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Efficacy of local eradication treatments against the sudden oak death epidemic in Oregon tanoak forests

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Abstract

Phytophthora ramorum, cause of sudden oak death, has been distributed widely across the United States in horticultural situations, but is not established in forests outside of California and Oregon. Here, it has triggered widespread concern and, especially in Oregon, an intensive disease management programme. Now, we provide the first systematic evaluation of the efficacy of that effort.

This paper evaluates four measures of the efficacy of Sudden Oak Death (SOD) local eradication treatments: inoculum availability; inoculum from tree species other than tanoak; disease spread from treated areas; and cumulative infested area with and without treatment. We conclude that local treatments demonstrably reduce local inoculum levels. Eradication of SOD from infested sites is difficult but not impossible. The disease usually does not persist after cutting infected trees but spread on the landscape continues because the pathogen may be present on undetected new infections for a year or two before whole tree symptoms are visible. This limits early detection and coupled with delays in completing eradication treatments, prolongs the chances for long-distance aerial dispersal.

KEYWORDS
canker < disease type, disease control, other < host genus, sudden oak death

1 | INTRODUCTION

Phytophthora ramorum Werres, De Cock & Man In't Veld, causal agent of Sudden Oak Death (SOD), is an invasive pathogen in North America in horticultural nurseries and in coastal forests in northern California and south-western Oregon. In this region, mixed evergreen forests (Franklin & Dyrness, 1973) often have upper canopies dominated by Douglas-fir (*Pseudotsuga menziesii*) or redwood (*Sequoia sempervirens*), with several species of sclerophyllous oaks (*Quercus*) or tanoak *Notholithocarpus densiflorus* (Hook Arn.) Rehd.) in mid-canopy (Goheen et al., 2002; Rizzo, Garbelotto, Davidson, & Slaughter, 2002; Rizzo, Garbelotto, & Hansen, 2005). *Phytophthora ramorum* is subject to state (Oregon Revised Statutes, 2001), Federal (Code of Federal Regulations of the United States of America, 2006) and international quarantines, and there is continuing concern about its potential spread to the large areas of known hosts beyond the current pathogen distribution as well as to forests around the world where related plants grow. Although infected nursery stock has been distributed widely across the United States (Goss, Larsen, Chastagner, Givens, & Grünwald, 2009; Mascheretti, Croucher, Vettraino, Prospero, & Garbelotto, 2008), to date it has not become established as a pathogen in forests outside of California and Oregon. In these states, however, it has triggered widespread concern and, in Oregon, an intensive, expensive, multifaceted and inter-agency disease management programme. Here, we provide the first systematic evaluation of the efficacy of components of the Oregon disease control effort.

Tanoak (*N. densiflorus)* is an ecologically important component of the mixed evergreen (*Pseudotsuga*-sclerophyll) forest zone (Franklin

& Dyrness, 1973) in the coast ranges of south-western Oregon. The climate is mild and Mediterranean, with wet winters and warm, dry summers. Average annual precipitation between 1995 and 2015 in the epidemic area (Red Mound Remote Automated Weather Station, RAWS) (http://www.wrcc.dri.edu/cgi-bin/rawMAIN.pl?orOREM), almost all occurring as rain, is 3,208 mm, ranging from 10 mm in July to 620 mm in December. Average daily temperatures range from 7.5°C in December to 18.5°C in July. Tanoak is a sprouting species, regenerating from the stump after disturbance by fire or forest harvest. Depending on site disturbance history, it may grow in extensive pure stands or as scattered individuals beneath a Douglas-fir overstory. The Oregon forest affected by SOD has been greatly modified by recent human disturbance, ranging from rural residential development to intensive commercial forest management (Bowcutt, 2015). As a result, the landscape is a mosaic of vegetation types. About 20% is young Douglas-fir plantations with scattered tanoaks of sprout origin. Another roughly 20% is mature tanoak stands that succeeded old-growth Douglas-fir after the initial harvest in the early 1900s. The remainder is largely mixed stands, often dominated by Douglas-fir, but containing variable components of tanoak.

