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HETEROGENEITY SHAPES INVASION: HOST SIZE AND ENVIRONMENT INFLUENCE SUSCEPTIBILITY TO A NONNATIVE PATHOGEN

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Abstract. Theoretical study of invasion dynamics has suggested that spatial heterogeneity should strongly influence the rate and extent of spreading organisms. However, empirical support for this prediction is scant, and the importance of understanding heterogeneity for real-world systems has remained ambiguous. This study quantified the influence of host and environmental heterogeneity on the dynamics of a 19-year disease invasion by the exotic and fatal pathogen, *Phytophthora lateralis*, within a stream population of its host tree, Port Orford cedar (*Chamaecyparis lawsoniana*). Using dendrochronology, we reconstructed the invasion history along a 1350-m length of infected stream, which serves as the only route of pathogen dispersal. Contrary to theoretical predictions, the temporal progression of the disease invasion was not related to a host's downstream spatial position, but instead was determined by two sources of heterogeneity: host size and proximity to the stream channel. These sources of heterogeneity influenced both the epidemic and endemic dynamics of this pathogen invasion. This analysis provides empirical support for the influence of heterogeneity on the invasion dynamics of a commercially important forest pathogen and highlights the need to incorporate such natural variability into both invasion theory and methods aimed at controlling future spread.

Key words: disease invasion; environmental heterogeneity; host heterogeneity; invasion ecology; nonnative pathogen; spatial spread.

INTRODUCTION

Empirical studies of biological invasions have provided considerable evidence that invadable sites are heterogeneous in their susceptibility to invasion. In fact, this is perhaps the most ubiquitous and general result from a large and growing body of work (Moyle and Light 1996, Stohlgren et al. 1999, Mack et al. 2000, Ferguson et al. 2001). Thus, for most scenarios of invasive spread, we should expect strong heterogeneities to exist that can influence the rate and pattern of invasion. And yet, there are still relatively few studies that have attempted to quantify the influence of spatial heterogeneity on the invasion process (Rouget and Richardson 2003, Hastings et al. 2005). This lack of data is fast becoming a hindrance to the development of predictive and useful models of spread. Moreover, lack of empirical support has meant that the importance of including measures of heterogeneity in studies aimed toward management of invasive species remains unclear.

One important reason for the scant number of studies that carefully assess the role of spatial heterogeneity in the invasion process is the difficulty of following spread across a meaningful temporal scale, while simultaneously quantifying important sources of heterogeneity (Smith et al. 2002, Rouget and Richardson 2003). In the work presented here, we have used a 19-yr spread history of a nonnative plant pathogen to study invasive spread over a naturally complicated landscape, where variation in host susceptibility represents an important source of heterogeneity capable of mediating the invasion process. Disease invasions are unique among biological invasions due to the dependence of the pathogen on a native host. However, when a pathogen is first colonizing new hosts, disease spread is driven by pathogen production, dispersal, and host susceptibility, factors which are equally important to non-disease invasions (Mollison 1986). Additionally, there are numerous sources of heterogeneity that have the potential to mediate the rate and pattern of plant disease invasions (Colhoun 1973, Burdon et al. 1989). Among the factors influencing susceptibility are environmental conditions (Jarosz and Burdon 1988), genetic variation (Parker 1985, Burdon and Jarosz 1991, Alexander and Antonovics 1995), and physical factors such as host size (Roche et al. 1995, Morrison 1996).

While the role of various sources of heterogeneity remains largely unexplored, the rich history of research on plant pathogens in agricultural systems does provide some important insights. For example, spatial models evaluating the utility of crop mixtures indicate that the velocity of pathogen spread should increase with the proportion of susceptible plants (van den Bosch et al.

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1990). In a large-scale field test of these ideas, Zhu and co-workers (2000) found lower disease levels in fields planted with mixtures of resistant and susceptible cultivars. However, in natural systems host and environmental heterogeneity often vary continuously and may lead to complex dynamics different than those predicted for agricultural systems (Alexander 1989).

One of the most intriguing and general results from work on nonplant diseases is the prediction that heterogeneity in transmission rates has a stronger effect on disease progress than that of mean levels. For example, in the study of HIV in humans, May and Anderson (1988) have shown that heterogeneous sexual behavior will speed up epidemic progress initially but will also lead to more sustained infection rates after the peak of the epidemic (i.e., at the tail of the distribution). In one of the few studies in natural disease systems where host heterogeneity has been investigated empirically, Dwyer et al. (1997) have shown that variation in host susceptibility leads to more strongly nonlinear transmission dynamics of the nuclear polyhedrosis virus in gypsy moths (*Lymantria dispar*). In this system, host heterogeneity acts to facilitate epidemic development due to the presence of a few highly susceptible hosts. Interestingly, heterogeneity in host susceptibility also acts to stabilize longer-term disease dynamics in this system (Dwyer et al. 2000). Similarly, heterogeneous behavior (in this case, movement) of a few individuals can increase invasion speed compared to that predicted by average behavior alone (Goldwasser et al. 1994).

