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SPREAD OF AN INVASIVE PATHOGEN OVER A VARIABLE LANDSCAPE: A NONNATIVE ROOT ROT ON PORT ORFORD CEDAR

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Abstract. Understanding biological invasions requires information on the history of spatial spread, as well as measures of landscape and biotic features that control habitat invasibility. Because invasive species often spread quickly over large areas, attaining these two sets of information simultaneously is uncommon. We studied the spread of a fatal nonnative root pathogen, *Phytophthora lateralis*, across a heterogeneous landscape of its host, Port Orford cedar (*Chamaecyparis lawsoniana*). Within our 37-km^2 study area in southwestern Oregon and northwest California, Port Orford cedar populations are generally restricted to riparian zones along creeks. The pathogen is spread between watersheds in two ways: (1) by spore-infested material being dislodged from vehicles, and (2) by animals or people moving infested mud (i.e., via foot traffic). Using dendrochronological techniques, we determined the date of infection for dead cedars and reconstructed spread history across our study area from 1977 to 1999. Twenty-six of the 36 (72%) separate infection events we identified were caused by dispersal via vehicles along roads, and the remainder by foot traffic. Survival analysis demonstrated that cedar populations in creeks crossed by roads were more likely to be infected than those creeks that were not crossed by roads. Also, a comparison of minimum dispersal distances showed infections that moved via road moved significantly farther than those vectored by foot traffic, and the distance infection traveled declined significantly through time. We also coupled our spread history with measures of landscape and host features, including abundance of potential host trees, the distance from the road surface to the nearest potential host, length of road in immediate contact with the riparian zone, catchment area (a measure of stream flow), elevation, slope, and solar radiation. Our results show that catchment area, host abundance, and proximity to the nearest tree are significantly and positively associated with infection risk. Our study demonstrates that increased connectivity between invasible sites created by the presence of roads can increase invasion success of a plant pathogen. We also document that successful pathogen invasion can be governed by both physical landscape features and attributes of host plant populations.

Key words: Chamaecyparis lawsoniana; *dendrochronology; disease ecology; forest pathogen; invasibility; invasion ecology; landscape heterogeneity;* Phytophthora lateralis; *plant pathogen; Port Orford cedar; spatial spread.*

INTRODUCTION

Attention to the biology of invasive species has remained strong among ecologists since the time of Elton's treatment (1958) of the subject over 40 years ago. Invasive species have apparent and well-known ecological consequences for the structure of native communities, including changes in species composition and shifts in disturbance regimes (Drake et al. 1989, Van Driesche and Van Driesche 2000), as well as large economic consequences resulting from invasive agricultural pests, forest pathogens, and diseases of game species (Pimentel et al. 2000). Unfortunately, studies that have attempted to understand the process of invasion have always been hindered by the lack of two crucial components: (1) detailed maps of spread and (2) measures of landscape and biotic heterogeneity that determine differences in the invasibility of regions, communities, or populations. Both components have limited the formation of general rules about factors governing the rate and extent of biological invasions.

Historical accounts, especially maps, have been important in studies of invasive species (e.g., Levin 1992, Shigesada et al. 1995). Time-series maps describing the spread of muskrats across Europe in the late 1800s, cheatgrass across western North America in the early 1900s, and the spread of Africanized honeybees through South America, among others, are familiar to invasion ecologists, and provide valuable information about the rate of invasion and the large-scale patchiness of the expanding distribution of species (Elton 1958, Mack 1981, Williamson 1996). Most often, however,

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it is impossible to document an invasion accurately because of the speed of the invasion and the enormous effort required to track spread over large areas (e.g., Stodart and Parer 1988, Johnson and Carlton 1996). Because the scale of resolution is usually too coarse to determine unique landscape features related to spread, for instance habitat types where spread was relatively fast or slow, most maps of invasions are of limited use for connecting rates and patterns of spread to measured attributes of the native communities they invade.

For invasive species, the spatial distribution of suitable sites and the variation found among sites can mediate the characteristics of spread, and both of these sources of heterogeneity are a key component for understanding invasion processes (Andow et al. 1990, Levin 1992). However, most studies of invasion have ignored spatial and site heterogeneity, either because it is impossible to study differences in invasion rates post hoc given the lack of fine-scale spread data, or because the magnitude of the study needed to collect data on site characteristics across large areas is simply impractical (Mack et al. 2000). This has been especially true for invasive diseases, where both landscape features and host characteristics (e.g., density, vigor, and patchiness of the host organism) can influence the likelihood and severity of disease dynamics (Anderson and May 1986, Jeger 1989, Dwyer et al. 1997). Most studies of disease spread have ignored natural variation and instead have focused on the role of average host and pathogen densities (see Burden and Chilvers 1982), or on the spatial dynamics of disease over relatively homogeneous landscapes of hosts and abiotic conditions (e.g., van den Bosch et al. 1990*a*, *b*, Dwyer 1991).

Any generalized rules we hope to ascribe to invasion processes will benefit from detailed study of the spatial expansion of an invasive population, starting with its initial introduction, across heterogeneous landscapes where site invasibility is readily measurable. In the study presented here, we investigated the role of both abiotic and biotic heterogeneity in governing the spread of an invasive disease on a patchily distributed conifer. Port Orford cedar (*Chamaecyparis lawsoniana*) is infected by a fatal, nonnative root pathogen, *Phytophthora lateralis*. Landscape-level spread of *P. lateralis* spores (i.e., between cedar populations) occurs via vehicles carrying mud and organic material containing spores along roads, and by foot traffic carrying mud and organic material (e.g., hikers and hoofed animals; Hansen et al. 2000). Using dendrochronological methods that enabled us to determine the date of infection for trees, we established the initial infection date of populations within a 37-km^2 study area and reconstructed the spread of the disease over a 23-yr period. Further, we quantified landscape and host attributes that have influenced the successful invasion of healthy cedar populations. This work has three general objectives: (1) accurately describe the invasion dynamics of *P. lateralis* through time; (2) distinguish differences in spread dynamics attributable to the two major vectors (vehicles vs. foot/hoof traffic); and (3) determine to what extent variation in landscape features and host characteristics influences the probability of invasion.

Methods

The host species

Port Orford cedar is a conifer endemic to a relatively small region of northern California and southwestern Oregon. Individuals can attain sizes of up to 3.65 m diameter at breast height (Zobel et al. 1985) and ages of up to more than 900 yr (M. Creasy, *personal communication*). In the range of the cedar winters are cool and wet, and summers are warm and dry (Zobel et al. 1985), restricting Port Orford cedar to areas that maintain significant soil moisture throughout the summer (Zobel and Hawk 1980). Over much of the cedar's range, this moisture requirement restricts the cedar to areas immediately adjacent to water, such that the cedar's distribution closely follows the network of creeks and rivers (e.g., Fig. 1). Consequently, the roots of Port Orford cedar are frequently in direct contact with standing or moving water (Hansen et al. 2000).

The pathogen and its spread

Phytophthora lateralis is a water mold (Oomycetes) that infects the root system of Port Orford cedar and eventually girdles the tree's primary root collar (Hansen et al. 2000). Initial infection of individual cedars occurs when flagellate *P. lateralis* zoospores disperse through water and contact fine root hairs. Infection is fatal for the host plant, with mortality occurring in as little as 2–3 wk for seedlings and 1–4 yr for larger trees (Zobel et al. 1985, Hansen et al. 2000). Although promising testing of resistance is underway, no fully resistant genotype of Port Orford cedar is yet available for outplanting (Hansen et al. 2000). *Phytophthora lateralis* can also infect Pacific yew (*Taxus brevifolia*), although yew infections are relatively uncommon (DeNitto and Kliejunas 1991, Murray and Everett 1997).

