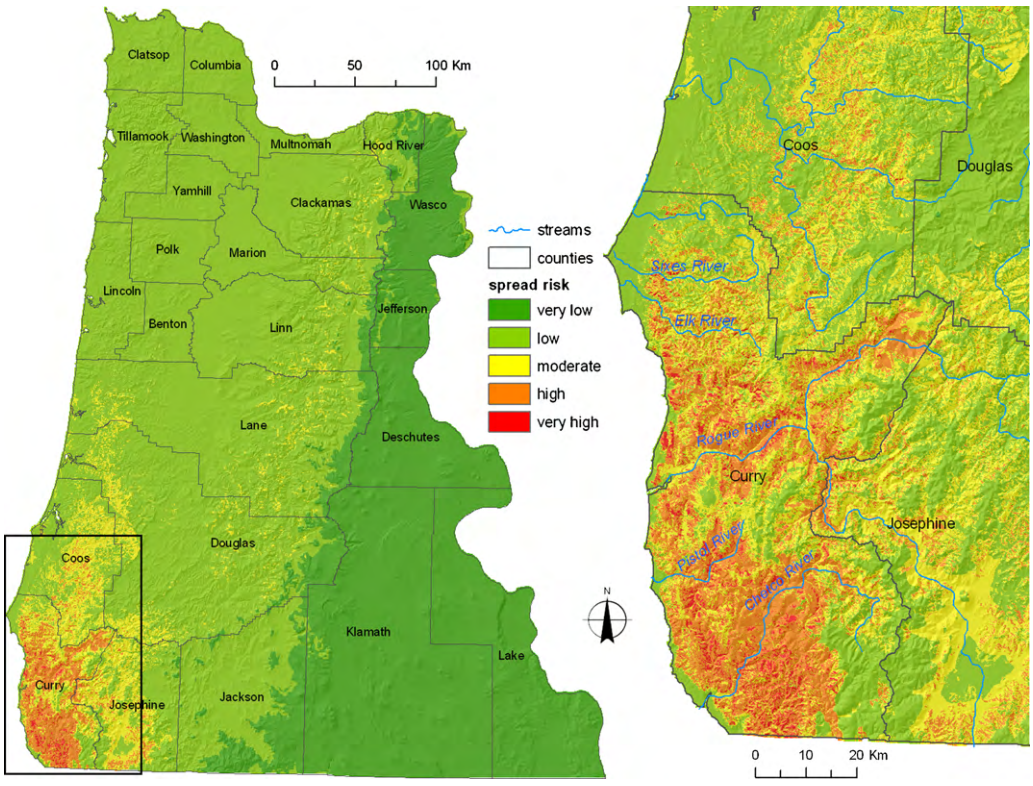

Fig. 2. Predicted spread risk map for *P. ramorum* in western Oregon based on heuristic model of potential distribution. The inset shows southwest counties with the highest spread risk levels.

tions but with lower values of host vegetation index. In the Klamath region, it forms a buffer-like pattern around high risk areas. In the Western Cascades ecoregion, moderate risk occurs in small patches (mean patch size = 1.5 ha) and extends to Oregon's northern boundary, following the patchy distribution of rhododendron species in Douglas-fir dominated forest communities.

Over 66,530 km^2 (59.6%) of western Oregon was mapped low risk and 38,708 km^2 (34.7%) of the area was mapped very low risk. Low risk habitats are generally larger in area (mean = 65.3 ha) and extend over a vast portion of the northern part of the Coast Range ecoregion and the entire Willamette Valley ecoregion. Low risk is often associated with moderately suitable temperature and

Table 4
Land area of predicted spread risk levels in Oregon counties (in km^2 and percent of total county area).