How important are such sources of heterogeneity and how general is their influence in natural systems? To inform the further development and validation of invasion models, we still need to characterize, in a more formal and quantitative fashion, the degree to which heterogeneities govern invasion dynamics. In this study, we utilized standard tools of dendrochronology (Douglas 1934) to reconstruct a 19-yr invasion by the introduced root pathogen, *Phytophthora lateralis*. The invasion was reconstructed within a large stream population of the pathogen's only common host tree in western North America, the native conifer, Port Orford cedar (*Chamaecyparis lawsoniana*). Port Orford cedar is restricted to riparian corridors in our study area, and the pathogen is dispersed downstream in flowing water, infecting new hosts along the stream bank as the epidemic progresses and killing all hosts that become infected. Using this reconstructed invasion history, we sought to quantify the influence of two distinct forms of heterogeneity: (1) differences in host size and (2) proximity to the stream, on the spatial and temporal dynamics of invasion.

Material and Methods

Study system

Port Orford cedar is endemic to northwestern California and southwestern Oregon, where it frequently attains sizes of 100 cm diameter at breast height (maximum, 3.8 m; Zobel et al. 1985) and is an important component of riparian forests (Hansen et al. 2000). The cedar is the primary North American host of *P. lateralis*, which was first found in the northern part of the cedar's range in 1952 (Roth et al. 1957). Although the geographic origin is unknown, *P. lateralis* is clearly nonnative (Hansen et al. 2000) and has spread throughout much of the cedar's range since the time of its introduction (Zobel et al. 1985). The pathogen attacks the cedar's root system, killing seedlings in two to three weeks and larger trees in one to eight years, by girdling the tree's primary root collar (Zobel et al. 1985, Kauffman 2003). To date, no evidence exists for fully resistant cedar genotypes; once infected, all trees eventually die (Hansen et al. 2000).

Long-distance spread between cedar populations found in different watercourses occurs as resting spores (oospores and chlamydospores) are moved between watersheds on vehicles and logging equipment (Jules et al. 2002). Local (within population) spread occurs when resting spores germinate and produce flagellate zoospores that disperse in surface water (typically beginning at a road crossing). In this study, we focus on the downstream invasion and epidemic development of *P. lateralis* within a single riparian population of cedars. Within a stream, infected host trees produce both resting spores and zoospores that are transported in running water to infect downstream trees (Zobel et al. 1985). Resting spores, which are likely produced by the pathogen until its host tree dies (Trione 1974), are capable of persisting, but not growing, in soils without a live host. Hansen and Hamm (1996) found that recovered isolates of the pathogen from buried root fragments declined sharply with time since infection, although a few of their samples continued to yield isolates up to the end of their 7-yr study. Following the introduction of the pathogen into a stream reach, the temporal development of a local epidemic is likely to be driven almost exclusively by the production of new inoculum from the first infected cedars and those downstream cedars that are subsequently infected.

Importantly for our work, the directionality of pathogen dispersal within an infected stream is highly predictable. Spores produced by infected trees only disperse in running water, spreading downstream via the stream channel; there is no effective dispersal by aerial means. While large mammals (such as elk) can vector the disease, this form of dispersal is thought only rarely to facilitate long-distance colonization events of uninfected stream reaches and not to substantially modify within-stream inoculum levels (Jules et al. 2002). Because spread across the larger landscape (i.e., between streams) is so conspicuous, it is possible to delineate the beginning of an isolated stream invasion.

Reconstructing infection history

We conducted a spatial and temporal reconstruction of the invasion of *P. lateralis* within an isolated pop-

ulation of Port Orford cedar along a mid-montane stream in southwestern Oregon, USA (Little Elder Creek, Siskiyou National Forest, 42°04′15″ N, 123° 33′34″ W). Little Elder Creek was infected in the late 1970s from a logging road that crosses the stream approximately one kilometer above our study area (Jules et al. 2002). Because the forested section of stream immediately below the road crossing was clear-cut logged in 1966, the study plot began just below the clear-cut.

To estimate the infection dynamics of hosts since the beginning of the invasion, we delineated a 1350 m long study plot as a narrow band encompassing cedars potentially at risk of infection on either side of the stream. The width of the study plot was set to include all trees growing within the portion of the stream canyon exposed to dispersing spores (typically no more than 10 m from the stream center on either side). Because epidemic spread has occurred in the stream since the late 1970s, we used the spatial distribution of infected cedars in combination with local stream topography to delineate the plot boundaries. The study plot was of variable width, being wider in areas with a large floodplain and narrower in stream sections with steep banks. Trees on the hill slope that were not exposed to dispersing spores, which comprised ≈50% of the total host population, were not included in the analysis.

