Temporal Epidemiology of Sudden Oak Death in Oregon

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ABSTRACT

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An effort to eradicate *Phytophthora ramorum*, causal agent of sudden oak death, has been underway since its discovery in Oregon forests. Using an information-theoretical approach, we sought to model yearly variation in the size of newly infested areas and dispersal distance. Maximum dispersal distances were best modeled by spring and winter precipitation 2 years before detection, and infestation size the year prior. Infestation size was best modeled by infestation size and spring precipitation the year prior. In our interpretation, there is a 2-year delay between the introduction of inoculum and onset of mortality for a majority of sites. The year-long gap in between allows ample time for the production of inoculum contributing to the spread of *P. ramorum*. This is supported by epidemic development following changes in eradication protocols precipitated by an outbreak in 2011, attributable to a 2009 treatment delay and an uncharacteristically wet spring in 2010. Posteradication, we have observed an increase in the total area of new outbreaks and increased frequency in dispersal distances greater than 4 km. Although the eradication program has not eliminated *P. ramorum* from Oregon forests, it has likely moderated this epidemic, emphasizing the need for prompt treatment of future invasive forest pathogens.

Thirteen years have passed since the invasive pathogen *Phytophthora ramorum* (Werres, DeCock & Man in't Veld) was first detected in Oregon forests. Dissimilar to other areas with established populations at the time, sites were small enough to warrant the attempted eradication of *P. ramorum*. Although eradication has not been achieved, this program has produced a detailed, spatially and temporally explicit dataset representing the distribution of mortality attributable to *P. ramorum*. Our goal in this work is to identify, through a modeling approach, factors contributing to yearly variation in epidemic severity and to discuss the impact the eradication effort has had on the dynamics of disease spread since the initial introduction of *P. ramorum*.

Capable of infecting over 100 species in 35 genera (APHIS 2011), *P. ramorum* causes a nonlethal leaf blight or dieback in most hosts (Rizzo and Garbelotto 2003). On others, particularly tanoak (*Notholithocarpus densiflorus*, syn. *Lithocarpus densiflorus*) and the red oaks (*Quercus* spp. section Lobatae), *P. ramorum* causes bleeding bole cankers and tree death (McPherson et al. 2010; Rizzo and Garbelotto 2003). This disease, termed sudden oak death (SOD), was first recognized in the mid-1990s as extensive tanoak mortality in northern California (Rizzo et al. 2002, 2005). It has since spread to forests in 13 coastal counties in northern California, with two isolated outbreaks in Humboldt County, CA and Curry County, OR (APHIS 2011; Rizzo et al. 2005).

Oregon's infestation was first identified in 2001 in the Douglas-fir and tanoak forests outside the coastal town of Brookings (Fig. 1) (Goheen et al. 2002). At the time of first detection, the infestation was confined to a small area and eradication was deemed possible. Under the eradication program, aerial surveys have been performed two or more times each year to identify recently killed tanoak. Follow-up ground surveys confirm the coordinates of potentially infected trees with a global positioning system and collect samples of inner bark, leaves, and stems with characteristic symptoms of *Phytophthora* infection for confirmation of the pathogen (Hansen et al. 2008, Kanaskie et al. 2008).

After positive identification, SOD sites have been extensively surveyed. Eradication protocol required the removal of all main hosts, usually accomplished with cutting and burning, within a minimum buffer of 100 m from the outermost sample infected by *P. ramorum*. Greater treatment areas have been implemented when possible. Although most of the vegetation at the periphery of the eradication area is not infected, this distance has proven effective at eliminating those few trees that had been exposed to local spread but were asymptomatic at the time of surveys (Hansen et al. 2008; Kanaskie et al. 2008).

Despite concerted efforts to stop the expansion of SOD in Oregon, newly infected sites have been identified each year since 2001. Prior to 2006, new sites were concentrated within the Joe Hall and North Chetco watersheds (Fig. 1). In 2006, SOD was detected at two new areas to the east and west of the quarantine boundary at the time (Hansen et al. 2008); the western site, named Borax, has continued to spread (Fig. 1, insert A).

Two aspects of this epidemic have been particularly inhibitory to the program's eradication goal: the annual emergence of new infections at the periphery of known sites and the occurrence of long and unpredictable jumps between sites (Hansen et al. 2008). The eradication program has operated under the presumption that, by reducing site size, we could effectively limit both the size and number of new infected patches and the likelihood of long-distance dispersal. It remains unclear whether the eradication program has met these goals. The delay between initial infection and detection, as well as how to explain variation in epidemic severity between years, is also unknown, potentially leading to erroneous conclusions on the effectiveness of the eradication program.

Modeling is a common approach utilized to develop hypotheses about the behavior of plant pathogens (Madden et al. 2007). In the case of invasive pathogens in heterogeneous environments, however, models are generally difficult to interpret (Holdenrieder et al. 2004). The SOD epidemic in Oregon is one of few nonagricultural examples in which heterogeneity is manageable. Tanoak is abundant across

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most elevations within the quarantine area and surrounding forests of Curry County (Hansen et al. 2008; Tappeiner et al. 1990). Moreover, among the bole hosts, tanoak presents the highest rates of mortality but the least amount of inheritable resistance (Hayden et al. 2011; Maloney et al. 2005; McPherson et al. 2005). Although there is some variation in susceptibility among tanoak populations (Hayden et al. 2011), differences in susceptibility to *P. ramorum* infection can be negligible relative to the influence of local environmental conditions (Anacker et al. 2008). Finally, because Oregon's infestation has only one lineage (NA1) with one clonal population dominated by a single genotype, we expect less variation in pathogenicity relative to other forest populations (Prospero et al. 2007).