Phytophthora ramorum infects leaves, shoots and stems of many woody plants. It causes epidemic disease in forests in Great Britain (Webber, Mullet, & Brasier, 2010) and western North America (Rizzo et al., 2005). Four phylogenetic lineages have been described (Ivors et al., 2006; Van Poucke et al., 2012). EU1 and EU2 are A1 mating type. They were first recognized in horticultural situations in Europe, and EU1 was transported to North America in nursery stock (Goss et al., 2009). The NA1 and NA2 lineages are A2 mating type and are only known from North America; until recently, only the NA1 lineage was known from forests (Grünwald et al., 2016). In Oregon, *P. ramorum* is especially destructive on tanoak, and in California, tanoak and coast live oak (*Quercus agrifolia*) are severely affected. Many other forest plants are susceptible (including Douglas-fir), but for the most part suffer little damage (Hansen, Parke, & Sutton, 2005) and with the exception of Oregon myrtle (*Umbellularia californica*, also called California laurel or California bay), contribute little if anything to the airborne inoculum load. Long-distance dispersal is by human transport of infected nursery stock (Goss et al., 2009; Mascheretti et al., 2008) and, for distances up to several kilometres, by aerial movement of propagules, presumably sporangia, through turbulent dispersal (Hansen et al., 2008). Local disease intensification, however, is largely through splash dispersal (Peterson, Hansen, & Kanaskie, 2015). About 80% of new infections are found within 300 m of previously infected trees (Hansen et al., 2008). The pathogen produces deciduous sporangia on infected leaf and twig tissue primarily of tanoak in Oregon and is regularly recovered from rainwater (Davidson, Patterson, & Rizzo, 2008; Davidson, Wickland, Patterson, Falk, & Rizzo, 2005; Hansen et al., 2008) and from soil (Fichtner, Lynch, & Rizzo, 2007; Goheen, Kanaskie, Navarro, & Hansen, 2017; Peterson Hansen & Hulbert, 2014) beneath infected trees. *Umbellularia californica* is less common in south-western Oregon than in California and is less frequently infected. Douglas-fir and grand fir (*Abies grandis*) suffer dieback when growing beneath infected tanoak and *P. ramorum* does sporulate on inoculated twigs (J. LeBoldus, personal communication), but symptoms are not seen on mature trees in Oregon forests.

Examination of the upper crowns of tanoak trees with bole cankers and of adjacent trees without bole cankers reveals twig infections on most cankered trees and on a few adjacent trees. Infection of understory host plants is found only in the immediate vicinity of cankered trees, suggesting top-down spread, presumably in stem flow, rain drip and splash from the infected overstory (Hansen et al., 2008; Peterson et al., 2014). Soil infestation presumably results from sporangia or infested leaves fallen from the canopy.

In 2001, when *P. ramorum* was first discovered in Oregon (Goheen et al., 2002), SOD was limited to a few small clusters of dead and dying tanoak trees close to Brookings (42.07 N, −124.27 W) in Curry County (Goheen et al., 2017) (Figure 1). Regulatory agencies in Oregon responded quickly (Oregon Revised Statutes, 2001), establishing a 23 km^2 quarantine area. As SOD continued to spread, the quarantine area was expanded; in 2017, it covered 1,333 km^2. An early detection survey and local eradication programme was initiated in 2001, relying on aerial survey to locate recently killed trees with red crowns and ground checking with verification by direct isolation or PCR (Winton & Hansen, 2001) to confirm new infestations, and cutting and burning diseased tanoak trees and associated host plants (Goheen et al., 2002, 2017; Kanaskie et al., 2008). The eradication treatment has evolved through time as new information became available. At present, the optimum treatment calls for early detection, primarily through aerial and stream survey (Sutton, Hansen, Reeser, & Kanaskie, 2009), with follow-up ground survey and confirmation of the pathogen. The extent of local infection is determined by intensive ground survey to detect trees not yet exhibiting crown symptoms visible from the air and augmented by spore trapping in rain traps placed beneath tanoak trees around the perimeters of critical areas. After delimitation of infested sites, treatment begins. Infected trees plus a surrounding 100 m or 200 m buffer of visibly healthy but susceptible trees are killed, usually using herbicide to reduce resprouting ("hack and squirt" injection of Imazapyr prior to cutting). On some sites, treatment areas are considerably larger, depending on importance of the site in terms of future disease spread and landowners' management objectives. A few landowners attempt to protect their tanoak with Agrifos (phosphonate) bark sprays (Garbelotto & Schmidt, 2009). Infected and buffer trees and other host plants are cut to quickly get the inoculum sources close to the ground and reduce opportunities for turbulent dispersal. In most cases, slash is burned (Figure 2). All tanoaks are cut; Oregon myrtle trees are usually cut; and Douglas-fir and other canopy trees are often left, depending on long-term management objectives for the site. The goal is to detect infections as early as possible and halt subsequent sporulation and dispersal of the pathogen as quickly as possible. Despite the sustained effort, the pathogen continues to spread in Curry County, and in 2017, a new infestation of the EU1 lineage started to spread. In 2012, the goal of the programme was restated from "eradication" to "slow the spread" (Goheen et al., 2017).