County	Area (km^2)	Very low risk		Low risk		Moderate risk		High risk		Very high risk	
		km^2	%	km^2	%	km^2	%	km^2	%	km^2	%
Benton	1758	0.0	0.0	1738.3	98.9	2.8	0.2	0.0	0.0	0.0	0.0
Clackamas	4866	114.0	2.3	4652.6	95.6	77.4	1.6	0.0	0.0	0.0	0.0
Clatsop	2083	0.0	0.0	2077.0	99.7	0.0	0.0	0.0	0.0	0.0	0.0
Columbia	1693	0.0	0.0	1693.4	100.0	0.0	0.0	0.0	0.0	0.0	0.0
Coos	4135	0.0	0.0	3161.3	76.5	759.5	18.4	210.1	5.1	3.8	0.1
Curry	4195	0.0	0.0	1534.8	36.6	1084.1	25.8	1333.3	31.8	243.3	5.8
Deschutes	4688	4683.8	99.9	4.5	0.1	0.0	0.0	0.0	0.0	0.0	0.0
Douglas	13,088	1027.5	7.9	11,304.5	86.4	726.8	5.6	28.0	0.2	1.3	0.0
Hood River	1347	504.0	37.4	826.2	61.3	17.1	1.3	0.0	0.0	0.0	0.0
Jackson	7248	2307.1	31.8	4936.3	68.1	4.4	0.1	0.1	0.0	0.0	0.0
Jefferson	1723	1644.8	95.5	71.5	4.1	0.0	0.0	0.0	0.0	0.0	0.0
Josephine	4240	66.1	1.6	2858.4	67.4	1021.7	24.1	290.9	6.9	3.5	0.1
Klamath	15,846	15,769.2	99.5	62.9	0.4	0.0	0.0	0.0	0.0	0.0	0.0
Lake	8353	8334.2	99.8	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Lane	11,938	913.6	7.7	10,651.5	89.2	370.8	3.1	2.5	0.0	0.0	0.0
Lincoln	2520	0.0	0.0	2513.5	99.7	6.4	0.3	0.0	0.0	0.0	0.0
Linn	5980	313.9	5.2	5541.1	92.7	105.0	1.8	0.0	0.0	0.0	0.0
Marion	3092	76.1	2.5	2981.2	96.4	34.4	1.1	0.0	0.0	0.0	0.0
Multnomah	1125	0.0	0.0	1121.3	99.7	3.4	0.3	0.0	0.0	0.0	0.0
Polk	1927	0.0	0.0	1926.6	100.0	0.1	0.0	0.0	0.0	0.0	0.0
Tillamook	2832	0.0	0.0	2832.2	100.0	0.0	0.0	0.0	0.0	0.0	0.0
Wasco	3274	2954.1	90.2	299.7	9.2	2.8	0.1	0.0	0.0	0.0	0.0
Washington	1881	0.0	0.0	1881.4	100.0	0.0	0.0	0.0	0.0	0.0	0.0
Yamhill	1860	0.0	0.0	1860.2	100.0	0.0	0.0	0.0	0.0	0.0	0.0
Total	111,694	38,708.4	34.7	66,530.4	59.6	4216.7	3.8	1864.9	1.7	251.9	0.2

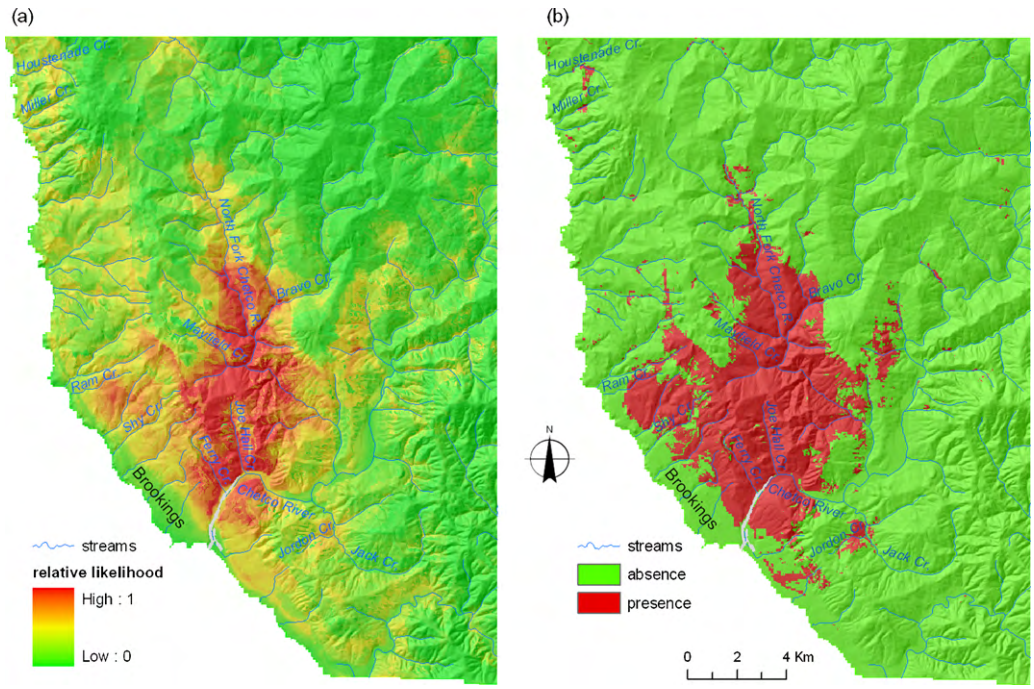

Fig. 3. Predicted actual distribution of *P. ramorum* in southwest Curry County based on maximum entropy model. Map (a) shows relative likelihood of pathogen's presence. Map (b) shows presence/absence realization of actual distribution based on probability threshold that maximized specificity and sensitivity of the model.