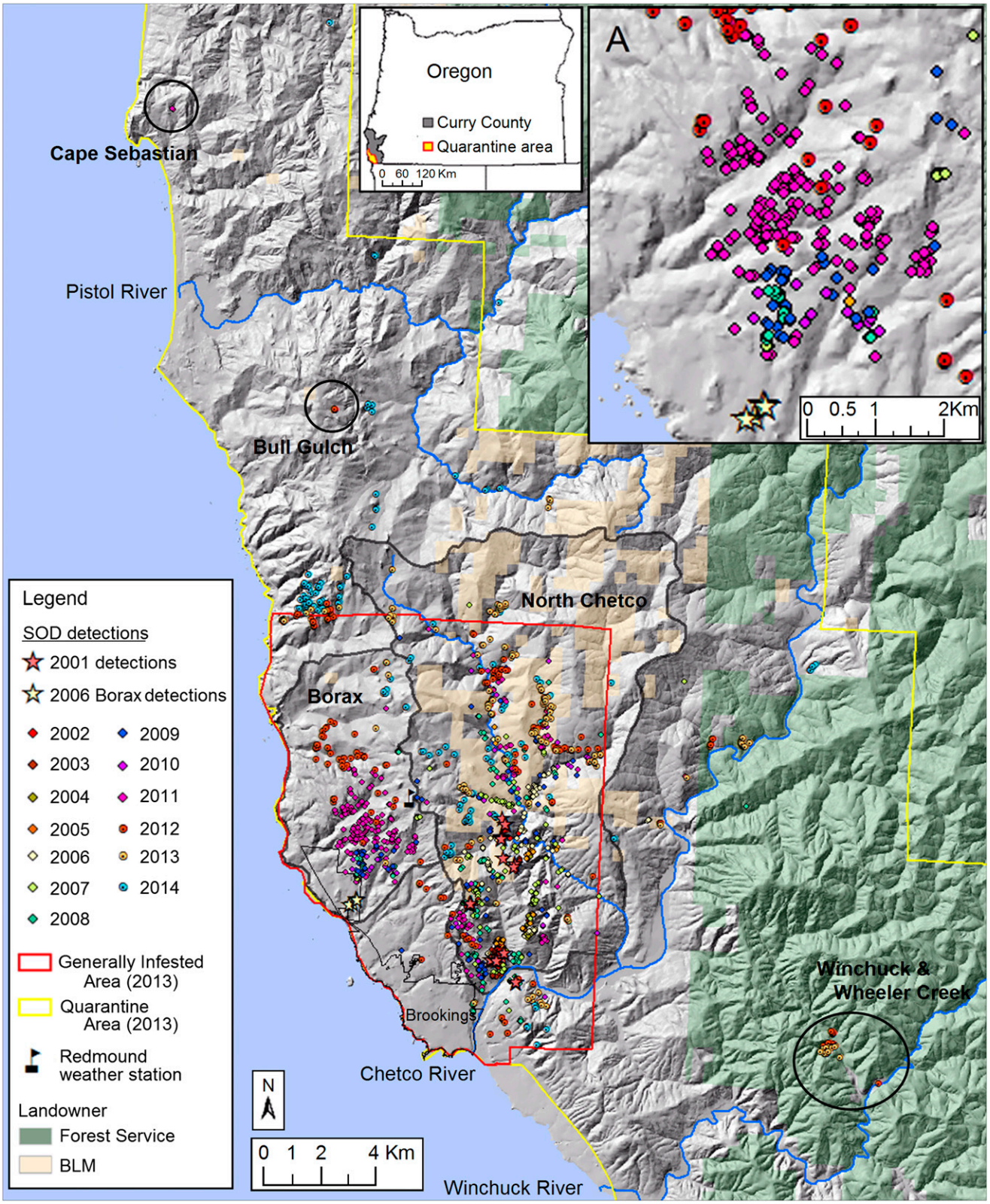

Fig. 1. Map showing the distribution of *Phytophthora ramorum* in southwestern Oregon forests since its initial detection in the Joe Hall and North Chetco watersheds in 2001. **A,** Insert depicts the epidemic developing in the Borax study area since its first detection in 2006.

Without significant changes in host composition, pathogenicity, or treatment protocol, the yearly variation in the size of newly infested areas and distance between sites can reasonably be attributed to changes in weather between years, particularly those conditions affecting the ability of *P. ramorum* to sporulate. Sporulation by *P. ramorum* is one of the more thoroughly investigated aspects of SOD. The recovery of inoculum from rain splash and foliar lesions indicates that consistently greater spore loads are produced with increased precipitation, and in forest types with moister microclimates (Davidson et al. 2008, 2011). The effects of temperature are less well understood, although recovery of inoculum from individual lesions is greatest during late spring rains (Davidson et al. 2008). This effect was heightened during El Niño years, when precipitation extended longer into the warm season (Davidson et al. 2008). Also in support of an influence of temperature, disease severity on California bay laurel (*Umbellularia californica*) is positively associated with maximum daily temperatures from December through May (Condeso and Meentemeyer 2007).

Both the maximum distance moved and infestation size of a given year may vary significantly in response to conditions augmenting sporulation and spread. Per standard dispersal and disease curves, we expect to observe disease at further distances and greater amounts of disease at a given distance following environmentally conducive years, resulting in larger sources of primary inoculum (Supplementary Fig. S1) (Madden et al. 2007). Whereas the size of an infestation in a given year will be augmented by the amount of primary inoculum establishing new sites and the amount of secondary inoculum contributing to local spread in subsequent years, the maximum dispersal distance will result exclusively from the amount of inoculum present during the year of production. Because of the delay between initial infection and detection, however, we expect a delay between the year of inoculum production and the detection of mortality at a distal location.

The year 2011 marked a dramatic increase in SOD-infested acreage and the range of movement. This is potentially attributable to a 2009 delay in eradication of the infestation within the Borax portion of the quarantine area. Although other sites detected in 2009 were treated that year, detections within Borax were not eradicated until 2010. As a consequence, 2010 marks the last year that full eradication was attempted, heralding the adoption of a slow-the-spread regime and designation of the generally infested area (GIA) where eradication is no longer being attempted on most lands.

Although the eradication program has not eliminated *P. ramorum* from Oregon forests, we want to investigate whether reducing primary inoculum sources has affected epidemic development and identify factors contributing to variation in epidemic extent. To do so, we modeled variation in epidemic severity between 2001 and 2010, prior to changes in management protocols in 2011. Cumulative statistics describing all SOD detections in Oregon up to 2014 support additional discussion on past successes and difficulties of the eradication program, and highlight the future challenges of managing *P. ramorum* in natural forested ecosystems.

MATERIALS AND METHODS

Study area. Infested sites in southwest Oregon fall within the mixed-evergreen forest type dominated by Douglas-fir (*Pseudotsuga menziesii*) and tanoak. The climate is characterized by wet and mild winters with dry and warm summers. Tanoak is commonly found in the understory, as a codominant species mixed with emergent conifers, or in dense, pure tanoak stands (Tappeiner et al. 1990). Although epidemiologically important in California, bay laurel is more sparsely distributed within the quarantine area in Oregon and has not been found infected when not in association with tanoak mortality (Hansen et al. 2008; Peterson et al. 2014).