Flagellate zoospores of *P. lateralis* are not able to move upstream to any significant extent and thus subsequent infections via water transport only travel downslope and downstream (Zobel et al. 1985). In contrast, movement of the disease over long distances, for example overland from an infected creek to an uninfected creek, is accomplished via chlamydospores, a type of resting spore. These thick-walled spores are more tolerant of dry conditions than zoospores and can be carried in mud or organic material (Hansen and Hamm 1996). Infested mud can be carried along roads by vehicles or heavy equipment (e.g., logging equipment); many infected cedar populations are found in creeks at locations below where roads cross creeks, and are a clear result of mud dislodging from vehicles. While

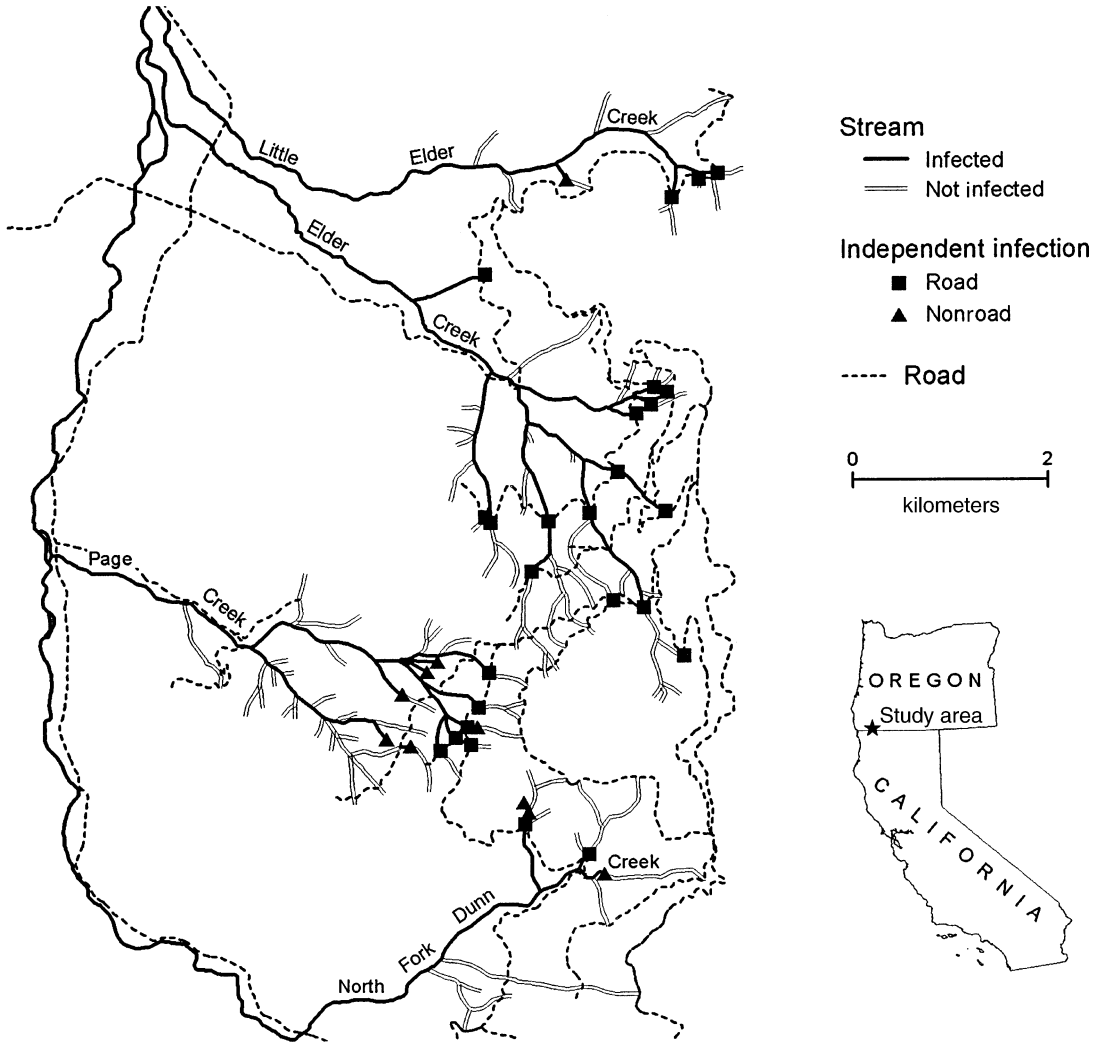

FIG. 1. Distribution of Port Orford cedar and its associated pathogen, *Phytophthora lateralis*, in 1999 within the Page Mountain study area, southwestern Oregon and northwestern California, USA. Cedars in the study area are generally restricted to riparian areas, such that their distribution follows the network of creeks. Independent infection events are those that required transport from an infected creek to an uninfected creek. Squares indicate independent infections via vehicle traffic along roads, and triangles indicate independent infections via animal and foot traffic. Infection from these events spread downstream in water.

vehicles appear to be the primary mechanism of *P. lateralis* dispersal across landscapes, foot traffic also spreads the disease. Wildlife, cattle, hikers, and workers in the woods can transport the pathogen by moving spore-infested mud on feet and boots (Hansen et al. 2000). Hereafter, we use ''foot traffic'' to indicate both foot and hoof traffic.

While infecting a cedar, *P. lateralis* will produce additional zoospores that are transported in flowing water and can infect healthy trees downstream. In some stream surveys, all trees with their roots within the normal winter high water level died from infection (Hansen et al. 2000). Trees on upland sites experience lower mortality rates, which depend on flood frequency and hillslope position, among other factors. In general, entire lengths of infected creeks will contain many standing dead Port Orford cedar within a decade after initial infection.

The initial discovery of *P. lateralis* occurred in 1923 in a nursery near Seattle, Washington, USA, well outside the cedar's natural range (Zobel et al. 1985). In 1952, the disease was first detected in the northwestern portion of the cedar's range near the coastal town of Coos Bay, Oregon, USA (Roth et al. 1957). Spread of the disease was rapid along coastal Oregon over the next few years, and slower into the drier interior. However, as road building and timber harvest accelerated in the 1960s and 1970s, *P. lateralis* invaded remote parts of the range (Hansen et al. 2000). The disease is now found throughout much of the cedar's range. The

origin of the pathogen is still unknown, although, given how rapidly the disease has spread and that it was first observed outside the cedar's range, it is well accepted that *P. lateralis* is nonnative (Zobel et al. 1985, Hansen et al. 2000).

Study area

The 37-km^2 Page Mountain study area is located in the Siskiyou Mountains, straddling the border of Oregon and California (Fig. 1), and is entirely within the Siskiyou National Forest. Vegetation on upland sites is dominated by mixed conifer forest; the most common trees are Douglas-fir (*Pseudotsuga menziesii*), incense cedar (*Calocedrus decurrens*), tanoak (*Lithocarpus densiflorus*), and Pacific madrone (*Arbutus menziesii*). The most common resource management activity has been timber harvesting; by 1998, 35% of the area had been logged (USDA Forest Service and USDI Bureau of Land Management 2000).

Within the study area, Port Orford cedar is highly restricted to creeks and is the dominant tree in most riparian areas. The area is ideal for our study because the distribution of cedar populations and the distribution of the disease are both discrete (i.e., it matches the distribution of creeks; Fig. 1), and there are at least 63 km of creeks with Port Orford cedar dissecting the study area. Also, there are 93 km of roads throughout the study area with 86 creek crossings. Almost all of these roads are gravel and were built exclusively for timber harvest before the time of initial infection by *P. lateralis*. There is strong evidence to suggest that roads serve as the primary routes for disease vectors (Hansen et al. 2000), thus ample opportunity for infection has been present in the area. Lastly, heterogeneity associated with factors that likely govern *P. lateralis* spread is high. For example, heterogeneity in the ''host landscape'' exists in the abundance of cedar at each creek crossing (i.e., the ''target'' for infection; Fig. 2), as well as the physical characteristics of each creek crossing (e.g., length of road in contact with cedar; Fig. 2).