moisture conditions but low abundance and susceptibility of host vegetation. The low risk level was predicted over more than 95% of Benton, Clackamas, Clatsop, Columbia, Lincoln, Marion, Multnomah, Polk, Tillamook, Washington, and Yamhill counties. Very low risk habitats form nearly one large area in both East Cascades ecoregions but extend west in several large patches (mean patch size = 37 km^2) to the Western Cascades and Klamath Mountains ecoregions. Very low risk areas occur further from the coast (>150 km) at higher elevations (>1200 m) with cold temperatures and low precipitation. There are no hosts species mapped in 77% of the very low risk areas and only species with low abundance and susceptibility (mostly Douglas fir) are mapped in the remaining 23%. The very low risk levels were predicted over nearly 100% of Deschutes, Jefferson, Klamath, Lake, and Wasco counties.

3.2. Predicted geographic patterns of actual invasion

The model of actual distribution predicts the relative likelihood of *P. ramorum* current invasion in the 2009 quarantine area in Curry County (Fig. 3a). Using the threshold that maximized model effectiveness, we estimated pathogen's presence across 65.4 km^2 of land area in southwest Curry County, northwest of the town of Brookings in the Chetco River watershed (Fig. 3b). All areas predicted as being infected occur within 15 km of the Pacific coast. Two areas with the highest likelihood of infection occur at the lower section of Chetco River between Joe Hall Creek and Ferry Creek, and across the valley hillsides of North Fork Chetco River and its tributaries Mayfield Creek and Bravo Creek. Outside of the watershed, a large patch of forest predicted by the model occurs north of the town of Brookings between Ram Creek and Shy Creek. Two locations, modeled as being likely infected but in which disease has not been confirmed to date, were identified along Jack Creek and Jordon Creek in the southern portion of the Chetco River watershed and between Houstenade Creek and Miller Creek in the northwest part of the quarantine area. These locations are relatively close to known infected sites (~4 km) and coincide with areas mapped as having high abundances of tanoak and evergreen huckleberry.

The jack-knife test of variable importance (Fig. 4) shows that the variable with the highest gain when used in the model in isolation was dispersal pressure, having the most useful information that contributes to final prediction. Similarly, the dispersal pressure term decreased the model gain the most when it was omitted, having the most variability that is not present in other predictors. After the dispersal pressure variable, precipitation, maximum temperature, and elevation followed in their relative importance for model gain. From all 14 host species used for prediction, evergreen huckleberry, tanoak, and Douglas-fir were identified in the respective order as being the most important for model gain.

3.3. Model evaluation

The *T*-test for the model of potential distribution showed that modeled risk is significantly higher at sites identified as infested between 2001 and 2009 (n = 802) than at randomly selected loca-

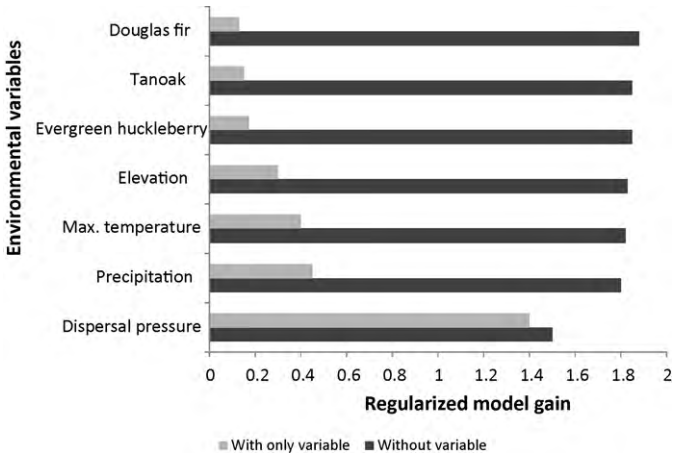

Fig. 4. Jack-knife test of variables' relative importance. Graph shows seven most important environmental variables and their influence on regularized model gain when they were used in isolation or omitted.

Table 5a
Model evaluation. Potential distribution: *T*-test of sites infected by *P. ramorum* versus random sites in predicted risk levels.

T-test ($P < 0.0001$)				
Risk level	Infected sites ($n = 802$)		random sites ($n = 802$)	
	#	%	#	%
Very high	53	6.6	4	0.5
High	386	48.1	19	2.4
Moderate	131	16.3	24	3.0
Low	232	28.9	463	57.7
Very low	0	0.0	292	36.4

Table 5b
Model evaluation. Actual distribution: evaluation statistics for Maxent model calculated with 2009 samples.