We modeled epidemic development during eradication (2001 to 2010) in two areas with independent and extensive disease development: the original Joe Hall Creek and North Fork Chetco River drainages (hereafter called the "North Chetco study area"), and a second area originating from the smaller Borax site first identified in 2006 (hereafter called the "Borax study area") (Fig. 1).

Movement of *Phytophthora ramorum* over the ridges defining the eastern and western edges of the North Chetco and Joe Hall Creek watersheds has been rare over this pathogen's history in Oregon (Hansen et al. 2008). As such, we assumed that all infestations located west of the North Chetco study area were attributable to the expansion of the Borax infestation, as is supported by microsatellite analysis of the two populations (Kamvar et al. in press). Differentiation between the two epidemics was only possible until 2011, prior to the expansion of the epidemic to the northern edge of the quarantined area (Fig. 1).

Characterization of epidemic development, 2001 to 2014. Change in the SOD epidemic over time was characterized by quantifying the total size of the infested area and range of movement between new SOD detections in annual intervals.

Infestation size. The size of the annual infested area was assessed by creating 30-m buffers around the coordinates of all *P. ramorum*-positive isolations identified within a single year. Isolations were made from both tanoak cankers and infection of understory foliage as part of the eradication efforts to delineate the outer boundary of each infestation. The distance of 30 m was selected to approximate the area in which we find the majority of infections as a result of secondary, within-site spread. We dissolved all buffers by year of detection and study area, then calculated the total size of each polygon in hectares.

Distances between sites and maximum distance moved. To characterize the distances between new SOD detections of a given year to the nearest known inoculum source, we first reduced all adjacent positive isolates to site coordinates to minimize the bias of oversampled locations. Sites were defined as the centroid of all newly identified SOD-positive isolations located within 60 m of one another. We calculated the dispersal distance moved between years by performing spatial joins of sites in each year to sites detected in any year prior (e.g., between all sites detected in 2006 and all sites detected between 2001 and 2005).

Epidemic variables were calculated for all Oregon SOD detections between 2001 and 2014. We additionally calculated epidemic variables separately for all sites located within the North Chetco and Borax study areas up to 2011. All geographic datasets were projected in the OR NAD83 Lambert coordinate system and analyzed in ArcGIS (ver. 10.2; ESRI).

Weather data. Weather data were obtained from the Redmound remote access weather station, maintained by the Bureau of Land Management (BLM) and located within the current quarantine area (latitude: 42°07′24″ N, longitude: 124°18′02″ W, elevation: 534 m; http://www.raws.dri.edu/index.html) (Fig. 1). Average daily maximum temperature (°C), average daily precipitation (mm), and average daily humidity (%) from 1 October 1998 to 31 December 2013 were compiled into autumn (October through December), winter (January through March), spring (April through June), and summer (July through September) averages.

Modeling of epidemic development during eradication, 2001 to 2010. Multiple linear regression was used to identify weather conditions that can model infestation size and maximum distance moved within the North Chetco study area between 2001 and 2010 (Table 1). Sites located within the Borax area were additionally included after 2006 (Table 1). Explanatory variables included the seasonal weather conditions of the year of detection and 1 and 2 years before detection (Table 2). A preliminary analysis indicated that conditions 3 years before detection did not significantly increase the explanatory power of our models (data not shown). We have little reason to suspect that crown mortality is delayed much longer than 3 years post introduction given the relatively small size of most sites when detected, indicating that the pathogen has had limited time to spread locally. Humidity and all weather variables during the

summer were also eliminated in preliminary analysis (data not shown). Infestation sizes at 1 and 2 years before detection were also considered as explanatory variables (Table 2).

We used an information-theoretic approach to evaluate combinations of main-effects models predicting maximum distance moved or infestation size in either the North Chetco or Borax study areas (Burnham et al. 2011; Grueber et al. 2011). First, a global model was built with all weather and epidemic variables with correlation coefficients stronger than ±0.2 and, due to their reported role in California epidemiology, average spring precipitation and maximum winter temperature of 1 and 2 years before. Climate variables were screened for collinearity using Pearson correlations prior to model selection (Grueber et al. 2011). Any pair of variables with a Pearson's $r > 0.8$ were considered collinear. For each collinear pair, the variable with the greater correlative value in a Spearman rank-order analysis was retained in the global model.

Potential submodels were generated with the R-package *glmulti* (Calgano 2013). Due to the small number of data points for each response, we restricted models to all combinations of up to three explanatory variables (Grueber et al. 2011). To identify the best set of models from all weather and time-lag combinations, we calculated the Akaike information criterion with small sample correction (AIC_c) for each model, where lower AIC_c values indicate a greater strength of evidence of one model relative to others in the model set. All models were then ranked by the difference between their AIC_c and the AIC_c of the best model (ΔAIC_c), and by model

TABLE 1. Characterization of the Oregon sudden oak death (SOD) epidemic during eradication (2001 to 2010) and after changes in treatment protocols to a slow-the-spread regime (2011 to 2014)[a]

	North Chetco study area			Borax study area			All Oregon SOD sites		
Year	Infest. (ha)	Max. (m)	New	Infest. (ha)	Max. (m)	New	Infest. (ha)	Max. (m)	New
2001	5.06	n/a	10	n/a	n/a	n/a	5.47	n/a	11
2002	10.15	574	17	n/a	n/a	n/a	10.43	574	18
2003	10.88	2,713	18	n/a	n/a	n/a	10.88	2,713	18
2004	5.70	565	10	n/a	n/a	n/a	5.70	565	10
2005	9.11	914	17	n/a	n/a	n/a	9.11	914	17
2006	22.38	2,192	46	3.11	n/a	3	24.39	3,975	50
2007	25.81	4,261	61	1.54	3,494	2	29.94	4,901	67
2008	18.19	2,449	50	3.68	2,352	7	26.27	2,449	63
2009	15.58	2,634	44	6.98	859	16	23.40	2,634	62
2010	7.61	1,259	21	3.06	249	9	10.95	1,259	31
2011	22.05	1,042	62	45.42	3,863	106	68.56	19,677	169
2012	n/a	n/a	n/a	n/a	n/a	n/a	46.10	10,912	122
2013	n/a	n/a	n/a	n/a	n/a	n/a	42.23	4,560	105
2014	n/a	n/a	n/a	n/a	n/a	n/a	37.64	6,688	84
Total	…	…	…	…	…	…	351.08	…	827

[a] Data are presented for all Oregon sites detected within a single year (2001 to 2014), or are segregated for all sites falling within the North Chetco or Borax study areas until 2011. Infest. = infestation size, Max. = maximum distance, New = number of new sites, and n/a = not applicable.