Mapping

During the summers of 1998 and 1999 we censused all creeks within the study area for the presence of Port Orford cedar and infection by *P. lateralis*. All creeks were assessed as either occupied by uninfected cedar, occupied by infected cedar, or unoccupied by cedar. Using a Geographic Information System (GIS) produced by the USDA Forest Service, the information was assigned to each creek in the database. We digitized creeks we noted in the field that were not present in the existing database. Also available for the study area were spatially referenced data for the road system and elevation that were incorporated into our GIS.

Defining infection sites

Unique *P. lateralis* invasions across the landscape are best described by first determining all points at which new infections have started via transport of the pathogen from one creek to another. Characterizing this type of spread does not require describing the general movement of the pathogen downstream in moving water. Instead, the location of new infections, in general, is indicated by the highest tree killed by infection along a creek. In most cases, this point is just below where a road crosses a creek (Fig. 1). However, we also mapped infections that initiated at points other than road crossings. At these sites, *P. lateralis* was apparently introduced by something other than vehicular traffic; invasions occurred in cedar populations that did not have roads in contact with the creek and were not accessible by All Terrain Vehicles. Thus, all infections were assessed as being either a ''Road'' (via vehicles) or ''Nonroad'' (via foot traffic) infection (Fig. 1). Given their conspicuous association with road crossings, Road infections were assumed to only occur via vehicle travel and never via foot traffic.

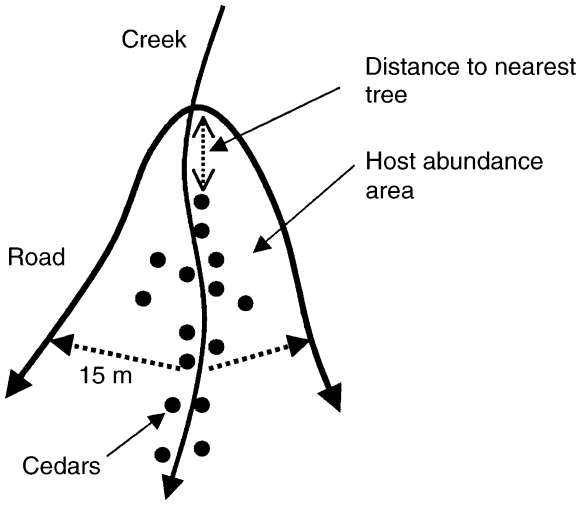

FIG. 2. Example of a road crossing a creek where host cedars may come in contact with spores of *Phytophthora lateralis* deposited by vehicles. Host target abundance below road, distance to nearest cedar, and road distance in contact with creek are shown. Road distance is the amount of road within 15 m of the creek.

We used Arc/Info version 7.2.1 (Environmental Systems Research Institute [ESRI], Redlands, California, USA) to generate basic statistics regarding the distribution of cedar, disease, roads, and creek crossings in the study area. These included the proportion of creek length infected and uninfected, overall road length and density in the study area, number of creek crossings, and elevational extent of the disease.

Reconstructing infection history

Because a creek can be crossed by more than one road, infection can enter some creeks from multiple points and at different times. Thus, for an infected creek, it is impossible to use only our distribution map of infection (Fig. 1) to assess whether infection entered

(1) only once from the highest, upstream crossing, or (2) first at the lower crossing and then later at the upstream crossing.

To solve this problem, we determined date (year) of each potential infection point by assessing the date of death of standing dead cedar using a standard dendrochronological technique called ''crossdating'' (Douglass 1939, McGraw 2000). Crossdating is based on the premise that the relative size of annual growth rings depends on climatic conditions and, therefore, creates a distinctive temporal pattern (Douglass 1920). For example, during drought years Port Orford cedar usually produces small rings relative to the ring size of the years before and after. For any dead cedar, it is possible to determine the date of death by aligning its growth ring pattern with the pattern derived from healthy trees, where the date that each ring was produced is known (see Stokes and Smiley [1968] and Swetnam et al. [1985] for details). Because the wood of Port Orford cedar is highly rot resistant (Zobel 1986), the annual growth rings of dead cedars are generally intact for many years and can still be analyzed decades after death. Initially, cores of 19 living trees in the study area were used to construct a ''master chronology'' of climatic variation to be used for crossdating. These cores were continually augmented by the addition of growth ring patterns from live trees and dead trees once their ring-width chronologies had been determined. Because of the large number of cores we collected, and because one of us was conducting a related tree coring study in the same area (M. J. Kauffman, *unpublished data*), the resulting master chronology eventually utilized data from 965 cedars.

To determine the dates of all new infections in the landscape, we collected tree ring cores from dead trees at every potential infection site (i.e., in cedar populations below every road crossing and at the top of all Nonroad infections). Wherever possible, two to three cores were taken from each of six trees at each site. In order to be certain that core samples were not missing any of the later rings due to decay, we only accepted core samples for which we could visually detect the smooth patina found at the surface of the vascular cambium (i.e., the outer layer of the last year of growth). In general, we selected the largest trees nearest the top of the infection since, as the largest targets (i.e., those with the greatest root area), large trees are the most likely to be infected first (M. J. Kauffman, *unpublished data*). In total, we collected 973 cores from 436 dead trees in the study area. Crossdating of the cores from dead trees was done using a combination of visual and computer-based (Holmes 1983) techniques. First, the cores were assessed to locate the key ring-width patterns reflected in the wood that are apparent under a stereomicroscope. Using these patterns, the cores were visually crossdated and assigned a year of death. Then, each core was measured to 0.001-mm precision using the Velmex measuring device (TA 4030H1-S6 Unislide, Bloomfield, New York), producing a numeric sequence of ring widths. Using the CoFecha software (Holmes 1983), each individual sequence was compared to the numeric version of the master chronology to produce a level of confidence in the crossdating. Only those cores with high confidence values were included in the analysis.

Crossdating established a year of death for each tree. In many cases, the tree was infected in a year preceding death, creating a latency period defined as the number of years between infection and death. In cores with a latency period, the infection date was estimated as the beginning of a marked and visually apparent decrease in ring width.

Using the infection dates acquired from crossdating, we produced a series of maps that present the spreading distribution of *P. lateralis*. These maps show precise locations of individual infection initiations through time (hereafter termed ''independent infections''), and estimated movement downstream. Downstream dates are based on crossdated trees at various downstream locations and on intensive studies of all trees along several 1–2 km lengths of creek (M. J. Kauffman, *unpublished data*). While we show this estimated downstream movement in order to more completely characterize the invasion process, it should be noted that none of our analyses depend on this estimated rate of downstream movement.

Landscape features

To characterize factors that govern the spread of *Phytophthora lateralis* invasion across the landscape, we measured numerous features at each potential Road infection. We selected features that we believed may be related to the probability of infection entering a creek at a road crossing, such as factors associated with spore movement and viability, and the amount of contact of spore vectors with host targets. To estimate the amount of contact of roads with potential hosts, we measured the length of road surface within 15 m of the creek (''road crossing length''; Fig. 2). Using a GIS, we also calculated elevation, potential solar radiation (in joules per square meter) over seven peak hours of summer solstice (SOLARFLUX model; Rich et al. 1995), slope, and catchment area (amount of land drained to that particular point) for each road crossing. Catchment area reflects the amount of water that may be present in the creek. Catchment area may be important because many of the creeks in our study area become dry during summer, and because survival of *P. lateralis* spores depends on moisture (Hansen and Hamm 1996).