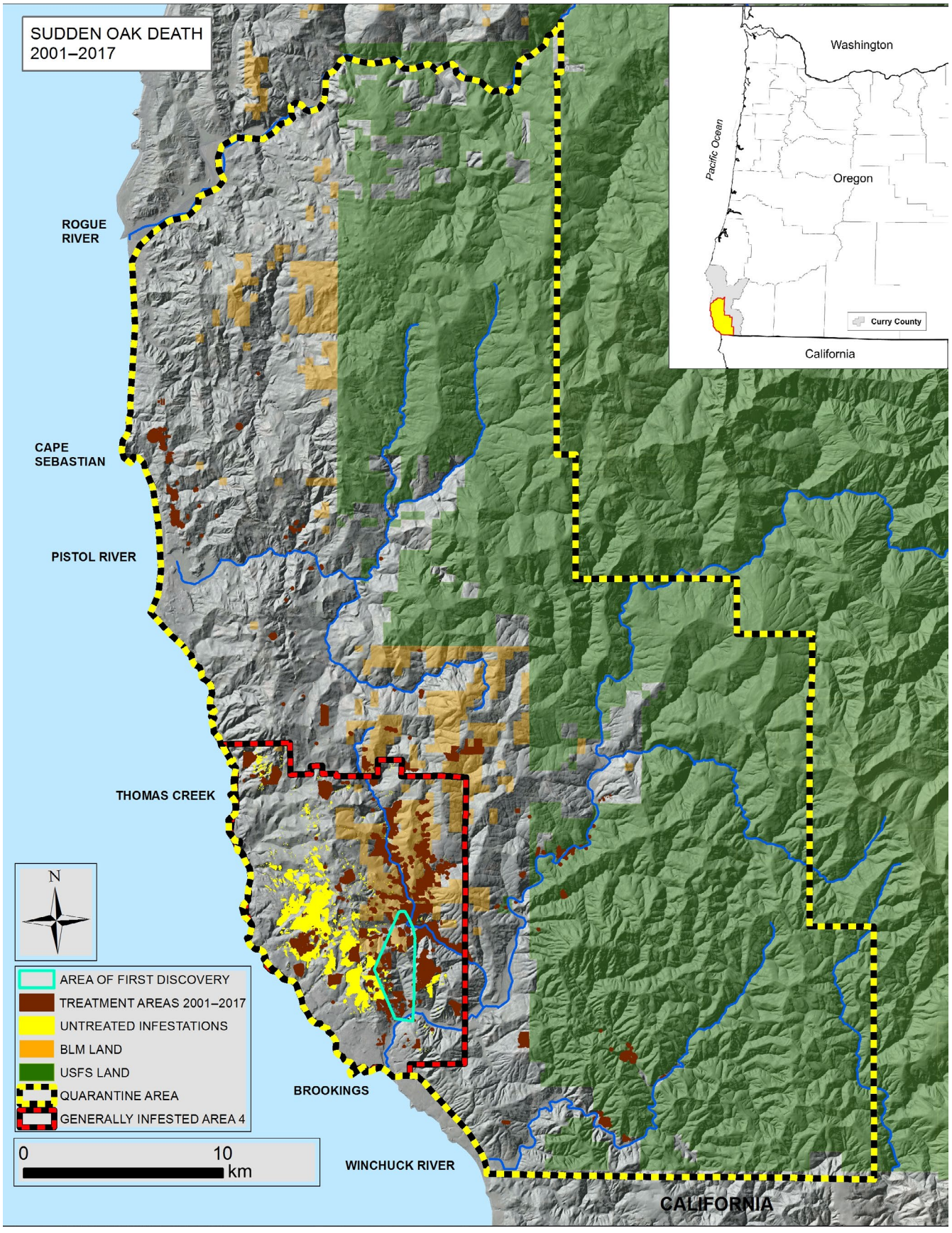

FIGURE 1 SOD quarantine area in Curry County, SW Oregon, showing the quarantine area, the generally infested area and local eradication treatment areas as of 2017. The area of the original 2001 infestations is highlighted