TABLE 2. Potential weather and epidemic terms considered as explanatory variables for regression modeling of maximum distance moved and infestation size by year[a]

		r^{2a}	
Variable code[b]	Description	Maximum distance moved	Infestation size
Epidemic			
Infest(t-1)	Infestation area year before	**0.137 (+)**	**0.509 (+)**
Infest(t-2)	Infestation 2 years before	0.040 (+)	0.112 (+)
Weather			
Year of			
AuMaxT(t-0)	Autumn maximum temperature	**0.066 (+)**	0.011 (+)
AuPrecip(t-0)	Autumn precipitation	0.112 (+)	**0.058 (+)**
WMaxT(t-0)	Winter maximum temperature	**0.000**	**0.002 (+)**
WPrecip(t-0)	Winter precipitation	0.035 (–)	0.000
SpMaxT(t-0)	Spring maximum temperature	**0.061 (–)**	**0.041 (+)**
SpPrecip(t-0)	Spring precipitation	**0.122 (–)**	**0.044 (–)**
1 year before			
AuMaxT(t-1)	Autumn maximum temperature	0.034 (–)	0.022 (–)
AuPrecip(t-1)	Autumn precipitation	0.028 (+)	0.001 (-)
WmaxT(t-1)	Winter maximum temperature	**0.306 (–)**	**0.034 (–)**
WPrecip(t-1)	Winter precipitation	**0.202 (+)**	0.002 (–)
SpMaxT(t-1)	Spring maximum temperature	0.004 (–)	0.028 (–)
SpPrecip(t-1)	Spring precipitation	**0.060 (+)**	**0.003 (+)**
2 years before			
AuMaxT(t-2)	Autumn maximum temperature	0.061 (+)	0.038 (+)
AuPrecip(t-2)	Autumn precipitation	0.032 (+)	0.005 (+)
WMaxT(t-2)	Winter maximum temperature	**0.328 (+)**	**0.087 (+)**
WPrecip(t-2)	Winter precipitation	**0.084 (–)**	0.008 (–)
SpMaxT(t-2)	Spring maximum temperature	0.107 (–)	0.003 (+)
SpPrecip(t-2)	Spring precipitation	**0.438 (+)**	**0.065 (+)**

[a] Direction of the correlation (positive or negative) is indicated. Those variables included in the initial global models of maximum distance moved or infestation size are in bold.

[b] Epidemic = epidemic variables and Weather = weather conditions for year of and 1 and 2 years before detection. Example: for a tree detected in spring or summer of 2009, 1 year before detection is defined as average daily conditions from October to December (autumn) 2007, January to March (winter) 2008, or April to June (spring) 2008.

weights (the probability that of all considered models any particular model has the lowest AIC_c) (Calgano and de Mazancourt 2010).

All models with a $\Delta AIC_c < 4$ units (Burnham et al. 2011) were averaged with the goal of calculating weighted parameter estimates using the R-package *MuMIn* (Bartoń 2012). Although we lacked the power to detect significant differences between model variables, interpretation of explanatory variables in the final averaged model was based on average parameter estimates and cumulative parameter weights (an estimation of the importance of a given term, defined as the summed weights for all models in which each term appears) (Calgano and de Mazancourt 2010). The averaged model was used to calculate infestation size and maximum distance moved expected in 2011 in each of the study areas had the eradication proceeded uninterrupted in 2009. Significant deviation between observed and expected values was assessed by calculating 95% confidence intervals for model predictions. Because the delay preferentially affected the Borax area, we hypothesized that the 2011 Borax epidemic would exceed model predictions whereas the North Chetco area would not.

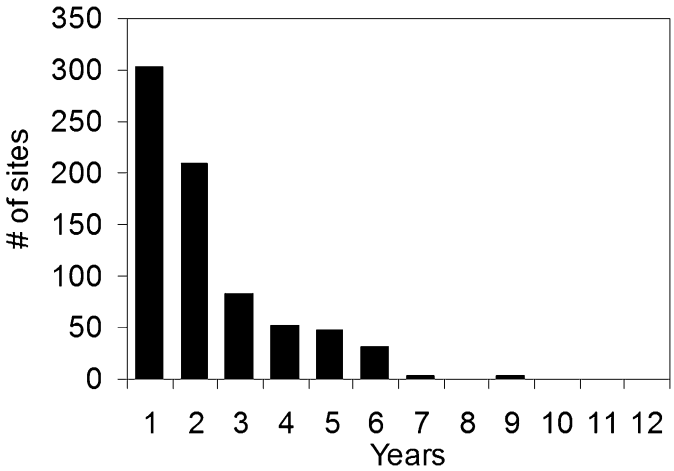

Fig. 2. Frequency distribution of the length of time (in years) between the detection of new sudden oak death sites and the detection of the closest site of any previous year. Data are presented for all sites identified between 2001 and 2014.

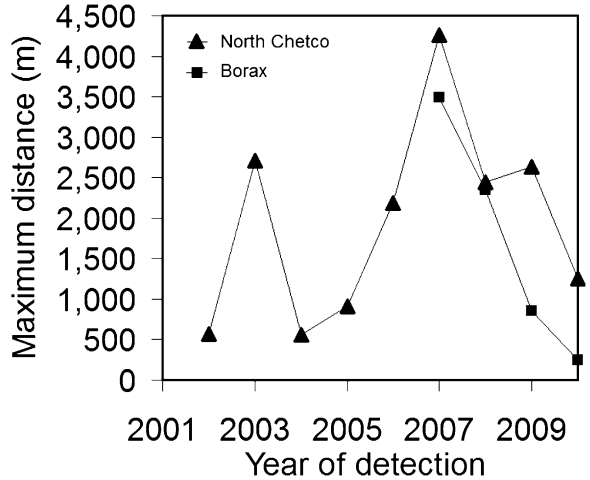

Fig. 4. Maximum distance between new sites in each year to the closest site of any previous year in the North Chetco and Borax study areas. Because the Borax area was first detected in 2006, the first between-site dispersal was recorded in 2007.