We determined the disease status (healthy, dead, or dying due to previous infection) for each cedar tree within the infection zone based on visual signs of infection, including discoloration of foliage and the existence of a red-brown stain on the cambium tissue of recently infected trees (Hansen et al. 2000). Highly deteriorated snags or other dead trees with obvious causes of death (e.g., fire) were identified as natural mortality. Natural mortality of Port Orford cedar is quite low (M. Kauffman, *unpublished data*), and this long-lived conifer is capable of surviving >900 yr. Therefore, we assumed that all large and recently deceased trees (post-1980) without obvious signs of physical trauma were killed by *P. lateralis* infection. For each healthy and infected (dead or dying) tree, we measured its diameter at breast height (or alternatively, total height for trees <2.0 m tall) and mapped its spatial location using a Laser Rangefinder (Laser Technology, Enderson, Colorado, USA). Because it is the route for dispersing spores, we also mapped the center of the stream throughout the plot. Spatial locations of individual trees and the stream course were analyzed using routing methods in Arc/Info (version 7.2.1; ESRI, Redlands, California, USA) to calculate the relative distance down the length of the stream from a fixed point at the top of the study plot (furthest upstream location = 0 m). A proximity-to-stream measure was also calculated as the distance from the center of each tree (spatial locations were adjusted for tree diameter during field surveys) to the closest point on the stream route.

We extracted two core samples at 1.3 m height from opposite sides (stream side and hill side where possible) of each dead tree for dendrochronological analysis using standard methods (Stokes and Smiley 1968). Whole-stem (disc) samples were taken from dead trees too small to core. To avoid missing outer 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). Core samples were glued into wooden mounts, sanded to 300 grit, and ring widths were measured to 0.001 mm precision using a Velmex measuring device (TA 4030H1-S6; Unislide, Bloomfield, New York, USA).

To determine the calendar year of death for infected trees, we analyzed core samples using the dendrochronological method of crossdating, which relies on the shared climatic signature that trees display in their annual growth patterns (Douglas 1939). During drought years, Port Orford cedar usually produces small rings relative to the ring size of the years before and after such an event. Thus, for any dead cedar, it is possible to determine the date of death by aligning its ring-width pattern with the pattern derived from currently live, healthy trees, where the date that each ring was produced is known (Jules et al. 2002; see also Stokes and Smiley 1968, Swetnam et al. 1985 for details). Because the wood of Port Orford cedar is highly rot resistant (Zobel 1985), the annual growth rings of dead cedars remain intact for many years and can still be analyzed decades after death. Individual trees were crossdated either by aligning key ring-width patterns visually and/or by using the program COFECHA (Holmes 1983) to statistically compare the floating ring widths of dead trees to a master ring-width chronology. The master chronology was built initially from 24 cores from 19 living trees. As the study progressed, crossdated core samples from dead trees were added to the master chronology, which ultimately utilized chronologies from a total of 965 cores taken from 593 trees.

Because cedars do not necessarily die the same year they are infected, we assessed cores under a stereo microscope for initial signs of infection. For many infected trees, we were able to detect the beginning of a physiological response to the invading mycelium as a marked reduction in ring width and a relatively thin band of latewood several years prior to death (Jules et al. 2002). The beginning of this growth reduction, coupled with the crossdated year of death, was used to estimate the year of first detectable infection for each dead tree. Previous analyses of these data indicate considerable variation in the time from infection to death for infected trees (mean = 2.03 yr; SD = 2.26), with significantly longer time-to-death periods for larger diameter hosts (Kauffman 2003).

Statistical analyses

To summarize host population characteristics and invasion progress down the length of the stream, we di-

vided the stream into 10 135-m segments and tallied the cumulative proportion of trees in each segment that were infected in each year of the invasion. For each stream segment, we also plotted the infection year (or healthy status) of all host trees in relation to their diameter and proximity to the center of the stream channel. As a measure of downstream disease progression, we conducted a linear regression of stream distance (0 to 1350 m downstream) on the infection year of individual hosts for which an infection date could be determined.

We used proportional hazards regression to test for the influences of host and environmental heterogeneity on the probability of host infection during the invasion period (1981–1999). Proportional hazards regression uses time-to-infection as a continuous random variable. A hazard function defined as $h(t, \mathbf{x}) = h_0(t)\exp(\beta\mathbf{x})$ is fit to the survival data, where $h_0(t)$ is the baseline hazard function, $\mathbf{x}$ is a vector of explanatory variables, and β is a vector of fitted coefficients (SAS procedure PHREG; Cantor 1997). The hazard function, $h(t, \mathbf{x})$, is the instantaneous rate of change in the probability of death for a host with a vector of attributes $\mathbf{x}$, given that it has survived to time t. By integrating the hazard function over time, the survival function $S(t)$ is obtained, which describes the probability of an infected cedar remaining alive for at least t years as a function of its individual attributes.