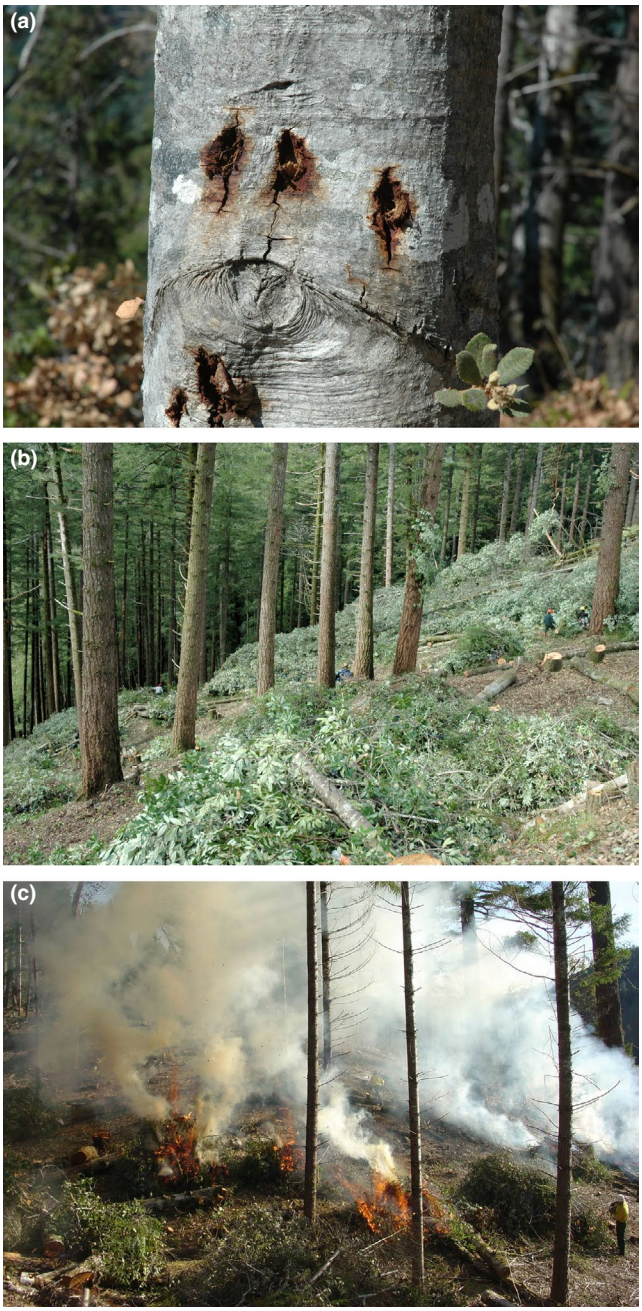

FIGURE 2 Oregon SOD Eradication Treatment. (a) Hack & squirt tanoak with herbicide prior to cutting to kill hosts and prevent stump sprouting; (b) Cut tanoak, rhododendron, huckleberry, sometimes Oregon myrtle; (c) Burn (piles)

Absent treatment, the local intensification of SOD in Oregon has been rapid. Using high-resolution colour aerial photography, Goheen et al. (2017) documented the increasing number of tanoaks killed by SOD between 2012 and 2016 on 10, 1 ha plots in untreated areas of Curry County. Tanoak mortality increased in 4 years from <5% to an average of 87% (range 71%–99%) on the plots.

Treatment details vary from site to site due to fluctuations in the availability of funding for treatments and differing land ownership and management objectives. In 2010, a surge in new detections coupled with delays in funding the eradication effort led to an unsurmountable backlog of untreated areas in the most severely infested part of the SOD distribution. Consequently, an 8,816 ha "Generally Infested Area (GIA)" was delimited in 2012 and local eradication efforts were halted on private land within it. On these lands, aerial detection was limited to broad scale sketch mapping of recently killed tanoaks, and location of individual trees and ground checking for cause of death were curtailed. Survey and treatments continued outside of the GIA and on the Bureau of Land Management (BLM) lands within the GIA. The GIA has been expanded four times since, as necessary to balance available funding with the costs of control. As of 2017, the GIA had increased to 15,107 ha. Expenditures are concentrated on treatments near the advancing front of SOD. Since 2001, about 2,826 ha have been treated.

The net effect of Oregon eradication efforts has been an apparent reduction in the rate of disease increase (Kanaskie et al., 2010; Hansen et al., 2008) compared with areas in California where no consistent treatment programme has been possible. Epidemiological modelling (Václavik, Kanaskie, Hansen, Ohmann, & Meentemeyer, 2010) suggests that prompt treatment of new infestations will slow the epidemic. Despite the demonstrated threat and widespread concern, however, there has been very little research on the practical efficacy of strategies to eradicate the pathogen from forest areas or to limit its spread (Goheen et al., 2017).

The eradication mandate and associated quarantine requirements prevent conventional experimental designs with comparable untreated controls to evaluate the efficacy of treatments. At the same time, the intensive early detection and disease monitoring efforts create unprecedented opportunities for measuring attributes of pathogen dynamics. In preliminary work, we described recovery of the pathogen in infested areas from rainwater at all times of the year (Hansen et al., 2008).

This paper evaluates the effectiveness of several components of the Oregon SOD eradication programme by combining treatment and monitoring data from previous years with current landscape-scale spatial and temporal data on disease advance. We evaluate four measures of the efficacy of SOD local eradication treatments (NA1 epidemic only):

1. Inoculum availability in SOD areas before, during and after local eradication treatments, as measured in rain traps;
2. Inoculum production from tree species other than tanoak often left untreated after local eradication;
3. Disease spread from treated areas;
4. Cumulative infested area with and without treatment.