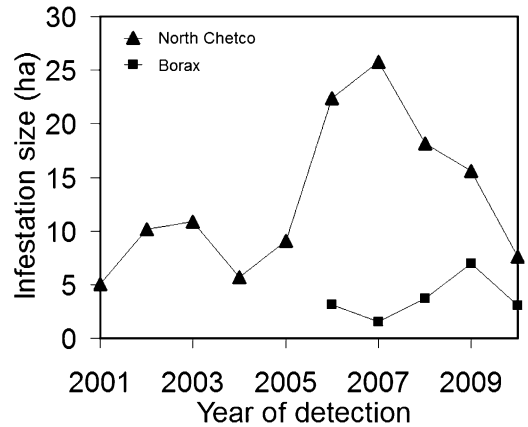

Fig. 5. Total size of newly detected infested areas in hectares (ha) by year in the North Chetco and Borax study areas.

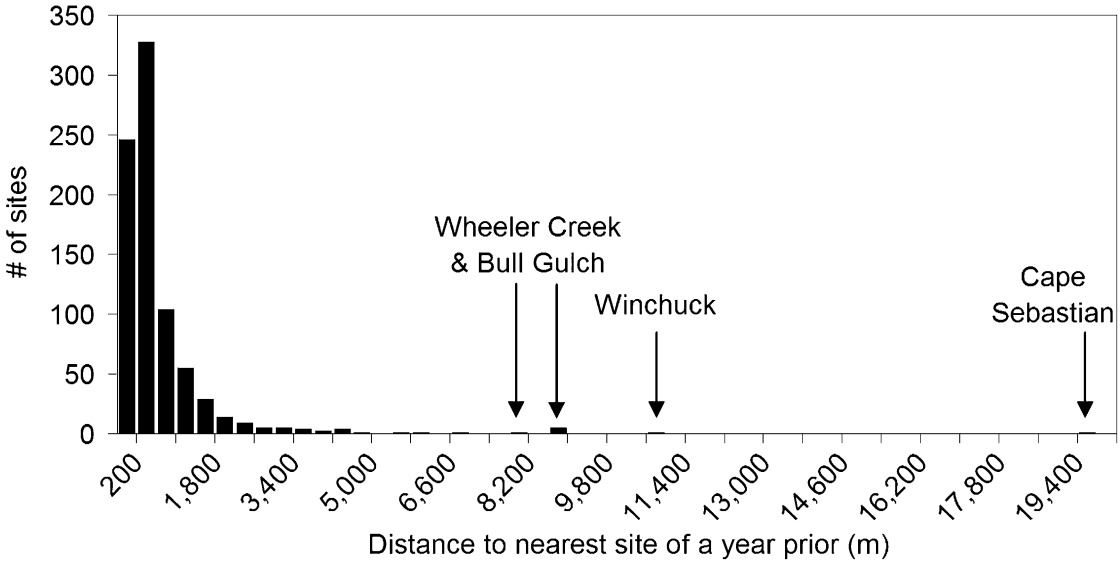

Fig. 3. Frequency distribution representing the distance from any site in a given to year to the closest site in any year prior for all sites detected between 2001 and 2014. The number of new sudden oak death detections located within 200 m of sites of a previous year is underrepresented, because the eradication program has largely removed these new infections from detection. Dispersal events greater than 8 km from the nearest known source (Wheeler Creek, Bull Gulch, Winchuck, and Cape Sebastian) are indicated.

RESULTS

Epidemic development, 2001 to 2014. For all SOD sites detected between 2001 and 2014, the majority of new sites were closest to SOD sites identified the previous year (Fig. 2). There was a steep decline in distances between sites of a given year to any year prior (Fig. 3). The mean and median distance moved for all sites detected between 2001 and 2014 was 687 and 317 m, respectively. Rarely have new sites been located greater than 3 km from sites of any year prior; however, nine sites have been detected >8 km from the nearest known inoculum source.

Dispersal distances >4 km were observed in 2007 (establishing sites 4.3 and 4.9 km from the nearest known inoculum source), 2011 (establishing the Cape Sebastian site, also known as Hunter Creek, 19.7 km moved), 2012 (seven sites at Bull Gulch and Wheeler Creek ranging from 8.1 to 8.6 km from the nearest known inoculum source, and the Winchuck site, 10.9 km moved), 2013 (three sites ranging between 4.3 and 4.6 km moved), and 2014 (three sites ranging between 5.4 and 6.7 km from the nearest known inoculum source) (Figs. 1 and 3; Table 1).

Modeling of epidemic development during eradication, 2001 to 2010. Both maximum distances moved and infestation size of a given year have been highly variable between 2001 and 2010 (Table 1; Figs. 4 and 5). The longest jumps for both the North Chetco and Borax areas were observed in 2007 (Table 1; Fig. 4). That year had the largest total new infested acreage in the North Chetco area but the smallest total new infested acreage in the Borax area (Table 1; Fig. 5).

TABLE 3. All models with a $\Delta AIC_c < 4$ built to describe maximum distance moved or infestation size in the North Chetco and Borax study areas between 2001 and 2010

Model	df	logLik	ΔAIC_c	Weight	R^2
Maximum distance moved					
MaxDistance ≈1 + SpPrecip(t-2)	3	–106.85	0[a]	0.22	0.438
MaxDistance ≈1 + Infest(t-1) + SpPrecip(t-2)	4	–105.03	0.69	0.16	0.575
MaxDistance ≈1 + WPrecip(t-2) + SpPrecip(t-2)	4	–105.31	1.26	0.12	0.556
MaxDistance ≈1 + WMaxT(t-2)	3	–108.00	2.30	0.07	0.329
MaxDistance ≈1 + WMaxT(t-1)	3	–108.21	2.72	0.06	0.307
MaxDistance ≈1 + WMaxT(t-2)+ SpPrecip(t-2)	4	–106.14	2.93	0.05	0.495
MaxDistance ≈1 + WMaxT(t-0)+ WMaxT(t-1)	4	–106.15	2.94	0.05	0.495
MaxDistance ≈1 + Infest(t-1) + WMaxT(t-2)	4	–106.20	3.03	0.05	0.491
MaxDistance ≈1 + WPrecip(t-1)+ SpPrecip(t-2)	4	–106.32	3.29	0.04	0.481
MaxDistance ≈1 + AuMaxT(t-0)+ SpPrecip(t-2)	4	–106.47	3.58	0.04	0.469
MaxDistance ≈1 + SpMaxT(t-0)+ SpPrecip(t-2)	4	–106.52	3.69	0.04	0.465
MaxDistance ≈1 + WMaxT(t-1) + SpPrecip(t-1)	4	–106.54	3.72	0.03	0.463
MaxDistance ≈1 + SpPrecip(t-1) + SpPrecip(t-2)	4	–106.55	3.73	0.03	0.463
MaxDistance ≈1 + WMaxT(t-0) + WMaxT(t-2)	4	–106.60	3.84	0.03	0.458
Infestation size					
InfestArea ≈1 + Infest(t-1)	3	–39.61	0[b]	0.33	0.509
InfestArea ≈1 + Infest(t-1) + SpPrecip(t-1)	3	–37.58	0.28	0.29	0.641
InfestArea ≈1 + Infest(t-1) + SpMaxT(t-0)	3	–38.35	1.81	0.13	0.596
InfestArea ≈1 + Infest(t-1) + AuPrecip(t-0)	3	–38.81	2.73	0.08	0.566
InfestArea ≈1 + Infest(t-1) + SpPrecip(t-2)	3	–39.21	3.53	0.06	0.539
InfestArea ≈1 + Infest(t-1) + SpPrecip(t-0)	3	–39.25	3.61	0.05	0.536
InfestArea ≈1 + Infest(t-1) + WMaxT(t-1)	3	–39.39	3.90	0.05	0.525