Host heterogeneity was characterized by quantifying the effect of host size on infection risk, because saplings and small trees are expected to have lower infection rates than larger trees due to the smaller target that their roots present for dispersing spores. We assessed the influence of environmental heterogeneity by looking for effects of a host's position relative to the stream, with infection risk likely to decline with decreasing proximity to stream (a measure of soil condition and exposure to dispersing spores). Thus, in the fitted hazards model for this analysis, $\beta\mathbf{x} = \beta_1(\text{diameter}) + \beta_2(\text{proximity to stream}) + \beta_3(\text{interaction})$. To test for these effects, likelihood ratio tests were conducted between full and reduced models that excluded each parameter of interest. For infected trees, this analysis relies on knowing the year of infection. Thus, trees whose date of infection could not be determined by the crossdating method—mostly smaller trees with too few rings to crossdate accurately—were excluded from this analysis.

In a separate analysis, which included these smaller trees, we used logistic regression to assess the role of heterogeneity on the dichotomous disease status of all individual trees (healthy or infected in 1999). This analysis tested the same explanatory variables (host diameter, proximity to stream, and their interaction) as the proportional hazards regression. However, since the dependent variable is assessed after 19 years of epidemic development, it is less likely to be influenced by the stochastic realization of annual spatial spread. As for the prior analysis, the significance of explanatory variables was determined using likelihood ratio tests on nested models.

To assess how variation in susceptibility is distributed within the host population, we estimated the infection risk (as a function of diameter and proximity to stream) from the proportional hazards regression for all trees that were healthy when the invasion began. We estimated host infection risk on an annual basis as $\sqrt[T]{\theta}$, where T is the number of years of the study (19), and θ is the probability of infection by the end of the study in 1999. Thus, this analysis provides a picture of the underlying heterogeneity in infection risk that is inherent in the population of susceptible hosts before the invasion began.

Results

Crossdating and reconstruction

Overall, 555 trees within the infected stream segment were mapped, measured, and included within the plot boundaries. Of these, 243 (44%) were classified as having been previously infected by *P. lateralis* and 312 were healthy when surveyed in 1999. Of the previously infected trees, 156 (64%) were successfully crossdated and 87 (36%) were unable to be crossdated. In almost all cases, core samples that could not be crossdated came from young trees with too few annual growth rings for crossdating analysis.

Reconstructed dates of death revealed a large and persistent pulse of infection due to the *P. lateralis* invasion beginning in 1980, reaching a peak of 28 trees infected in 1985, and declining to only one or two trees becoming infected each year in the late 1990s (Fig. 1). Although biased downward, mortality dates from the standing dead snags in the study plot (i.e., trees dying prior to the *P. lateralis* invasion), suggest that the death rate from nondisease causes was far lower than that observed after 1980 when *P. lateralis* invaded this stream. A small cluster of non-*P. lateralis* mortality events in the mid to late 1960s was also evident, which was likely caused by the historic 1964 flood in this region (Fig. 1).

Within the 10 stream segments, there was considerable variation down the length of the stream in the distribution of healthy and infected hosts; available hosts varied as well in their diameter and proximity to the stream channel (Fig. 2). Disease progress curves generated for each stream segment did not show a discernable wave front of infection traveling down the length of the stream. Instead, most stream segments experienced a temporal increase in the number of new infectives from 1982 to 1988, a pattern that mirrored the development of the epidemic at the stream level (Fig. 1). Consistent with this pattern, the temporal progression of newly infected trees was not related to distance down the length of the stream (Fig. 2; linear

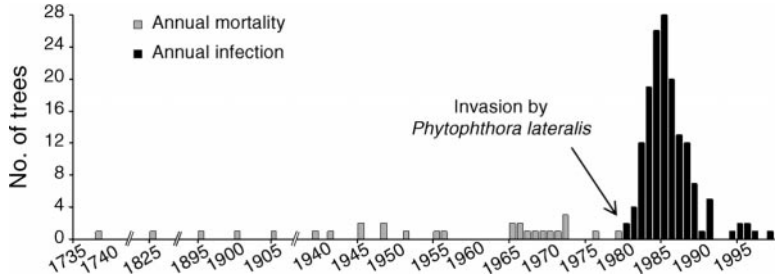

FIG. 1. Temporal development of an epidemic caused by the nonnative root pathogen, *Phytophthora lateralis*, invading a stream population of Port Orford cedar, *Chamaecyparis lawsoniana*. Based on dendrochronological analysis of tree rings, the first detectable infection from the pathogen occurred in 1980. Black bars indicate the number of trees newly infected each year. Gray bars indicate the historical nondisease mortality of dead snags still standing in the study plot.

regression of distance down the stream on infection year, $n = 156$, $F = 0.02$, $P = 0.88$, $R^2 = 0.0002$).