2 | METHODS

2.1 | Inoculum availability

Simple rain traps were used to capture propagules splashed from the overhead canopy. Traps were placed beneath tanoak trees and other vegetation in five situations: (a) areas 100 m or more distant

FIGURE 3 Rain trap setup. (a) Plastic bucket lined with a replaceable polyethylene bag, baited with rhododendron and tanoak leaves; (b) buckets covered with screen. Water was added to keep bait leaves fresh between rain events; (c) water level was recorded and bait leaves with plastic bags were exchanged at 2-week intervals; (d) tanoak forest showing placement of rain trap. Photographs by Mike McWilliams

from known infection; (b) in known infested stands before eradication treatments began; (c) in infested stands while eradication treatments were in progress; (d) in infested stands after treatments were completed; and (e) beneath the first residual trees around the perimeter of treated areas when operations were complete. Treatment details varied among sites, but generally included hack and squirt herbicide treatment, cutting diseased and buffer tanoaks, and burning the slash. Five or more traps usually were placed at each site, depending on size of the tanoak stand or eradication area. Stand infection status, canopy species, disease progress, and treatment type and progress were recorded for each trap placement. Traps were moved as the treatments progressed. Trapping was continued with various objectives and varying intensity from November 2006 through February 2016. Rainfall was recorded at the Red Mound Remote Automated Weather Station (RAWS) located within the SOD epidemic area, and by measuring water depth in the buckets themselves. Traps were placed at 79 sites for varying durations; more than 14,000 trap/intervals (one trap for one 2-week interval) were recorded in total.

Rain traps were two-gallon (3.8 L, 24 cm diameter) white HDPE plastic buckets lined with 1 mil polyethylene bags and baited with two rhododendron (*Rhododendron macrophyllum* D. Don ex G. Don) and two tanoak leaves. Bait leaves were placed in the bucket along with ca. 375 ml de-ionized water to keep leaves fresh through periods without rain. Each bucket was covered with fibreglass window screen to keep out large debris (Figure 3). At retrieval of baits (after two weeks of exposure), water depth was recorded as a measure of local precipitation and any visible soil splash into the bucket was noted. The plastic liner was replaced, and new bait leaves were added. Bait leaves were transported to the laboratory, where they were washed in tap water and blotted dry. Necrotic areas of leaves were plated in semi-selective CARP + medium (Corn meal agar base amended with 30 ppm Benlate 75WP, 10 ppm Na-natamycin, 200 ppm Na-Ampicillin, 10 ppm Rifamycin SV and 25 ppm hymexazol). Petiole ends were always included whether necrotic or not. Isolation plates were incubated in the dark at 20°C for 7–10 days and then microscopically examined for the growth of *P. ramorum*, which was recognized by a combination of distinctive hyphae, abundant chlamydospores and caducous, semipapillate sporangia. Several other *Phytophthora* species were recovered from rain traps, especially *P. pseudosyringae*, *P. nemorosa* and *P. pluvialis* from ITS Clade 3 (Hansen, Reeser, & Sutton, 2016). Individual traps were scored as positive or negative for *P. ramorum* for each two-week sampling interval based on culture results. Results were compiled as proportion of trap/intervals with positive recovery of *P. ramorum*.

2.2 | Disease spread from treated sites

If untreated, new infested sites may in turn become inoculum sources for further spread of SOD. To measure the effectiveness of the Oregon SOD local eradication strategy in halting subsequent local spread of the disease, we generated GIS maps with the locations of all trees confirmed with *P. ramorum* in each year from 2001 through 2017 (about 8,000 trees total). Each tree or clump of trees was assigned a "treatment site" number. Individual treatment sites that were distant by 0.5 km or more from other treatment sites identified in the same or previous years were selected for further analysis. Treatment sites were selected from maps showing mortality for each year through 2014, allowing disease monitoring in the immediate vicinity of each treatment site for at least three subsequent years, based on the annual aerial surveys.

Treatment effectiveness was measured as the number of years that the treatment site remained isolated by a distance of at least 0.5 km

TABLE 1 Characteristics of 45 sudden oak death treatment sites selected for "slow the spread" analysis, Curry County Oregon