[a] $AIC_c = 222.36$.
[b] $AIC_c = 87.89$.

TABLE 4. Model terms (β-estimate ± standard error [SE]) and relative importance of all variables included in the averaged model describing maximum distance moved (Distance) and infestation size (Infestation) in the North Chetco and Borax study areas between 2001 and 2010

Term	β-Estimate (± SE)	*z* Value	*P* value	Cumulative weight	Number of models with term
Maximum distance moved[a]					
(Intercept)	511.85 (± 6,003.05)	0.083	0.934	…	…
SpPrecip(t-2)	278.61 (± 216.36)	1.238	0.216	0.70	8
Infest(t-1)	12.43 (± 28.70)	0.416	0.677	0.21	2
WMaxT(t-2)	122.68 (± 285.15)	0.419	0.675	0.20	4
WMaxT(t-1)	–97.84 (± 263.36)	0.365	0.715	0.14	3
WPrecip(t-2)	–14.43 (± 46.87)	0.295	0.768	0.12	1
WMaxT(t-0)	–40.69 (± 156.54)	0.251	0.802	0.08	2
SpPrecip(t-1)	14.16 (± 72.27)	0.186	0.853	0.07	2
WPrecip(t-1)	3.88 (± 27.35)	0.132	0.895	0.04	1
AuMaxT(t-0)	11.58 (± 97.23)	0.109	0.913	0.04	1
SpMaxT(t-0)	5.93 (± 53.86)	0.101	0.920	0.04	1
Infestation size[b]					
(Intercept)	–2.57 (± 13.86)	0.175	0.861	…	…
Infest(t-1)	0.69 (± 0.20)	3.089	0.002	1.00	7
SpPrecip(t-1)	0.46 (± 0.84)	0.522	0.602	0.29	1
SpMaxT(t-0)	0.24 (± 0.76)	0.303	0.762	0.13	1
AuPrecip(t-0)	0.05 (± 0.21)	0.211	0.833	0.08	1
SpPrecip(t-2)	0.04 (± 0.24)	0.138	0.890	0.06	1
SpPrecip(t-0)	–0.02 (± 0.15)	0.131	0.896	0.05	1
WMaxT(t-1)	–0.04 (± 0.32)	0.100	0.921	0.05	1

[a] $R^2 = 0.573$.
[b] $R^2 = 0.609$.

Maximum distance moved was best explained by weather conditions from 2 years before detection (Table 3). The models with $\Delta AIC_c < 2$ incorporated spring and winter precipitation 2 years before detection, as well as the infestation size the year before detection (Table 3). Support was greatest for spring precipitation 2 years before detection, which had the greatest cumulative weight of all variables in our averaged model. This variable was included in 8 of the 14 models with $\Delta AIC_c < 4$, and had the strongest effect on distance relative to all other variables (Tables 3 and 4). For the remaining variables included in the models with $\Delta AIC_c < 4$, the three variables with the next highest cumulative weights (WMaxT(t-1), WPrecip(t-2), and WMaxT(t-0)) all had a negative impact on maximum distance moved when included in our averaged model (Table 4).

Infestation size of a given year was best modeled by the infestation size of the year prior, followed by spring precipitation the year before detection, then spring maximum temperature in the year of detection (Table 4). Support was greatest for infestation size the year prior, which had the highest cumulative weight and was included in all seven models with a $\Delta AIC_c < 4$ (Tables 3 and 4).

The regression estimates of our averaged models had large standard errors, likely due to the small sample size in our response and large number of explanatory variables. Regardless, both infestation size and maximum distance moved exceeded the bounds of the 95% confidence intervals calculated for the Borax study area in 2011. The upper 95% confidence interval expected in 2011 for maximum distance moved and infestation size in the Borax area were 3,519 m and 21.08 ha, respectively (Supplementary Tables S1 and S2). In 2011, we observed a dispersal distance of 3,863 m and an infestation size of 45.42 ha in the Borax area (Table 1).

Neither maximum distance moved nor infestation size observed in 2011 in the North Chetco study area were significantly different than expected per our averaged model. For the North Chetcon area, we observed a maximum distance of 1,042 m and infestation size of 22.05 ha, less than the calculated upper 95% confidence intervals expected at 4,224 m and 23.98 ha (Table 1).

DISCUSSION

Biological interpretation of model variables. In the theory of how epidemics develop over time and space, the velocity of an epidemic should increase over time in direct relation to the size of the infestation contributing inoculum or total inoculum load (Aylor 2003; Cowger et al. 2005; Sackett and Mundt 2005). This is assuming that conditions favoring sporulation and the distribution of hosts are constant over the range of observations. Infestation size was not, by itself, the best indicator of the amount of inoculum produced in the year contributing to the spread of *P. ramorum* in Oregon. Rather, differences in weather conditions between years exerted additional influence on dispersal distance and infestation size of subsequent years.