Effects of host and environmental heterogeneity

Both host and environmental heterogeneity had effects on the infection risk of cedar trees during the epidemic (Table 1). In particular, host diameter ($P < 0.0001$) and proximity to stream ($P < 0.0001$) were significant predictors in the proportional hazards regression, with infection risk increasing with host size and proximity to the stream. The interaction of these two factors was also significant ($P = 0.0205$), suggesting a stronger effect of proximity to stream for small hosts than for large hosts. This interaction effect is likely due to the extensive roots systems of large hosts that allow distant contact with stream inoculum. The effect of these sources of heterogeneity on epidemic development resulted in the largest and closest hosts to the stream becoming infected early in the epidemic, and the smaller and more distant hosts either remaining healthy or becoming infected later in the epidemic (Fig. 3). Predictions from the proportion hazards regression also indicate that, if rooted close to the stream, large hosts are over twice as likely to become infected as small hosts over the course of the epidemic (Fig. 4). However, for cedars rooted away from the stream, the increased infection risk due to larger diameter is greatly diminished. The logistic regression on the present-day distribution of healthy and infected trees ($n = 555$; including infected hosts that could not be crossdated) confirmed a significant effect of host size ($\chi^2 = 68.63$, df = 1, $P < 0.0001$), proximity to the steam channel ($\chi^2 = 6.36$, df = 1, $P = 0.0117$), and the interaction between these two factors ($\chi^2 = 20.78$, df = 1, $P < 0.0001$).

Based on the results of the proportional hazards regression, the average predicted infection risk was 0.018 (range 0.006–0.235), with the lowest infection risk for small hosts growing far from the stream and the highest infection risk for large hosts growing near the stream. A histogram of these annual infection probabilities shows that they are log-normally distributed, with many (small) hosts having low infection risk and a few (large) hosts having high infection risk (Fig. 5).

DISCUSSION

The simplicity of pathogen dispersal and infection in the cedar–*Phytophthora* system, coupled with the success of using dendrochronological techniques on this particular tree, allowed the influence of host and environmental heterogeneity to be quantified in the context of this biological invasion. These two sources of heterogeneity both appear to be important determinants of the course and progress of the invasion and may strongly alter the effects of inoculum production and dispersal. The importance of multiple, but potentially interacting, sources of epidemiological heterogeneity has been stressed in theoretical contexts (see

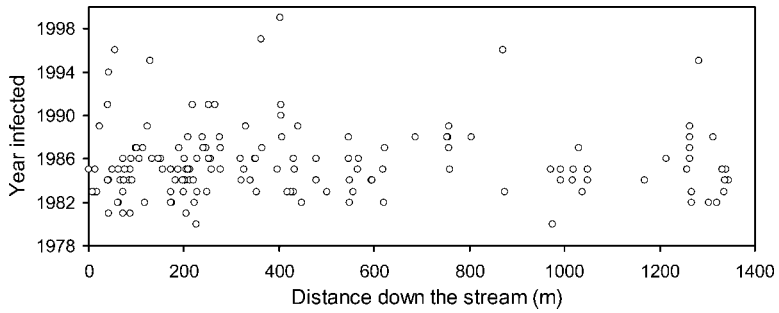

FIG. 2. The temporal distribution of *Phytophthora lateralis* infection of Port Orford cedar hosts was not related to distance down the length of the stream, which acts as a corridor for dispersing spores of this invasive pathogen.

TABLE 1. Likelihood ratio tests for significance of explanatory variables controlling the survival time (year of infection) of Port Orford cedar hosts during a 19-year invasion by *Phytophthora lateralis*.

Explanatory variable	Likelihood ratio χ^2	df	*P*
Host size	88.87	1	<0.0001
Proximity to stream	23.58	1	<0.0001
Host size × proximity to stream	5.37	1	0.0205
Full model	113.58	3	<0.0001

Notes: The likelihood ratio χ^2 value is calculated as follows: $-2(L_{\text{re-duced}} - L_{\text{full}})$, where L is the model log-likelihood. Models used in nested likelihood ratio tests were: host size only ($L = -899.335$), proximity to stream only ($L = -931.9805$), host size + proximity to stream ($L = -887.544$), and host size + proximity to stream + interaction (the full model, $L = -884.8595$). A constant-only model ($L = -941.65$) was used to test the full model.

Bolker et al. 1995). The reconstructed invasion dynamics evaluated in this study provide an empirical example of how two of these sources of heterogeneity influence epidemic progress in a disease invasion of conservation concern. The magnitude with which host size and position influenced invasion, in this case, strongly suggests that measures of heterogeneity are of practical concern for management, and they should be carefully considered for predicting and controlling invasive species.

Host heterogeneity had the largest effect on infection risk, with substantially higher infection rates for larger hosts compared to smaller ones. An effect of increasing infection risk with host size has been found in other plant–pathogen systems where spore deposition may limit infection rates (Roche et al. 1995, Morrison 1996). For large cedars, this is most likely a result of the larger target their roots present for dispersing spores, and the fact that larger hosts are more likely to have developed distant roots that are exposed to stream inoculum.