Because Nonroad infections are few and are not associated with particular, discrete routes (e.g., roads), we considered them separately from Road infections. We were interested in the changing spatial configuration of susceptible and infected host populations during the disease invasion, and in the spatial scale of dispersal for these two different types of infections. To measure

the spatial configuration of healthy and susceptible populations, we calculated the straight-line distance between each healthy stream population and the nearest infected stream segment in each year. To quantify differences between the two types of infection, we determined distance to the nearest potential source of infection for both Road and Nonroad infections. As with the identification of independent infection events, dates of infection were required for this component of the study. For each new infection, we calculated the distance from the top of the infection to the nearest infection that was already present in that year. In particular, we determined this distance using the shortest path from the top of the target infection to any infected creek segment in the study area. This calculation was done using ArcView Spatial Analyst version 2.0 (Environmental Systems Research Institute [ESRI], Redlands, California). Importantly, this method results in a conservative estimate of differences between Road and Nonroad infection distances for two reasons. First, our estimates underestimate the distance inoculum has traveled by vehicle (i.e., Road infections moved along roads, not in a straight line). Second, our estimate of distance for Road infections is a minimum estimate of dispersal distance for most infections, since we only include inoculum sources within the study area; actual dispersal distance could be greater if spores originated from an external source. Our experience suggests that there are no infections in watersheds directly adjacent to our study area that might have acted as sources closer than infections we used to estimate minimum distances.

Target host features

To characterize landscapewide variation in target host populations, we measured features of cedars at each road crossing. First, we counted the number of cedars within 15 m of the creek below roads (Fig. 2). This measure, referred to here as ''host abundance,'' reflects the number of hosts that are immediately available to become infected by spores deposited along the road. For this measure, we used only cedars $\geq$1 m in height because of the difficulty in counting seedlings. Also, we recorded the distance from the road surface to the nearest (first) cedar of any size along the creek (Fig. 2). Besides being a potential factor in disease incidence, this measure has practical applications for managers interested in determining the kinds of creek crossings at which to expect high risk; it is unknown if there is a distance too great for spores to travel downstream from roads and successfully infect cedar.

Statistical analyses

We used survival analysis to compare infection rates between creek populations susceptible to infection via Road and Nonroad vectors. Since this test allows for the use of time (such as time until infection) as a dependent variable, survival analysis increases the power to detect differences in infection risk between groups in many disease studies (Cantor 1997). For this analysis, we delineated individual creek segments having potentially independent infection fates. Each segment was bounded at their top and bottom by any creek confluence or road crossing, except in headwater areas where small branching stream segments were grouped to maintain similarity in drainage areas. We characterized each creek segment as to whether or not it was susceptible to infections transported along roads (i.e., crossed above by a road); all other creek populations were considered susceptible to infection only by foot traffic. Because of the conspicuous nature of Road infections (see *Methods: Defining infection sites*), we consider road crossings to only be susceptible to infection by vehicles. We used the Kaplan-Meier product-limit method to estimate the infection risk of these two groups (Cantor 1997; SAS Version 8.01). This method produces a survival function $S(t)$, which is the probability that an individual creek segment survives (escapes infection) for at least t years. Statistical significance was assessed using the log-rank statistic (Peto and Peto 1972).

To quantify differences in dispersal distances resulting in Road vs. Nonroad infection, we compared the minimum distance of spore travel (see *Methods: Landscape features*) for each infection type at the year of infection using a t test. To understand how the spatial dispersion of susceptible populations changed in relation to the location of infected populations, we calculated the distance to the nearest infection for each healthy and infected stream population in each year. For the healthy populations in each year, we further characterized the variation in distance to the nearest infection by calculating the 1st, 5th, 10th, 33rd, 66th, 90th, 95th, and 99th percentiles of the total distribution of nearest distances. To assess if minimum dispersal distances changed through time, we regressed minimum distance of spore travel for successful infections on year of infection.

Because most infections are directly below road crossings, a second goal of this work was to assess the attributes that facilitate *P. lateralis* invasion of cedar

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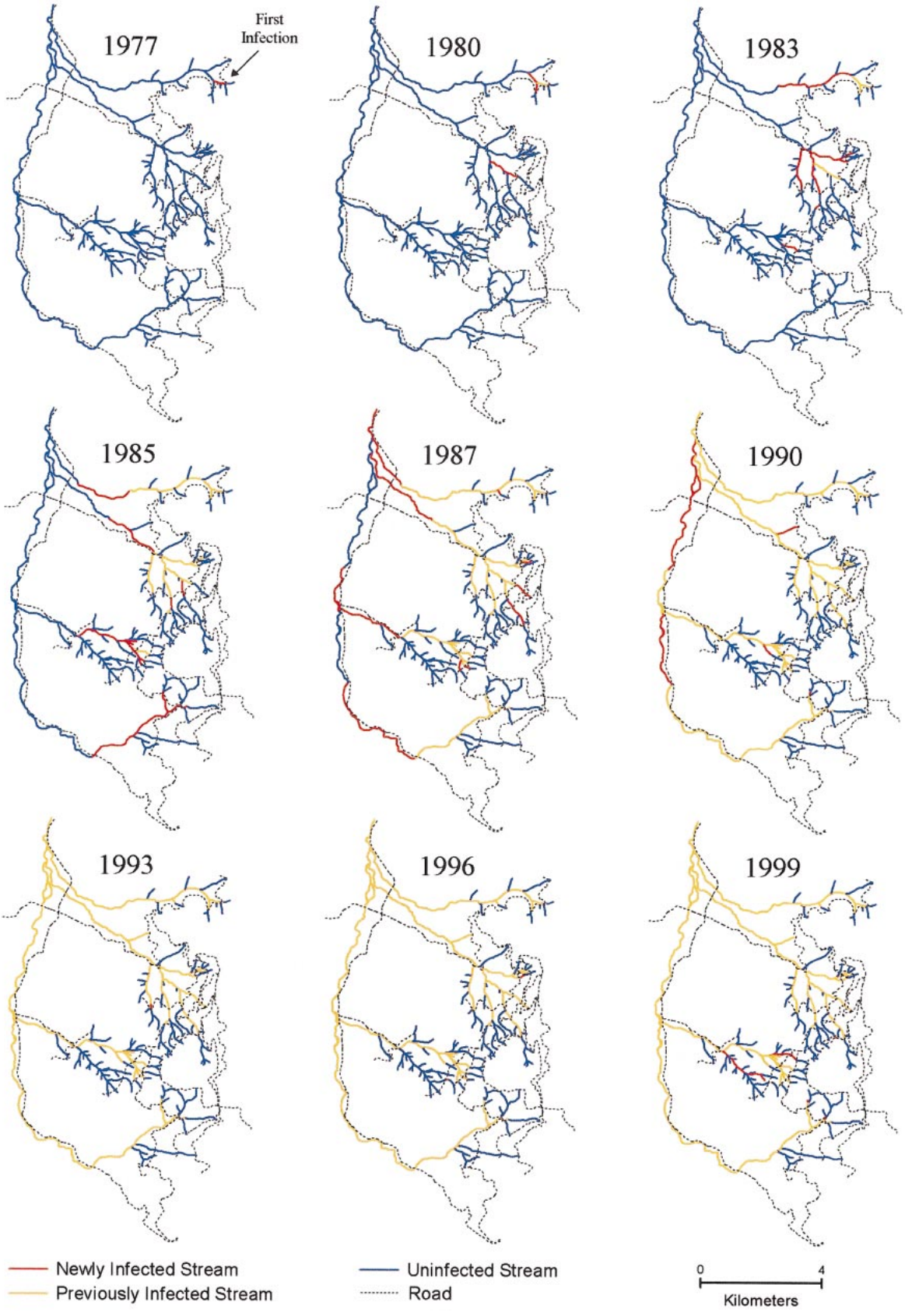
FIG. 3. Time-series maps indicating the spread of *Phytophthora lateralis* infection on Port Orford cedar across the Page Mountain study area, southwestern Oregon and northwestern California, USA, from 1977 to 1999. Note the initial 1977 infection in the northeastern corner of the map. Not all time intervals are equal. Intervals were selected to show most salient features of spread over the 23-yr period; 3-yr intervals are used in most cases, and 2-yr intervals during the peak infection period (1984/1985 and 1986/1987). Newly infected areas (red) indicate all infections initiated at any time during each time interval shown, and previously infected areas (yellow) indicate infections initiated in any previous interval.