Of the weather variables tested, spring precipitation remains the best overall predictor of epidemic severity both for maximum distance moved and infestation size of a given year. Additional support exists for the retention of winter or spring temperatures, and the elimination of autumn weather conditions from consideration as the dominant times or conditions conducive for epidemic development. Additional variables in models with ΔAIC_c values between 2 and 4, models which have some explanatory power but include variables whose effects on model function are less well supported, may have only been included in final models due to unavoidable collinearity between weather conditions within and between years (Holdenrieder et al. 2004). Alternatively, we may have excluded important variables in our effort to avoid the inclusion of highly correlated terms (infestation size of 2 years prior to detection, for example). Additional difficulties in our ability to statistically detect differences in model parameters arise due to the small sample size and large number of variables included in our averaged model. Nevertheless, the biological interpretation of the best supported models with $\Delta AIC_c < 2$ is consistent with all prior observations and modeled epidemiology regarding the behavior of *P. ramorum* in western U.S. forests (Condeso and Meentemeyer 2007; Davidson et al. 2008, 2011).

Variables best explaining maximum distance moved include weather conditions 2 years before the detection of new distal sites and the size of the infested area the year before detection (Table 4). The length of time between the death of the trees providing inoculum and those receiving inoculum need not be 2 years so long as the time between infection and detection is similar, albeit offset, between the source and sink trees. In our proposed model, whereas the delay between infection and mortality is 2 years within a site, the length of

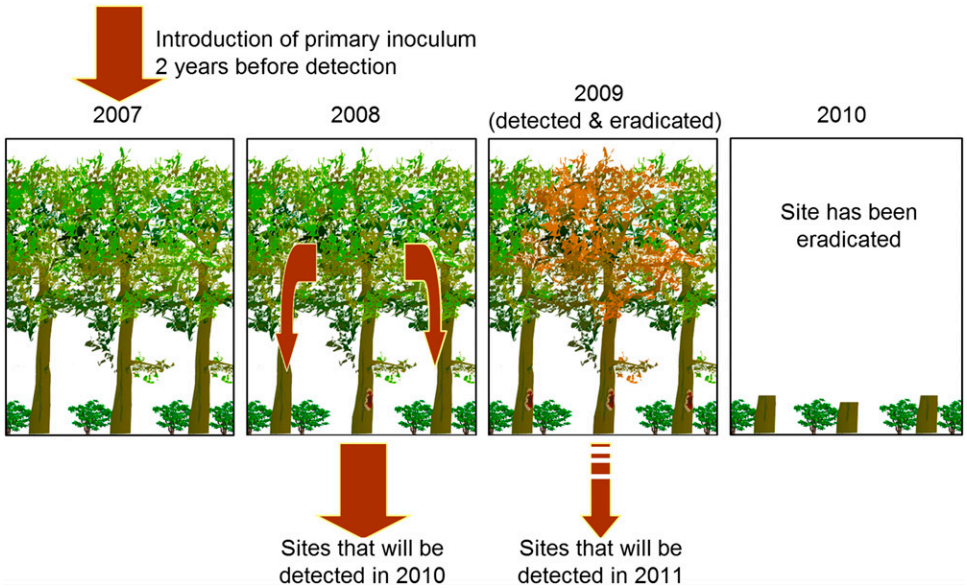

Fig. 6. Proposed temporal epidemiology of *Phytophthora ramorum* in Oregon forests during eradication, showing the delay between the introduction of primary inoculum and detection of overstory mortality and prompt eradication. Per our model, trees that would have been detected and eradicated in 2009 (which were infected by inoculum produced in 2008) are the most likely source of inoculum contributing to tree mortality detected in 2010. A greater than 2-year delay in mortality preventing detection and eradication in some sites in 2009 (or the retention of all sites in Borax of that year) would have contributed inoculum to mortality observed in 2011. Illustration adapted from Parke and Lucas (2008).

time between the deaths of the source and sink trees will only be offset by a single year (Fig. 6). For example, inoculum produced from a site in 2008 will infect trees for which the majority will die in 2010; those trees that were the source of this inoculum will most likely die in 2009, an observed delay of only 1 year (Fig. 6).

During the delay between initial infection and detection, weather conditions contributing to greater dispersal distances are also contributing to local spread. Our models predicts that spring precipitation the year before detection and spring temperatures the year of detection were contributors to the size of an infestation, although the best determinate was the infestation size of the year prior (Table 4). This may imply that the size of the infested area in a given year is determined more by the number of dispersal events contributing to new sites than by the rate of local spread within a site. However, this may be an artifact of how we calculated infestation size. Due to inconsistent sampling intensity within sites over the course of the eradication program, using the number of SOD-positive trees would bias our observations toward more heavily sampled years or locations. Instead, we chose to estimate site size by creating a boundary around the coordinates of positive samples, which are generally taken to estimate the outer boundaries of symptomatic plants. The use of buffers to estimate total infestation size is corroborated by spatially explicit sampling around infested sites (Peterson et al. 2014), and accounts for asymptomatic infection on the periphery of sites as a result of local spread. However, it does oversimplify our estimation of the actual number of infected trees, presenting a source of error in our models.

Our temporal model incorporates features of expected epidemiology, whereby spores contributing to the establishment of most new sites are most likely produced at sources closer in space and in time. Closer sites are the most likely sources of primary inoculum, because dispersal gradients result in greater numbers of new infections closer to the source than further away (Fitt et al. 1987). However, our analysis fails to capture a large portion of the variation inherent in this spatially and temporally complex system. In particular, closest sites are not necessarily the sources of primary inoculum and mortality would not strictly occur 2 years post-introduction. These alternative situations can account for those sites in which the closest site of any previous year was identified multiple years before, although they do occur with a decreasing frequency (Fig. 2). Although many sites may have slightly longer delays (2- or 3-year gaps between the detection of source and sink infections), we suspect that those pairings with longer intervals are not true sources. However, lacking direct evidence of a maximum time that an individual site can remain undetected, we chose to perform the analysis calculating dispersal distance from a single site to the nearest site of any year prior.

Without greater detail (tracing individual dispersal pathways, for example), we can only infer that our models are representative of what we would expect to see if new sites behave similarly to the majority of other sites in our analysis. That is, whereas spring precipitation or infestation size of the year prior would show the strongest effects in the model due to their dominance of affect upon epidemic development, we do not have the basis to exclude other factors. We emphasize the exploratory nature of this analysis, and limitations imposed by our dataset and methods. However, given the relative uniformity of our host and pathogen populations, it does provide a basis with which we can form hypotheses regarding the effect that the eradication program has had upon the development of SOD in Oregon.