Environmental heterogeneity in this system, as we have characterized it, is a function of stream habitat, with hosts growing close to the stream channel (i.e., on the stream bank) having a higher chance of becoming infected than those growing on the hillslope farther away. Since disease transmission occurs primarily via stream-dispersed spores, this variation in susceptibility is either caused by increased inoculum loads for hosts growing close to the stream or by their enhanced vulnerability, since moist areas favor pathogen growth and persistence (Trione 1959). However, we interpret the significant interaction effect between host size and proximity to stream, which yields a diminishing effect of host size at the furthest distance classes (Fig. 4), to be most consistent with the notion that inoculum loads diminish with increasing distance to stream. This explanation provides a mechanism whereby the ''target'' effect of host size is diminished at further distances due to limiting inoculum supply.

The variation in susceptibility due to host and environmental factors strongly influenced the rate and pattern of this *P. lateralis* invasion. The first and most obvious influence of these heterogeneities was a sharp increase in new infections within the first few years of the invasion, when the most highly susceptible hosts quickly became infected. A second influence was to extend the duration of new infections after the peak of the epidemic, when only the less susceptible hosts remained to be infected. Ultimately, this epidemic declined to low levels of infection due to a lack of highly susceptible hosts. By the end of the study period, all except three of the surviving hosts within 3 m of the stream were <20 cm in diameter (Fig. 3).

Interestingly, these two sources of heterogeneity may also influence disease transmission. Because of their large root mass, large hosts are expected to be a more productive inoculum source than smaller hosts, and hosts growing close to stream inoculum (which are highly susceptible) may make a strong and disproportionate contribution to stream inoculum once infected. This potential correlation between host susceptibility and infectiousness may have been responsible for the rapid increase in the number of new infections during the early years of the epidemic (Fig. 1). This effect of heterogeneity on epidemic progress largely follows May and Anderson's (1988) prediction of the effect of heterogeneous sexual activity in HIV epidemics.

While we have not characterized large-scale effects of spatial heterogeneity (i.e., the spatial positions of healthy and diseased hosts down the length of the stream), it is likely that this source of heterogeneity influenced epidemic progress as well. However, given that spore dispersal must follow the flow of water, it is notable that there was not a discernable wave of new infections progressing downstream (Figs. 2 and 3). Indeed, the classic result from reaction-diffusion models, a constant velocity of new infections (van den Bosch et al. 1988), is clearly lacking in the invasion studied here. One explanation is simply that disease propagules are not limiting within this stream population due to abundant production of propagules by the first infected hosts and their long-distance transport down the stream. Such a scenario seems unlikely, however, since the classic epidemic pattern (Fig. 1) suggests that inoculum was limiting in the beginning of the epidemic (i.e., the increasing number of new infections in years 1980–1985 are likely a result of increasing stream inoculum levels).

A second explanation for the lack of a downstream wave of infection is that a handful of long-distance dispersal events initiated disease foci along the length of the stream as the epidemic was beginning, and that subsequent disease spread was the result of more common, short distance dispersal. A third explanation, which is not exclusive of the existence of multiple foci, is that the spatial and temporal pattern of stream-based spore dispersal was eroded by the heterogeneous sus-