1977
First Infection
1980
1983
1985
1987
1990
1993
1996
1999
Newly Infected Stream
Previously Infected Stream
Uninfected Stream
Road
0
4
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populations in creeks that are contacted by roads. For this subset of cedar populations, we again used a type of survival analysis to detect attributes of host stands that significantly influence infection risk. In this case, we used proportional hazards regression, which uses a hazard function defined as $h(t,\mathbf{x}) = h_0(t) \exp(\boldsymbol{\beta}'\mathbf{x})$, where $h_0(t)$ is the baseline hazard function and $\mathbf{x}$ is a vector of covariates (SAS procedure PHREG; Cantor 1997). The fitted hazard function was used to obtain the survival function $S(t)$, which describes the probability of a cedar population escaping infection for at least t years based on population-level attributes measured in the field (Fig. 2). Explanatory variables tested included elevation, solar radiation, slope, catchment area, host abundance, distance to the nearest cedar, and road crossing length. Significance of explanatory variables was assessed using likelihood ratio tests for pairs of nested models that varied only in the inclusion of the parameter of interest (Hilborn and Mangel 1997). The statistic used for this test is $-2(\mathcal{L}_{\text{reduced}} - \mathcal{L}_{\text{full}})$, where $\mathcal{L}$ denotes the log-likelihood of the observed survival data given the full or reduced model; for all comparisons, the same full model was used, which included all explanatory variables. This test statistic is distributed as a χ^2, with degrees of freedom equal to the difference in the number of parameters included in the two compared models. We further explored the contribution of each of the significant covariates to infection risk by manipulating the fitted survival function. To do this, we varied each explanatory variable independently across a range of values found in the study, while holding all other variables at their mean values (SAS procedure PHREG; Cantor 1997).

Assessment of previous monitoring

Newly emerging diseases often force managers to utilize coarse surveys of disease distribution and a limited understanding of the biology of dispersal. Thus, we employed our census data to assess one such example of a coarse survey. The USDA Forest Service has been monitoring the extent of *P. lateralis* across federal lands occupied by Port Orford cedar (Greenup 1998). The extent and thoroughness of these surveys varies across the four National Forests that contain Port Orford cedar (Siskiyou, Rogue River, Six Rivers, and Klamath National Forests; D. Kroeger, *personal communication*). In some areas, for example, surveys were coupled with extensive vegetation surveys and may be quite accurate. In many other areas, most infections of cedar were mapped using surveys done by driving along forest roads, or by experienced Forest Service personnel who have extensive knowledge of particular areas (L. Pera, *personal communication*). In the Page Mountain study area, the Forest Service survey data note infections found, mainly through roadside surveys, up to 1995.

We compared the USDA Forest Service data with our own, using infections that would have been visible in 1995. Because no new infection occurred in 1992–1994, we were not concerned with latent infections that were not visible to surveyors in 1995. Specifically, we noted the proportion of Road infection sites not found by the USDA Forest Service, Nonroad infection sites not found, and the number of uninfected sites that were misidentified as infected. Because surveyors could not identify independent infections as we did using infection dates, we considered all road crossings that were infected in 1995, regardless of whether they were independent infections or those caused from downstream movement of spores. We also determined the number of Nonroad infections that were detected by 1995 in the USDA Forest Service survey.

RESULTS

Census results: disease, cedars, and roads

Port Orford cedar was distributed widely across the study area, occupying areas adjacent to almost every creek censused (Fig. 1). Approximately 63 linear kilometers of creek was occupied by cedar. This length, however, is an estimate because we did not locate the highest occurrence of cedar in creeks that were flowing from the top of watersheds with no roads above (i.e., headwaters). The few creeks without cedar were dry, south-facing creeks (not shown on Fig. 1). Cedar were found at most elevations within the study area, with the exception of high ridges forming the area boundaries.

Of the 63 km of creeks with cedar, *Phytophthora lateralis* infection was found along 29 km (46%; Fig. 1). No major watershed within the study area was free of the pathogen. The majority of independent infection events (26 of 36 events = 72%) began directly at locations where roads cross creeks (Road infections). Of the 86 road crossings of cedar-occupied creeks, 37 (43%) were infected by 1999. Of the total of 65 creeks without road crossings, ten (15%) were infected (Nonroad infections). Even though Nonroad infections were not in direct contact with any roads, all of these infections had a road somewhere uphill, above them. That is, only creeks in headwater positions, above the entire road system, always remained uninfected (Fig. 1).

Spread of infection through time

We were able to determine the earliest infection date for almost all potential infection initiation locations. Of the 436 dead trees sampled, 43 trees were observed dying during the course of the study and cores from 10 trees were too damaged to attempt crossdating. Of the 383 remaining trees, 81.7% (313 trees) were crossdated with high confidence (i.e., crossdated with certainty using both visual and statistical methods). These trees afforded an average of 196 annual ring-width measurements used in the crossdating (range 59–486 yr). Importantly, in all but two locations, most of the six trees cored were crossdated with high confidence,

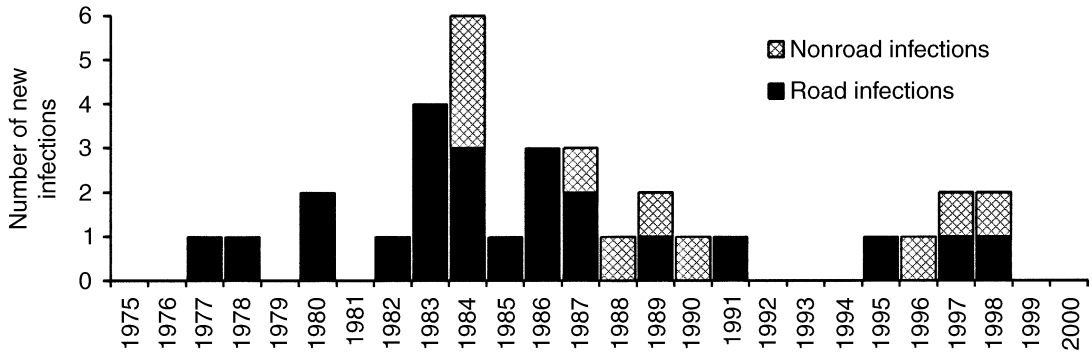

FIG. 4. The number of new independent infection events of *Phytophthora lateralis* on Port Orford cedar occurring between 1977 and 1999. Infections that traveled by vehicles (Road infections; solid bars) and animals/hikers (Nonroad infections; hatched bars) are distinguished.

allowing for a satisfactory date to be determined. The two locations where trees were too young for infection to be determined were omitted from both survival analyses.

Cedars used in the crossdating analysis ranged up to 634 yr in age, and the master chronology included growth information for dates starting in the year 1356. Crossdating revealed that some standing dead cedar in the site had not died of infection by *P. lateralis* and showed the longevity of decay-resistant Port Orford cedar. For instance, we crossdated (with high statistical confidence) one tree that had been dead since 1738. Similarly, several other trees died >100 yr before we collected our cores (e.g., 1858, 1890, 1895).

The study area was free of infection in 1976 and was infected by an outside source the following year (Fig. 3, northwest corner). It is unclear where the initial infection came from, although by 1977 infection was common in coastal areas of Oregon and probably inland sites closer to our study area. After the initial 1977 infection, the next infection was nearby along the same road in the same watershed (Fig. 3). A longer distance movement occurred in 1980, when a new infection was initiated in the watershed south of the first infection (Fig. 3). Between 1977 and 1999 the disease spread over much of the study area. The first Nonroad infections occurred in 1984 (Figs. 3 and 4). In total, we identified 36 independent infection events (Fig. 3). Downstream movement of infection proceeded from these infected sites throughout the 23-yr period (Fig. 3). In eight cases, a creek crossed by two roads was infected first at the lower crossing and later at the higher crossing.