Treatment site #	Year of *Phytophthora ramorum* detection[a]	Years without new infection within 0.5 km[b]	Distance to previously known infected tree (km)[c]	Area treated (Ha)[d]	Outside (O) or inside (I) generally infested area (2012 GIA)
2	2001	1	0.5	1.0	I
8	2001	7	1	0.1	I
11	2002	1	2	0.1	I
17	2002	1	0.5	0.1	I
29	2003	1	1.3	0.1	I
33	2003	2	0.5	0.4	I
80	2005	1	0.9	0.5	I
83	2006	1	0.6	3.5	I
87	2006	8	3.3	4.8	O
99	2006	1	0.9	1.0	I
101	2007	8	4.3	2.3	O
107	2007	5+	1.7	4.5	I
109	2007	1	0.9	1.4	I
114	2007	4	0.8	2.1	I
120	2007	10+	1.2	21.2	O
121	2007	10+	2.5	5.4	O
135	2007	1	0.5	3.1	I
150	2008	1	0.6	14.9	I
154	2008	4+	0.5	3.7	I
179	2008	8	3.4	2.8	O
184	2008	2	0.7	2.7	I
196	2008	3	0.8	2.8	I
206	2009	1	0.5	2.6	I
223	2009	3+	0.6	9.3	I
239	2009	3+	1.9	2.6	I
240	2009	1	0.5	2.6	I
247	2009	1	0.5	2.6	I
248	2009	1	0.5	2.6	I
251	2009	2	0.8	2.6	I
295	2010	7+	1.3	2.6	O
372	2011	6	19.8	38.2	O
441	2012	5+	6.7	12.2	O
450	2012	5+	2.1	2.6	O
481	2013	2	1.8	1.6	O
500	2013	3	1.3	2.6	O
525	2014	3+	1.8	2.1	O
544	2014	3+	0.7	2.5	O
545	2014	1	0.6	6.2	O
546	2014	1	0.6	2.6	O
547	2014	3+	0.5	3.0	O
550	2014	3+	4.6	2.6	O
551	2014	2	4.6	2.9	O
552	2014	3+	1.3	2.7	O
558	2014	3+	1.3	2.6	O
559	2014	3+	6.1	2.5	O

[a]Year of initial detection.
[b]Years before new infections recorded in adjacent stands.
[c]Distance from selected stand to nearest infection, at time of detection.
[d]Area treated by felling and/or hack and squirt.

from new mortality in surrounding stands. Distances were measured on the maps using ARCGIS mapping software. Forty-five treatment areas met these criteria (Table 1). For each selected treatment site, year of detection, distance to nearest treatment site, size of the treated area, number of trees infected at time of treatment and the number of years before new disease was mapped within 0.5 km were recorded.

3 | RESULTS

3.1 | Inoculum availability

3.1.1 | Period of sporulation

The average annual period of sporulation of *P. ramorum* in the SOD epidemic area in SW Oregon (Figure 4) was determined from a subset of the rain trap results collected between 2007 and 2014. Presence or absence of *P. ramorum* in trap baits was averaged by month for all traps placed beneath standing symptomatic trees in 38 known infested, untreated stands, before treatments started. An average of 177 (range 111–255) trap/intervals was scored for each month across the 8-year period. There were a total of 1935 trap/intervals tabulated.

Overall, *P. ramorum* was identified in 41% of the trap/intervals. In the wettest winter months (December and January), *P. ramorum* was recovered from bait leaves in rain traps in 63% of the trap/intervals. Periods with reduced rainfall tended to have reduced detection of *P. ramorum*. Lowest average recovery was in the dry summer months of August and September. Notably, sporulation continued at high spring levels (39% of bucket intervals) into July, even as average rainfall dropped (Figure 4). This was correlated with scattered short duration rain events at individual sites in some years. Untreated site BRT (conifer resistance test), for example, had only 2 days of rain in June and July in 2011. In those trap intervals with rain, however, 60% and 87% of 14 traps were positive for *P. ramorum*.

3.1.2 | Effects of eradication treatments

Phytophthora ramorum is not generally distributed in the Curry County quarantine area. It was recovered only once (<1 per cent of trap/intervals) from apparently healthy stands away from known infested areas (Table 2). The one rain trap with positive baits from otherwise healthy stands represented early detection of a new infection. Symptoms including bole cankers developed on this tree during subsequent monitoring and *P. ramorum* was isolated from it.

In infested stands, sporulation decreased with time, regardless of treatment. As trees died and foliage dried, turned brown, and eventually fell, recovery of *P. ramorum* in rain traps decreased. Site BRT, for example, was within the GIA and no treatments were attempted. In March 2011, rain traps were placed beneath 14 recently diagnosed symptomatic tanoak trees. Trees exhibited stem cankers typical of SOD, and *P. ramorum* was confirmed by direct isolation. Bait leaves in traps were monitored for *P. ramorum* at regular intervals through 2014 (Table 3). In that period, recovery declined from 43% of trap/intervals in 2011 to 11% of trap/intervals in 2014.

In known infested stands due for eradication treatment, *P. ramorum* was recovered at a high frequency (40% of the trap/intervals; 17 infested sites, all seasons of the year before treatments began) (Table 2). Recovery was reduced to 26% on average as treatment progressed (all treatment stages before burning slash) and declined to 7% of trap/intervals once treatments were completed (all tanoak trees cut and slash burned). Rain traps placed beneath healthy appearing tanoaks on the perimeter of treatment areas were seldom infested (2%).