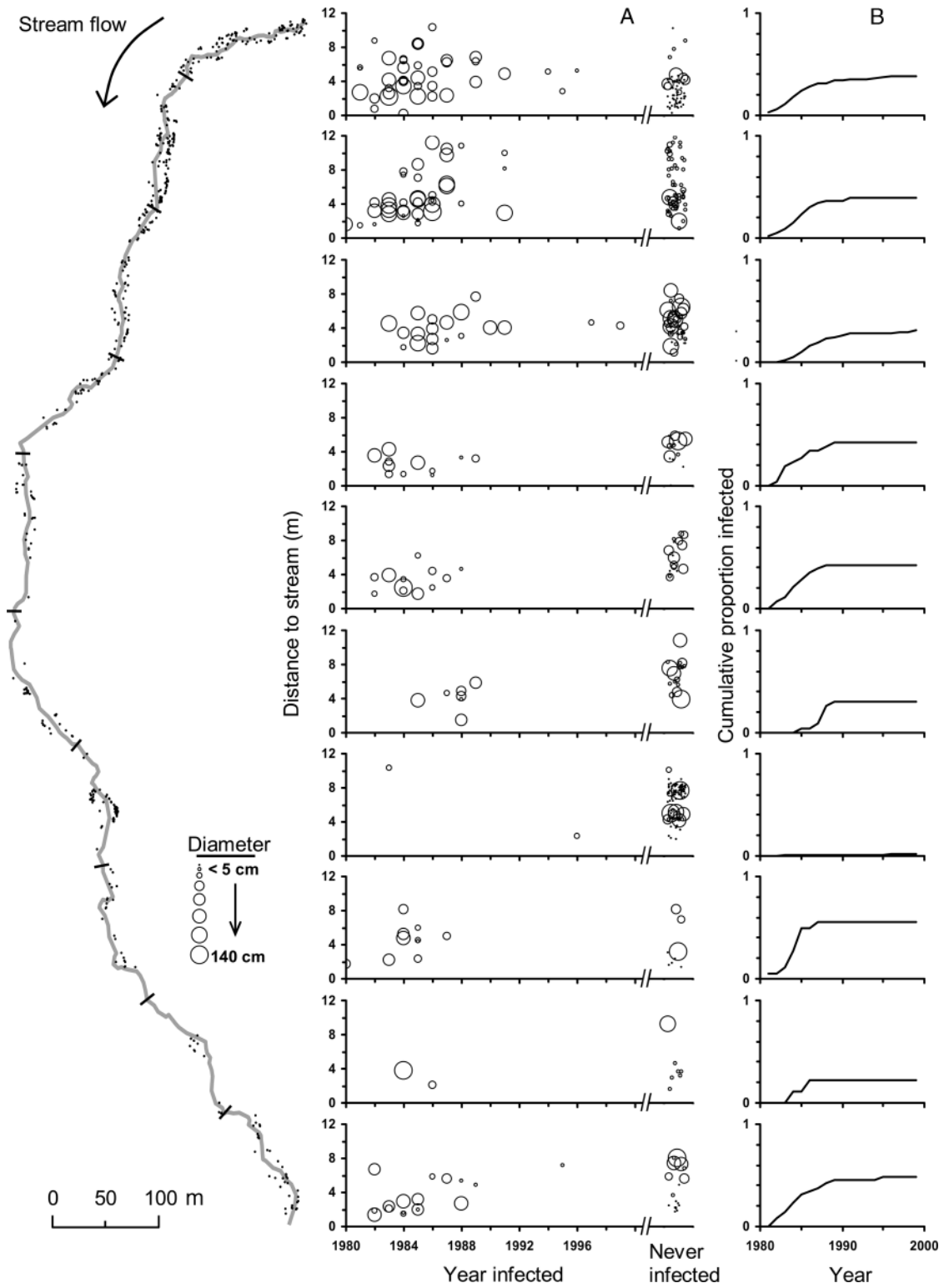

FIG. 3. Spatial and temporal progression of the spread of *Phytophthora lateralis* through a stream population of Port Orford cedar. Left-hand panels (A) show, for each 135-m stream segment, the relationship between the infection year of crossdated trees and their diameter (indicated by size of bubble) and proximity to the stream channel. Large cedars that were growing close to the stream channel were infected earlier in the epidemic. After the peak of the epidemic (1985), newly infected hosts were generally smaller and growing farther from the stream (as were hosts that were never infected). Right-hand panels (B) show the cumulative proportion of cedar hosts within each 135-m stream segment that were infected annually during the epidemic.

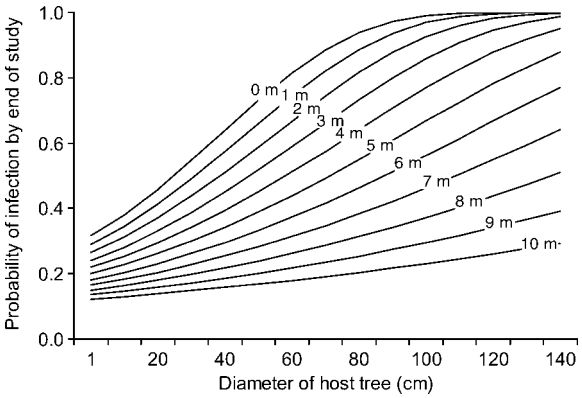

FIG. 4. Infection probabilities of Port Orford cedar hosts predicted by proportional hazards regression. The vertical axis denotes the probability of infection by the end of the study period (1999) for trees of varying host size (horizontal axis) and proximity to stream (individual lines).

ceptibility of hosts. We think an interaction between long-distance dispersal and host heterogeneity seems likely in this system, whereby downstream infection of the most susceptible hosts initiated multiple disease foci early in the invasion (Fig. 3). Theoretical models that produce a constant invasion speed almost always ignore sources of heterogeneity such as those quantified in this study.

This work suggests that heterogeneity in real-world invasions may be an underappreciated factor controlling observed invasion dynamics, and has implications for the theory and management of disease invasions. The strong influence of host heterogeneity in the Port Orford cedar system suggests that heterogeneity may not only erode, but drive, spatial patterns of disease spread. Thus, theoretical efforts should seek not only to account for sources of heterogeneity, but to incorporate them explicitly into predictive models of disease spread. Research that compares how disease spread is influenced by heterogeneity in susceptibility (due to host or environment) vs. the spatial structure of hosts and pathogen dispersal are particularly needed.