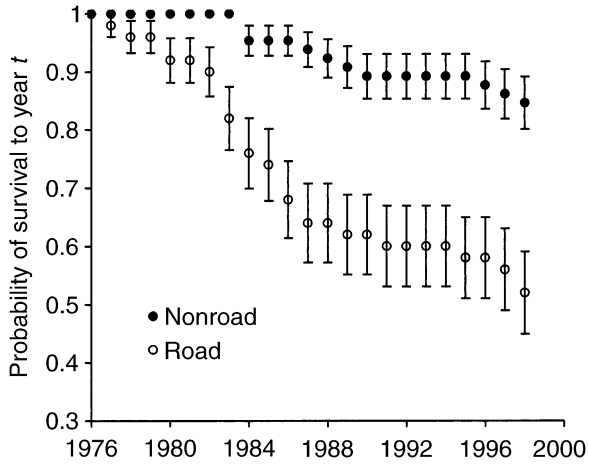

FIG. 5. Kaplan-Meier survival estimates for creek populations of Port Orford cedar in the Siskiyou Mountains of southwestern Oregon. The vertical axis denotes $S(t)$, the probability of escaping infection until the year t. Survival estimates are shown separately for those populations capable of receiving spores from roads and those that are not potentially infected via roads. Error bars indicate ±1 SE.

Statistical analyses

Survival analysis showed a significant difference in infection probability between creek populations susceptible to Road ($n = 50$ populations) and Nonroad ($n = 65$ populations) vectors (log-rank $\chi^2 = 16.33$, df = 1, $P < 0.0001$). Kaplan-Meier survival curves illustrate a longer mean survival time for sites which were not vectored by roads (Fig. 5). Minimum dispersal distances were also significantly different for these types of infections. The average distance to the nearest infection at the year of infection was 758 ± 193 m ($n = 23$ infections) for Road-vectored infections, and 168 ± 24 m ($n = 9$ infections) for Nonroad-vectored infections ($t = -3.03$, df = 23, $P = 0.003$). As well, throughout the 23 yr of invasion, the distance from infected sites to all susceptible, uninfected sites declined (Fig. 6), and the minimum distance traveled to new infections declined significantly ($r^2 = 0.223$, $P = 0.006$; Fig. 6).

Proportional hazards regression conducted on the subset of stream populations that were crossed by roads revealed the contribution of population-level attributes to invasion risk (Tables 1 and 2). The best-fit model showed strong influences on infection rates due to catchment area, host abundance and distance from the

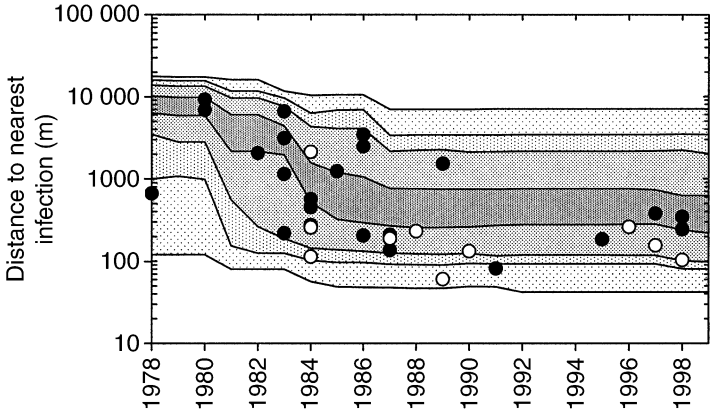

FIG. 6. Distance from all susceptible Port Orford cedar populations to the nearest *Phytophthora lateralis* infection from 1978 to 1999. Shaded sections indicate percentiles of the distribution of all susceptible populations (1st, 5th, 10th, 33rd, 66th, 90th, 95th, and 99th percentiles from bottom to top). Circles indicate the distances from populations that became infected that year to their nearest existing infection. Closed circles indicate Road infections, and open circles indicate Nonroad (foot traffic) infections.

road to the nearest cedar. Each of these factors was significant based on likelihood ratio tests on pairs of nested models (Table 2). Incidence of infection was positively associated with measures of drainage area and host abundance, and negatively associated with distance to nearest tree (Table 1). None of the additional explanatory variables (crossing size, elevation, solar radiation and slope) were significant (Table 2). It is important to note, that host abundance and distance to nearest tree were correlated (Pearson's correlation; rho $= -0.317$, $P = 0.006$). However, we tested the effect of each variable on a reduced model which included the effect of the other correlated variable, indicating that both measures are significant predictors of infection risk. Exploration of the fitted survival function suggests that all three significant variables play a meaningful role in controlling infection risk (Fig. 7).

Assessment of previous monitoring

U.S. Forest Service surveys of the Page Mountain study area were strikingly different than our census of the same area. Their survey revealed 15 of the total 31 (48%) infections present at road crossing in 1995 (Fig. 8). One of seven (14%) Nonroad infections were found, and 3 uninfected locations were misidentified as infected (Fig. 5). In total, 16 out of 38 (42%) infections were detected in the U.S. Forest Service survey.

DISCUSSION

We were able to successfully reconstruct the invasion of *Phytophthora lateralis* across a variable landscape of Port Orford cedar populations. By analyzing the spatial chronology of this disease invasion, we were also able to identify two different scales of pathogen dispersal that are important in mediating the rate and pattern of invasion. The network of roads serve as routes for the primary dispersal vectors (vehicles) connecting populations over landscape-scale distances, while local, small-scale invasions are maintained by short-distance dispersal via foot traffic. Furthermore, the success of long-distance invasion events along roads are clearly mediated by attributes of the connected populations. Specifically, our study shows that successful invasion of *P. lateralis* was higher in cedar populations that had high creek flows and dense stands of hosts in close proximity to road vectors.

Road vectors and dispersal limitation

Roads have been regarded as important in numerous other systems in which they facilitate dispersal of non-

TABLE 1. Summary statistics for road crossings containing Port Orford cedar that are either uninfected or infected by *Phytophthora lateralis*.

		Uninfected ($N = 48$ catchments)		Infected ($N = 26$ catchments)	
Measure	Units	Mean	1 SE	Mean	1 SE
Distance to nearest tree	meters	**117.7**	**32.0**	**10.5**	**6.3**
Host abundance	no. trees	**6.3**	**1.6**	**18.5**	**3.2**
Slope	degrees	14.2	0.9	13.8	1.3
Crossing size	meters	40.5	1.9	43.5	4.3
Elevation	meters	1157.3	27.1	1082.0	31.4
Catchment area	km^2	**1759.3**	**368.9**	**3924.5**	**910.6**
Solar radiation	joules/m^2	1.48×10^7	1.40×10^5	1.44×10^7	2.53×10^5

Notes: Infected crossings summarized here are only those crossings that were infected by independent infection events via vehicles, not infection that arrived in flowing water from upstream infection. Factors in bold indicate those with significant effects on infection risk (see Table 2).

TABLE 2. Proportional hazards regression model of *Phytophthora lateralis* infecting road populations of Port Orford cedar.