Infected sites in 2009 ($n = 102$) Uninfected sites in 2009 ($n = 34$)	
AUC	0.911
Threshold	0.124
Commission error (rate)	6 (0.18)
Omission error (rate)	13 (0.13)

tions ($P < 0.0001$) (Table 5a). Most of the 802 infected sites were mapped high risk (48%), followed by low risk (29%), moderate risk (16%), very high risk (7%), and very low risk (0%). Most of the 802 random locations were mapped as low risk (58%), followed by very low risk (36%), moderate risk (3%), high risk (2%), and very high risk (1%).

The ROC test for the model of actual distribution produced the AUC value of 0.91 based on the data from 136 samples analyzed in 2009 (Table 5b). The optimal probability threshold based on maximizing sensitivity and specificity of the model was relatively low ($t = 0.124$) and produced commission and omission error rates of 0.18 and 0.13 respectively.

4. Discussion

Mapping the geographic distribution of invasive species and diseases is essential for the examination of their impacts in natural ecosystems and implementation of effective management strategies (Holdenrieder et al., 2004). Predictive, spatial tools that identify current extent of biological invasions and habitats at potential risk of spread are increasingly needed to guide the management of biological invasions (Simberloff, 2003; Plantegenest et al., 2007). In this study, we developed two predictive models of *P. ramorum* potential and actual distribution in western Oregon to prioritize landscape context for early detection surveillance and invasion control.

The heuristic model of *P. ramorum* potential distribution identifies areas that can serve as potential habitats for disease establishment and propagation. Mapped risk is based on combined effects of host species availability and susceptibility, and climate conditions in the pathogen's major reproductive season (December–May). Based on the model criteria, our prediction indicates that numerous forests across the western region of Oregon face considerable risk of sudden oak death invasion. Although concentrated in the southwest part, very high and high risk habitats were mapped across the entire Curry County and identified at smaller scale in Coos and Josephine counties, more than 150 km away from the currently quarantined areas. This result corroborates findings of previous studies (Meentemeyer et al., 2004, 2008a) and suggests that *P. ramorum* is in relatively early stage of invasion, occupying only a small portion of its fundamental ecological niche.

The levels of *P. ramorum* establishment and spread risk agree closely with predictions from an equivalent model of potential distribution developed for California (Meentemeyer et al., 2004). Although our estimates for Oregon are based on GNN vegetation data that differ from those used as inputs for modeling in California (CALVEG dataset; USDA Forest Service RSL, 2003), predicted risk levels align considerably well across the border region of north-east California and south-west Oregon. Several discrepancies in risk levels (moderate risk in Del Norte County, high risk across the state border in Curry County) are caused by higher susceptibility ranking of tanoak and lower susceptibility ranking of Oregon myrtle in our model, based on documented differences in the epidemiological role of these species in Oregon (Hansen et al., 2008). Considering the similarity of environmental conditions and a prevalence of redwood-tanoak forests in northern California, we suggest the risk model developed for California may be slightly under-estimating the potential risk of *P. ramorum* invasion in the northernmost region.

The significant *T*-test suggests the risk model produced plausible predictions; however, 29% of currently infected sites were mapped as low risk. This type of underprediction is likely associated with the accuracy and precision of host vegetation data used as the most important criterion in model building. First, the 30 m spatial resolution of our vegetation data may be coarser than the scale at which the disease occurs. High resolution aerial photographs indicate there are small patches of host species that were mapped as non-host or no forest vegetation because these patches are smaller than the minimal mapping unit of the GNN data. Secondly, the GNN species distributions consist of a single field plot imputed to each pixel, resulting in species maps with small amounts of noise at a local scale. This fine-scale heterogeneity poses no significant problems for regional-scale analyses but may cause some infected locations to overlay with scattered pixels representing no host in the vegetation data and thus low risk in the predicted risk model. Lastly, a non-forest mask was applied to the vegetation model based on maps of ecological systems developed by the USGS Gap Analysis Program. There are 23 infected locations in close vicinity of the town of Brookings that overlap with pixels classified as low density or open space development in the vegetation model. In addition, "spread risk" in this model is defined as the potential to produce inoculum and further propagate the disease across landscape. Therefore, it places low importance to terminal hosts (e.g., oaks) that may get invaded for a short period of time but serve as epidemiological dead-ends (Rizzo and Garbelotto, 2003; Davidson et al., 2005).