For management, recognizing and characterizing intrinsic variation in susceptibility can improve our understanding of the spatial scale of pathogen dispersal and thus, disease risk. For example, in an earlier study, we found that the spread of *P. lateralis* among host populations (i.e., spread at the larger, landscape scale) is strongly controlled by both the density and proximity of cedars growing just downstream of road crossings (Jules et al. 2002). This lead us to believe that inoculum is more widely dispersed along the road systems than previously thought, but that invasions generally only occur within highly susceptible stream populations. In many host-pathogen systems, knowledge of variation in susceptibility (at the level of host or sub-population) will lead to better assessments of the spatial components of infection risk and their management. Ferguson et al. (2001) provide an example of such a case for the recent foot and mouth disease (FMD) in Great Britain. Their study showed that farm type (number and type of livestock species) had a strong influence on the transmission dynamics of FMD, and that the efficacy of culling efforts could be improved by including information on variation in farm susceptibility.

In addition to understanding epidemic progress and spatial spread, ecologists are increasingly interested in how heterogeneity influences longer-term host-pathogen dynamics (Bolker et al. 1995, Bolker and Grenfell 1995, Real and McElhany 1996, Thrall and Burden 1999, Park et al. 2001). In the system studied here, heterogeneous infection rates may influence the long-term dynamics of the cedar-*Phytophthora* system in unpredictable ways. Young trees that are not rooted near the stream edge represent a dynamic reserve of hosts that are unlikely to become infected until they have grown to a larger diameter. Thus, the population of susceptible hosts may not be as thoroughly depleted by the initial epidemic as would be expected in the case of constant host susceptibility, and these young trees may continually provide the pathogen with newly susceptible hosts as they grow in size. In this way, heterogeneous susceptibility may enhance host-pathogen coexistence. In contrast, the relative invulnerability of young trees may increase the likelihood of local pathogen extinction since new recruits (which may take over 20 years to reach 1.5 m; M. J. Kauffman, *unpublished data*) will not provide a ready supply of highly susceptible trees in the short term. Ultimately, the rates of cedar growth and inoculum decay will determine whether these sources of heterogeneity act to enhance or diminish the persistence of this nonnative pathogen.

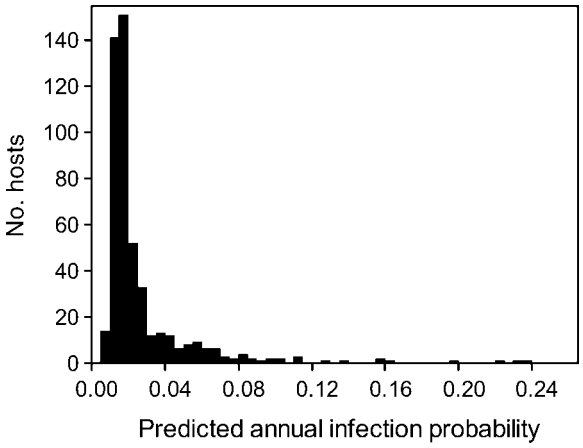

FIG. 5. Variability in annual susceptibility of Port Orford cedar hosts to infection by the nonnative pathogen, *Phytophthora lateralis*. Predictions are the results of proportional hazards regression, using empirical measures of host diameter and proximity to stream as predictors of host susceptibility. The long right-hand tail of the distribution represents the existence of a few highly susceptible hosts of large diameter and close proximity to the stream channel.

The results of this study have several implications for the management and control of *P. lateralis*. First, it is clear that once the pathogen colonizes a stream population, the entire stream will become infected, despite sections of low host density, and that this may happen in the time span of several years (Fig. 2). Second, the finding that streams with large, old-growth cedars growing in close proximity to water are at high risk of infection should be taken into account when deciding among alternative forest management practices, such as building new roads. Third, local infections of stream populations of Port Orford cedar may last for several decades, and managers should not assume that *P. lateralis* will eventually go extinct in previously infected stream reaches. We believe that pathogen extinction is most likely within Port Orford cedar populations where cedars are found solely on the banks of streams, as is often the case in the southern and eastern parts of its range. In the northern portion of its range, where Port Orford cedar is not restricted to stream banks, *P. lateralis* seems most likely to persist as an endemic pathogen, as uninfected hosts growing on slopes and ridges provide a continual supply of young hosts (seeds) to infected riparian areas.

Conclusion

By connecting reconstructed invasion dynamics with measured attributes of susceptible hosts, this study has shown that host and environmental heterogeneity can have a substantial influence on the spatial and temporal spread of a nonnative forest pathogen. Because dispersal of the pathogen is constrained to the stream corridor and unidirectional, we expected to see a detectable downstream pattern of annual infection. Surprisingly, we did not find a straightforward relationship between temporal development of disease and the downstream location of hosts along the length of the stream in this seemingly simply system. In addition to long-distance dispersal events, this study indicates that host and environmental heterogeneity may have eroded the spatial and temporal pattern of invasion predicted from theoretical models. Thus, the key result from this study is that the observed rates and patterns of real-world invasions may be strongly controlled by host- or site-specific factors. Because most natural landscapes exhibit various heterogeneities that influence invader colonization, infection, or establishment, this study highlights the need to better understand how such variability influences the spread of nonnative organisms.

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