Explanatory variable	Log-likelihood	χ^2	df	*P* value
Distance to nearest tree	−97.342	3.927	1	**0.0475**
Host abundance	−98.262	5.767	1	**0.0163**
Slope	−95.652	0.546	1	0.4600
Crossing size	−95.501	0.244	1	0.6213
Elevation	−95.379	0.001	1	0.9748
Catchment area	−98.195	5.633	1	**0.0176**
Solar radiation	−95.782	0.806	1	0.3693
Full model	−95.379	23.4093	6	0.0007

Notes: Results are of likelihood ratio tests for significance of explanatory covariates. For each explanatory variable, the log-likelihood is shown for the reduced model, which excludes that variable. The likelihood-ratio χ^2 value is calculated as $-2(\mathcal{L}_{\text{reduced}} - \mathcal{L}_{\text{full}})$. The reduced model used to test the full model has no covariates and a log-likelihood value of −107.205. Factors in bold have significant effects on infection risk.

native species and pathogens (Tyser and Worley 1992, Wilson et al. 1992, Parendes and Jones 2000). However, quantification of these effects has only rarely been carried out. Of the 36 independent infection events we found, 26 (72%) were vectored along roads. Due to their higher susceptibility, infection of creek segments below roads was not only more common, but occurred earlier in the 23 yr of infection (Figs. 3 and 4). The first Nonroad infection occurred 7 yr after the initial 1977 infection; during that same period 11 additional infections were initiated along the roads. All roads except one in the study area were built to aid in timber harvesting. Because large vehicles and heavy equipment associated with timber harvesting are suspected to transport more mud and organic material than passenger vehicles, timber harvest activity is often thought to increase disease incidence (see Hansen et al. 2000). Interestingly, there were no infections during the period 1992–1994 (Fig. 4), a period that coincides with a cessation of timber harvest due to legal injunctions concerning the northern spotted owl (*Strix occidentalis*; Marcot and Thomas 1997). This decline in new infections is partially illustrated in Fig. 3 (1993 and 1996 maps).

While less important then roads, foot traffic also vectored *P. lateralis* (Figs. 1 and 4). All of these infections appeared to be in areas into which people or animals could easily transport disease. In fact, all of the Nonroad infections were within the system of roads in the study area; no infection was found in roadless watersheds or in headwater sites above all roads. One Nonroad infection began in an apparent bear wallow, along a well-used wildlife trail.

Dispersal of infection via roads occurred over greater distances than those that moved via foot traffic (Fig. 3). Infections via roads moved up to 3.8 km, while the longest distance that infection traveled via foot traffic was 264 m. Importantly, the apparent transport between

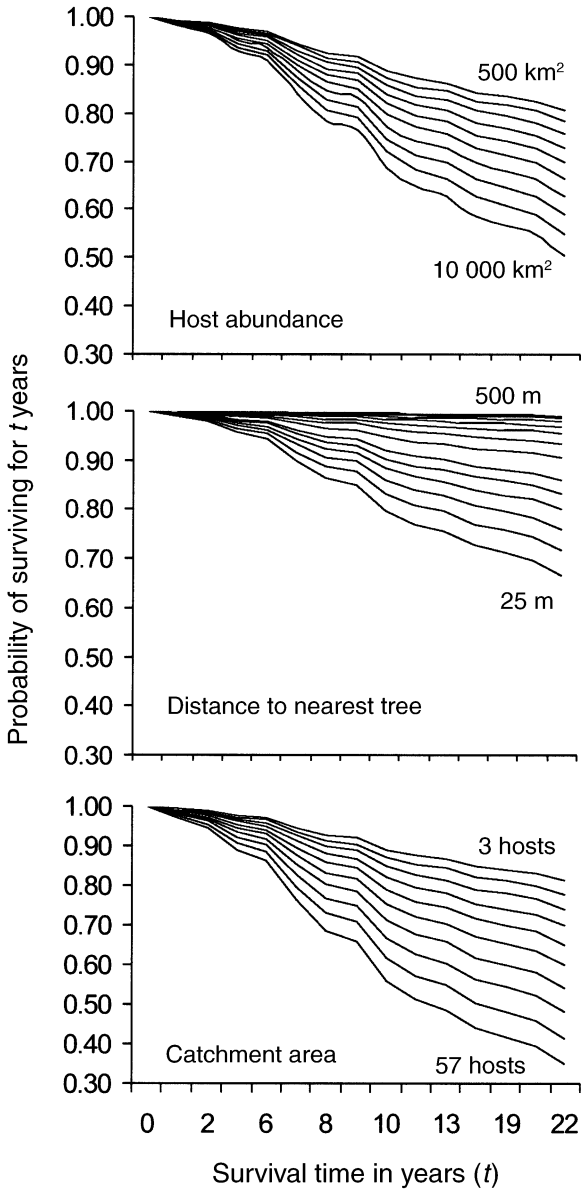

FIG. 7. Expected survival functions $S(t)$ for host populations which vary in their physical attributes. Each survival function estimates the probability of a cedar population with the given attributes escaping infection for at least t years. Functions were created by varying individual covariate values while holding other covariate values at their means: catchment area = 2520.01 km², distance to nearest tree = 80.05 m, and host abundance = 10.55 cedars. Catchment area was varied from 500 km² to 10 000 km² in increments of 500. Distance to nearest tree was varied from 25 m to 150 m by 25-m intervals, and from 150 m to 500 m by 50-m intervals. Host abundance was varied from 3 to 57 cedars in increments of 6 cedars.

the four subwatersheds in the study area (Fig. 1) always occurred by vehicles moving along the road system. Foot traffic appears to be a more localized phenomenon, acting diffusely to ''fill-in'' uninfected sites surrounding infected creeks after development of the ep-

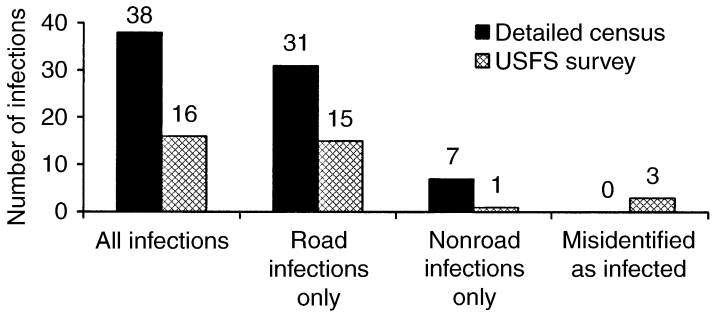

FIG. 8. Summary results of a comparison of a U.S. Forest Service survey and the census performed in the Page Mountain study area, southwestern Oregon and northwestern California, USA.

idemic throughout the landscape. The fact that these short-distance, nonroad infections did not occur until after the seventh year of this disease invasion is presumably a function of the number and spatial configuration of infected creeks (inoculum sources) relative to susceptible stream populations (Fig. 6). Our measure of spatial configuration seems to support this notion as well. First, and not surprisingly, the average distance to nearest infection declined throughout the study for all susceptible populations (Fig. 6). Second, the susceptible populations that did become infected were located more or less randomly across the nearest-neighbor distribution in the first half of the epidemic, but tended to occur at the shorter distances later on (Fig. 6).

Factors influencing host population invasibility

Invasion was also strongly mediated by variation in physical attributes and spatial configuration of cedar populations that were crossed by roads. Risk of infection for these creek populations increased with increasing catchment area, increasing cedar abundance, and decreasing distance to the nearest cedar located below the road crossing.

Catchment area was a highly significant factor affecting survivorship in the proportional hazards regression (Table 2, Fig. 7). Catchment area is most directly an indicator of streamflow in each creek. Crossings with high catchment areas were more likely to have flowing water during the dry summer months, while low catchment area sites were often seasonal (i.e., fall through spring) creeks (authors' *unpublished data*). That sites with large creeks or more persistent creeks are more likely to become infected fits well with the biology of *P. lateralis*, because high-flow creeks create higher quality habitat for deposited spores. Further, higher levels of moisture throughout the dry season increase both the length of the season that successful infection is possible, and the rate at which deposited resting spores may survive from one wet season to the next (Hansen and Hamm 1996).