At some sites, recovery of *P. ramorum* from rain traps after treatments were complete was associated in part with soil splashed into the traps placed on bare ground, as evidenced by traces of mud on and in the buckets. At Cape Sebastian (site 372) after treatment was complete, for example, soil splash was specifically noted in 11 of 73 trap/intervals. *Phytophthora ramorum* was baited from 64% of these buckets, but only from 8% of trap/intervals without observed soil splash. At other infested sites, soil splash was observed less frequently (2% of trap/intervals overall), and trap/intervals with soil splash were less often infested (14% recovery with soil splash and 13% without observed splash, n = 14,396). All reported recovery frequencies exclude results from the 268 trap/intervals where soil splash was observed.

3.1.3 | Sporulating hosts

Rain traps were placed beneath residual Douglas-fir, alder (*Alnus*) or Oregon myrtlewood (*Umbellularia*) trees after removal of infected

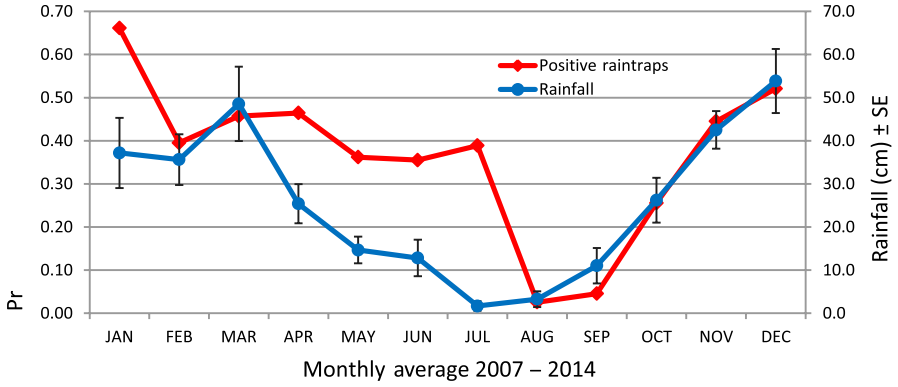

FIGURE 4 Proportion of rain traps positive for *Phytophthora ramorum* by month, averaged over the period from January 2007 to December 2014, and average monthly rainfall with standard error, recorded at the Red Mound Remote Automated Weather Station, includes all rain traps placed beneath known infected trees before eradication treatments were applied

Site condition	SOD treatment status	Number of trap/intervals	Trap/intervals positive for *P. ramorum*
Healthy	None	1,684	0.0006%
Infested	Before treatment	262	40%
Infested	Treatment in process	411	26%
Infested	Treatment completed	858	7%
Infested	Perimeter of treated area	1,145	2%

Note: Results from all seasons are averaged.

TABLE 2 Recovery of *Phytophthora ramorum* from rain traps in visibly healthy tanoak stands and in confirmed SOD infested stands where trapping was before, during and after site treatment and on the perimeter of treated areas in Curry County Oregon (October 2006–2015)

TABLE 3 Recovery of *Phytophthora ramorum* from rain traps beneath 14 known infected trees with years after initial detection at untreated infested site "BRT" in the generally infested area (GIA) in Curry County Oregon

Year	Trap/intervals	Percentage positive for *P. ramorum*
2011	270	43%
2012	220	36%
2013	161	21%
2014	154	11%

tanoak in 16 treated stands. Recovery of *P. ramorum* from the baited traps beneath residual trees was compared with recovery from traps placed beneath infected tanoaks before treatment. *Phytophthora ramorum* was isolated from traps beneath infected tanoaks in 40% of 2-week trap/intervals monitored (Table 2). Traps were placed beneath residual Douglas-fir at 11 sites; *P. ramorum* was isolated in four of the 961 trap/intervals monitored (0.003%). Residual red alder was monitored on four sites, and *P. ramorum* was recovered in three of the 141 trap/intervals (2%). Rain traps were placed beneath residual myrtlewoods at 16 sites and monitored for a total of 1,347 trap/intervals. *Phytophthora ramorum* was recovered at 10 of the sites and in 11% of the trap/intervals overall.

3.2 | Disease spread from treated sites

Forty-five initially isolated treatment sites that were: (a) detected between 2001 and 2014; (b) given operational local eradication treatments; and c. subject to follow-up aerial surveys for at least 3 years, were selected for analysis (Table 1). These treated sites remained distant from new infestations for an average of at least 3.2 years (range 1–10+ years). Both initial distance from other infested sites and size of the treatment area had significant but relatively weak relationships to the number of years the site remained isolated from new infestations ($p = 0.03$, correlation coefficient 0.35 and $p = 0.02$, correlation coefficient 0.33, respectively). The twenty-four treatment areas (53%) that had no new infestations within 0.5 km of the original treatment site after 2 years were more distant from other treatment areas at time of initial detection than the 21 sites where new mortality was mapped in the first two years after treatment (average 2.9 km vs. 1.0 km), and the area treated was larger than sites that had neighbouring mortality in the first 2 years (average 5.7 ha vs. 2.5 ha). Eleven of the 24 areas that were still isolated three years after treatment remained isolated after 5 years.