The MAXENT model of *P. ramorum* actual distribution quantifies the relative likelihood of disease infection calculated with data from the 2001–2008 field surveys. Mapped invasion is based on the statistical relationship between known infected sites and a set of climate, topographical, host availability, and dispersal pressure variables. Model predictions suggest that current invasion range covers approximately 15% of the current quarantine area. Locations with the highest relative likelihood of pathogen's presence occur along the Chetco River and its north fork, matching field observations of disease incidence. However, several places with no field observations were predicted as likely infected and thus indicate felicitous targets for early detection surveys.

The AUC value above 0.9 and low values of commission and omission error rates (<0.2) suggest relatively high prediction accuracy of the statistical model. However, the optimal threshold that maximized model effectiveness was relatively low and reflects the difficulty of predicting invasive organisms far from equilibrium with their environment (Václavík and Meentemeyer, 2009). As the motivation was to prioritize landscape contexts for early detection and invasion control, we selected a threshold that gives the same weight to commission and omission errors, in order to balance the importance of detecting the maximum number of infected sites with the relative cost of ground and helicopter surveillance (Meentemeyer et al., 2008a). If commission errors were preferred,

the model would produce a conservative scenario, decreasing the probability to detect disease outbreaks. If omission errors were preferred, large areas with marginal likelihood of *P. ramorum* presence would be predicted, increasing the cost of unnecessary field sampling.

Predictions of the statistical model were highly correlated with temperature and moisture conditions, elevation, and abundance of major host species. However, the most important variable based on jack-knife test of model gain was dispersal pressure, representing the force of pathogen's invasion. Considerably stronger effect of dispersal pressure is in contrast with results of Ellis et al. (2010) that identified environmental factors to be slightly more important than force of invasion for determining the spatial pattern of *P. ramorum* in northern California. Again, our findings indicate that the pathogen is in an initial stage of invasion, and are consistent with previous studies that recognized incorporation of range-confining variables based on space or distance metrics to be essential for predicting organisms under colonization-lag and non-equilibrium scenarios (Araujo and Pearson, 2005; Allouche et al., 2008; De Marco et al., 2008; Václavík and Meentemeyer, 2009). The early stage of invasion is also supported by the fact that dispersal pressure was significant for model prediction, although it was based on simple cumulative distance from initially invaded sites and did not account for potential landscape connectivity (Ellis et al., 2010) or long distance human-mediated forms of spread (Cushman and Meentemeyer, 2008).

In this study, we applied a heuristic and statistical model in the GIS to produce spatial predictions of the potential and actual distribution of *P. ramorum* invasion in Oregon forests. Several studies have mapped potential SOD risk at a state (Meentemeyer et al., 2004) or continental scale (Venette and Cohen, 2006; Kelly et al., 2007) and identified actual patterns of disease spread in California (Meentemeyer et al., 2008a). However, this is the first effort to model *P. ramorum* invasion in Oregon and use both iSDM approaches simultaneously, while clearly distinguishing between their meanings and purposes. While estimates of potential distribution provide a better picture of forests potentially threatened by disease invasion, the model of actual distribution quantifies the current range at unsampled locations. Application of the actual distribution model to on-the-ground management will increase the efficacy of detection and eradication of current outbreaks, especially under conditions of limited resources. Application of the potential distribution model will allow identifying habitats at the highest risk of future disease spread, to which preventive measures can be applied. When resources are available, field monitoring of high risk habitats will increase the chance to detect outbreaks introduced via long-distance dispersal events and minimize future disease impacts. Therefore, complementary knowledge from both types of spatial models is crucially needed to guide monitoring and control activities, especially for organisms in early stages of invasion that have considerable mismatch between their fundamental and realized niche.

iSDMs are getting increasingly popular to tackle early detection and eradication problems. However, models of potential and actual distribution are often confused and have never been used simultaneously for the same organism. In this application, we developed a heuristic model based on current knowledge of *P. ramorum* physiological and epidemiological requirements to represent its fundamental niche (potential distribution). We applied a statistical model trained by field data to portray pathogen's realized niche (actual distribution) and included data on dispersal pressure to constrain predicted range (Allouche et al., 2008; Václavík and Meentemeyer, 2009). Although much remains to be learned about possibilities to control further *P. ramorum* spread, these spatially explicit models provide simple yet effective management tools to prioritize landscape contexts for early detection and eradication of disease outbreaks. As new infected sites are discovered, models should be updated and validated with new data to continually refine management strategies. This research illustrates how the iSDM framework can be used to analyze the actual versus potential distribution of emerging infectious disease in a complex, heterogeneous ecosystem.

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