Abundance of the host species and its proximity to road surfaces also act to govern the spread of the invasion along the road system. Both host abundance and distance to the nearest tree were significant variables in the proportional hazards regression model (Table 2). Expected survivorship curves illustrate that the natural levels of variation in host abundance that we recorded may have large effects on risk of population infection (Fig. 7). When only a few hosts are present, the probability of a creek population remaining uninfected over our 23-yr study is over twice as likely (3 hosts = 81%) than when numerous individual hosts are present (57 hosts = 35% probability of escaping infection; Fig. 7). Most likely, this is a result of *P. lateralis* dispersal events that, while probably quite common across the landscape, deliver concentrations of propagules small enough to limit their probability of root contact and subsequent infection.

Model predictions also show that the distance to the nearest host from the presumed dispersal source (road surfaces) can affect infection probability over short distances. Indeed, the maximum observed distance that spores had successfully dispersed off a road surface in our study was 165 m. Model projections further indicate that stream populations located more than 200 m from the road surface have only a 10% probability of becoming infected, while populations located >400 m have little or no chance of infection. Successful spore dispersal in flowing water most likely follows a diminishing function with distance from source, controlled by the rate at which spores settle out of moving current, or the physical stress of water transport, which may render unsettled spores nonviable. It is important to also note that since these two measures of host abundance and distance are correlated, actual infection rates will vary more dramatically than we have estimated using mean (and fixed) covariate values.

Considerable debate exists on the relative importance of dispersal limitation and site susceptibility for governing the spread of invading organisms (Shurin 2000, Tilman 1997). Some researchers have suggested that dispersal limitation will be more important than local characteristics in the early stages of invasion, with site characteristics becoming more important after the invader becomes more common and dispersal is no longer limited (Wiser et al. 1998). In their 23-yr study of *Hieracium lepidulum*, Wiser et al. (1998) found that site characteristics explained more of the variation in invader distribution as the invasion progressed. We do

not test this relationship directly in our study; however, two site factors important in this study—host abundance and distance to nearest tree—will strongly interact with dispersal limitation.

We also document two important types of heterogeneity in this system: variable ''contact rates'' between infected and susceptible host populations and variable levels of susceptibility to infection among healthy populations. Both of these factors are likely to be important for disease dynamics in this system (Bolker et al. 1995). The first source of heterogeneity—variable contact rates—is due to the ways in which *P. lateralis* spores can be transported. An infected stream population can spread disease to nearby neighbors via foot traffic or it can come into contact with relatively distant neighbors through vehicle-mediated dispersal along the road network. The important result derived from other disease systems where variable contact rates exist (e.g., HIV in humans [May and Anderson 1988]; fox rabies [White et al. 1995]; foot-and-mouth disease [Ferguson et al. 2001]) is that the upper range of contact rates, not the mean level of contact, most strongly determines rates of spread. For example, Ferguson et al. (2001) demonstrated that the recent epidemic of foot-and-mouth disease across the Great Britain countryside expanded rapidly at first due to high rates of contact of infected animals with other susceptibles at livestock auctions. Similarly, large-scale pathogen dispersal along roads in the cedar–*Phytophthora* system is likely to be more important than mean dispersal distances in governing invasive spread rates in currently uninfected regions.

The variation in susceptibility to infection at creek crossings creates a second form of heterogeneity among host populations. We have shown that this type of spatial heterogeneity will be important in regulating current rates and patterns of disease spread (Fig. 7) and work on other systems suggests it will play a role in the long-term maintenance of pathogen endemicity as well (Bolker and Grenfell 1995). For instance, in currently uninfected watersheds, the number or severity of new infections needed to satisfy threshold conditions for new epidemics may be higher due to such host patchiness (Onstad and Kornkven 1992). Alternatively, these levels of heterogeneity may act to stabilize host–pathogen dynamics, enhancing the likelihood of endemic, long-term infections of this nonnative pathogen (Huffaker 1958, Thrall and Antonovics 1995, Bolker and Grenfell 1996).

Future spread and management considerations

Study of *P. lateralis* invasion through time suggests that we may expect a continued spread of the disease in the Page Mountain study area. Vehicles traveling along roads will continue to spread inoculum as there are many uninfected susceptible creek crossings. The U.S. Forest Service has instituted seasonal road closures (i.e., gating of roads in the wet season) and this may slow the spread. However, even if all roads were permanently closed to vehicular traffic, we believe that human and animal foot traffic would continue to spread the disease into uninfected creeks, albeit at a much slower rate. Importantly for rangewide considerations of Port Orford cedar, our data suggest that isolated, roadless watersheds (not found within our study area) may have extremely low risk of infection, because there is probably a limit to how far foot traffic can disperse *P. lateralis* spores. Maintaining areas free of vehicle traffic is likely to be an effective strategy for slowing *P. lateralis* invasion. Our results also help explain why spread of the pathogen was observed to be rampant when it was first introduced into the cedar's range in 1952. *Phytophthora lateralis* first invaded the moister coastal portion of the range, where cedars are not restricted to riparian areas, and we suspect that two significant factors we studied contributed to higher susceptibility of hosts. In general, host abundance near road crossings is greater in coastal populations and distance to the nearest tree is lower. These high-risk factors help explain the fast rates of *P. lateralis* movement in coastal populations.

A commonly suggested and employed management technique for slowing the spread of *P. lateralis* has been the reduction of cedar density (i.e., harvesting of cedar) in potentially infected sites near roads (see Goheen 2000). Although our results show that lower densities of cedar near crossings are strongly associated with decreased disease risk, the efficacy of harvesting healthy cedar is suspect for several reasons. Most importantly, the intensity of management effort needed to bring about substantive reductions in infection risk is rarely employed and difficult to achieve. Specifically, reducing host abundance and distance to the first tree to the extent needed to prevent infection with certainty is impractical and unrealistic (Fig. 7). Moreover, since Port Orford cedar easily recruits abundant seedlings in moist areas near roads (Greenup 1998, Hansen et al. 2000), maintaining cedar-free areas is likely to be impractical.

Managers creating strategies to slow *P. lateralis* spread that utilize existing U.S. Forest Service maps should be aware of potential shortcomings of past surveys (Fig. 8). In most areas of infection, managers should expect higher risks of spread. Similar cautions are probably appropriate for managers confronted with the numerous other forest pathogens currently spreading across large areas (Kelly 2001, Svihra 2001).

Conclusions

The rate and extent of invasion by *Phytophthora lateralis* across our study landscape was influenced by several factors that have been previously predicted to be important by invasion ecologists. Both physical landscape features (i.e., sufficient water) and biotic features (i.e., suitable host populations) appear to mediate spread. The heterogeneity of these factors amongst host

populations has, in part, governed the historical spread of this disease invasion. That many target populations are at low risk, and currently remain uninfected, indicates that the current distribution of the pathogen is a poor metric of long-distance dispersal events. The movement of *P. lateralis* on vehicles has probably resulted in many importations into creeks that failed to produce infection. Dispersal by smaller scale vectors, such as foot traffic, was probably limited by characteristics of susceptible populations, although we could not test this directly. Importantly, however, our study shows that the short-distance movement in a given area is first initiated by necessary dispersal over long distances.

Our study points to the importance of focusing attention on the spatial structure of potential invasion sites and its effect on invasions in general. This is especially important for disease invasions for two reasons. First, most disease invasion studies to date have only assessed spread across homogeneous populations and landscapes. Second, what limited empirical work exists, including ours, suggests that variation in susceptibility will be an important trait mediating spread. Our study is unique in that we were able to quantify variation in susceptibility resulting from physical factors of the landscape as well as population-level attributes of the host. Finally, the power of our analysis of spread was increased markedly because we were able to reconstruct invasion history from the time of the initial infection. Furthering our understanding of what governs invasion in other systems will likewise benefit from coupling accurate spread histories with measures of heterogeneity across landscapes.

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