Expansion of SOD in 2011. The first opportunity to observe how the Oregon epidemic might respond to a lack of eradication resulted from the delay in treatment of the Borax area in 2009, where sites were surveyed but not treated until 2010. The question remained as to whether this delay, which was not a factor in the North Chetco area, could account for the dramatic increase in infestation size and dispersal distance observed in 2011 (Table 1).

Retaining a sporulating population in 2009 is consistent with a larger infestation observed in 2011. Under normal eradication protocols, sites undetected in 2009 would have made the largest contribution to the establishment of those sites detected in 2011. In Borax, however, sites which would have been detected and eradicated in 2009 were allowed to continue contributing inoculum, establishing additional sites which would have been detected in 2011 (Fig. 6). Compounding the delay, the infestation size would have been large in 2011 due to an uncharacteristically wet spring in 2010 (Supplementary Fig. S2). This single condition would have resulted in larger sites in 2011 (1 year later).

Interestingly, 2011 was also the year of our longest dispersal event to date, 19.7 km to the Cape Sebastian site. Cape Sebastian was not included in our models due to our inability to conclusively discern if it originated from either the North Chetco or Borax areas, or if it originated from Oregon forests at all. Although genetically related to the lineage comprising the Oregon SOD infestation and originally thought to have dispersed from the Borax area, more recent analysis suggests that Cape Sebastian might be a new introduction into Oregon forests (Kamvar et al. in press). Regardless, the furthest site which could reasonably be attributed to Borax in 2011 was located 3.86 km away from the nearest site of any year prior, twice the distance expected from our models. The infestation size was also significantly larger than expected in Borax, encompassing more than five times the area expected by our models.

The events of 2009 were not the first time that trees had been left without treatment, either because of minor delays in funding or because some sites were wrongly identified as negative for *P. ramorum*. Yet we have not seen such a dramatic increase in infestation area immediately around these locations as was observed in 2011. Prior to 2009, minor delays did not extend a whole year, as happened in the Borax area in 2009, suggesting that they were treated before contributing significantly to spread. The misidentification of sites has been uncommon over the 10 years that the eradication program has operated, and the lack of their contribution is predominantly due to their small size. Larger inoculum sources have a greater potential to contribute to epidemic development relative to smaller sources (Madden et al. 2007). We suspect the difference in sizes between one missed site and the extent of the infestation left standing in the entire Borax area in 2009 can account for the disparity in epidemic development observed 2 years later. This also explains why the majority of SOD intensification is in close proximity to heavily infested areas, rather than emanating from the small, isolated distal sites on the extreme northern margins of the quarantine area.

We cannot conclusively demonstrate that the delay in eradication is the sole contributor to the dramatic increase in infestation 2011. Borax had overall smaller treatment areas than North Chetco, more public access allowing for greater opportunity for movement by people, and, being closer to the ocean, a slightly more favorable microclimate. All could contribute to greater rates of epidemic development in the Borax area in comparison with the North Chetco area. Regardless, it is reasonable to conclude that reducing the size of the sporulating population has had an impact on epidemic development. This is especially relevant to changes in treatment protocols post-2011.

Changes in epidemic development with the change from eradication to slow-the-spread. In addition to affecting infestation size in 2011, the wet spring of 2010 would have predicted larger maximum dispersal distance moved in 2012 (2 years later). Indeed, 2012 does mark the year of our second-longest dispersal event, to the Winchuck site located 10.5 km from the nearest known inoculum source (Fig. 1). Additional long-distance dispersal events that year also include dispersal in excess of 8 km to Wheeler Creek to the south and Bull Gulch to the north (Fig. 1).

The years 2011 to 2013 have seen a reduction in spring precipitation closer to average norms (Supplementary Fig. S2). Although we have not observed dispersal distances greater than 8 km in recent

years, the incidence of dispersal distances greater than 4 km has increased (Table 1). We have observed greater intensification of SOD within the GIA than what is represented in our maps, as early detection priorities have shifted to the north and east. As a result of this change in detection protocols, the amount of newly infested areas within the GIA is likely underrepresented in later years. Despite this, the size and number of newly documented sites within a given year has also noticeably increased post-2011 (Table 1).

Although the eradication effort has not succeeded in its attempt to eliminate *P. ramorum* from Oregon forests, it has forestalled the quarantine of all of Curry County and has likely reduced overall rates of spread on the landscape through the reduction of primary inoculum sources, benefiting commercial interests and allowing for additional time to generate adaptive management protocols. This is despite challenges which were unanticipated during the early years of epidemic development. A multiyear delay between the establishment of new sites and their detection and eradication leaves ample time for sites to contribute to landscape-level spread. Whereas the North Chetco is largely owned by the BLM and private lumber companies which supported aggressive and prompt removal of hosts, Borax is dominated by smaller rural-residential parcels which proved to be more difficult to treat. Finally, the delay in 2009 (potentially producing more sites than would have been expected) unfortunately coincided with the wet spring of 2010 (producing larger sites due to increased rates of local spread). The result was an outbreak in 2011 which was overwhelmingly impossible to treat with the available resources.

The abundance of tanoak within Curry County is not currently a limiting factor affecting the establishment of SOD, even within the GIA, which still has large patches of uninfected tanoak. Currently, no treatments are required for SOD falling within the bounds of the GIA, although the BLM will continue to treat SOD on all their land regardless of location. Outside the GIA, local eradication treatments continue, although the size and intensity of the treatments must be scaled back considerably due to lack of funds. As infections are left standing without eradication in the most heavily infested areas, we expect to see an increase in the amount of infection in subsequent years, especially on the periphery of known established areas. We also expect to see *P. ramorum* move longer distances with greater frequency as the size of infested areas is allowed to grow. Both responses, however, will continue to be significantly modified by weather conditions affecting the spread and establishment of *P. ramorum*.

It bears mentioning that *P. ramorum* was only described as a species in 2001, the same year that SOD was detected in Oregon (Werres et al. 2001). Despite the limited knowledge of the epidemiology of this invasive pathogen at the time, the coordinated response to SOD is undoubtedly one of the most important successes of the eradication program. The management of *P. ramorum* in Oregon could not have proceeded without the full support of multiple federal and state government agencies, research institutions, and private land owners in the Brookings area. All recognized how vitally important it was to respond quickly to the threat of SOD, for any delays would have significantly reduced our capacity to manage this disease in later years. New pathogens having the potential to severely affect native forests will continue to emerge as a consequence of the global movement of plants. Our response, or lack thereof, will ultimately determine the rates at which these pathogens spread on the landscape and the scale at which their impacts are realized.

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