Twenty-four of the 45 analysed treatment areas were located within the GIA that was established in 2012 (Table 1). Most of these treatment areas within the GIA were first identified and treated earlier in the epidemic than treatment areas outside the GIA, and the area treated was smaller on average. They were also closer to other established infestations, some of which were not treated due to lack of funds. Treatment areas within the GIA remained isolated an average of 2 years, compared to 4.6 years for treatment areas outside the GIA. The difference was highly significant ($p = 0.0004$) (Table 4). After 2 years, 81% of the treatment areas outside the GIA were still isolated from encroaching infestations.

3.3 | Cumulative infested area

From 2001 to 2005, all SOD infestations occurred in what would become the GIA, and all received local eradication treatments. From 2006 through 2009, new infestations occurred inside and outside of the eventual GIA and all received eradication treatments. From 2010 onward, eradication treatments were stopped on private land inside the GIA while they continued on BLM land in the eastern part of the GIA. Soon after the cessation of treatments in the GIA, the number of new infestations and the cumulative infested area began to increase rapidly in comparison with areas outside the GIA (Figure 5). Inside the GIA, all infestations on non-federal land remained untreated, and by the end of 2017, there was 1,060 ha of untreated infestations inside the GIA (Figure 1).

4 | DISCUSSION

The effort to eradicate, and now more realistically, to slow the spread of SOD in SW Oregon has been costly, but has proceeded diligently despite lack of experimental demonstration of efficacy. Here, we provide the first data to support the success of the programme, although it still lacks proper replication and a comparable untreated

TABLE 4 ANOVA for years isolated by inside or outside the GIA

Source	Sum of squares	*df*	Mean square	*F*-ratio	*p*-value
Between groups	74.4004	1	74.4004	14.55	0.0004
Within groups	219.911	43	5.1142		
Total (corr.)	294.311	44			

control area, the intensive detection and monitoring effort does provide documentation of the development of the epidemic, and we believe that with caution, useful inferences can be drawn.

Baited rain traps proved a simple and efficient method for monitoring sporulation of *P. ramorum* in tanoak. We maintained sampling nearly continuously from January 2007 through December 2014. *Phytophthora ramorum* was recovered from the bait leaves in at least one trap in 165 of the 192 (86%) 2-week sample intervals across that time. Continued trapping of *P. ramorum* at spring high levels into June and July as rainfall decreased (Figure 4) was unexpected. Although SW Oregon has a Mediterranean climate with relatively dry summers, prolonged drought periods are infrequent and summer fogs also appear to provide sufficient moisture to trigger occasional sporulation and drip from the canopy. July is the driest month, but even then only 44% of the 2-week sample intervals during our 8 years of record were without any measurable precipitation. Winters are mild, and summer temperatures are moderated by the nearby ocean. The climate is very conducive for sudden oak death. The disease environment in SW Oregon is very different from that in infested areas of California. Bay laurel is less abundant, and Davidson and others describe a much more strongly seasonal sporulation pattern, favoured only by occasional spring rains. Many years see little or no sporulation due to extended droughts (Davidson et al., 2008, 2005; Fichtner et al., 2007; Fichtner, Lynch, & Rizzo, 2009).

The needs of the eradication programme precluded monitoring individual infected trees on most sites through the entire treatment period, but by moving rain traps to newly detected infestations, we were able to provide a continuous record. Similar problems stemming from the primacy of the eradication effort prevented synchronized, replicated comparisons of the efficacy of different stages in the treatment process. Measurement of treatment effects on sporulation was confounded by seasonal changes in frequency of sporulation events. Still, it was evident that sporulation decreased in most situations as trees died from cutting or herbicide application. It is not surprising that once infected trees were cut and burned, *P. ramorum* recovery decreased dramatically (Table 2). *Phytophthora ramorum* was recovered at a low frequency from traps set on cleared ground after treatments were completed, but much of this appeared to be due to soil-borne inoculum splashed into the buckets set on bare earth in recently disturbed treatment areas.

There is also the possibility that other, non-symptomatic and consequently untreated inoculum sources remain in stands after the tanoak is removed. Tree species other than tanoak may be left standing on treated sites. Douglas-fir is a listed host of *P. ramorum*, and infected seedlings and saplings are occasionally observed beneath infected tanoak trees. In laboratory tests, *P. ramorum* does produce sporangia on infected Douglas-fir twigs (J. LeBoldus, personal comm). Symptoms are not seen on mature trees in the forest regardless of proximity to infected tanoak, however, and sporulation on Douglas-fir is not known in the forest. Red alder (*Alnus rubra*) is another common associate of tanoak, but is not known to be a host. Oregon myrtle, a known and important sporulating host in California (Davidson et al., 2005; Garbelotto, Schmidt, Swain, Hayden, & Lione,

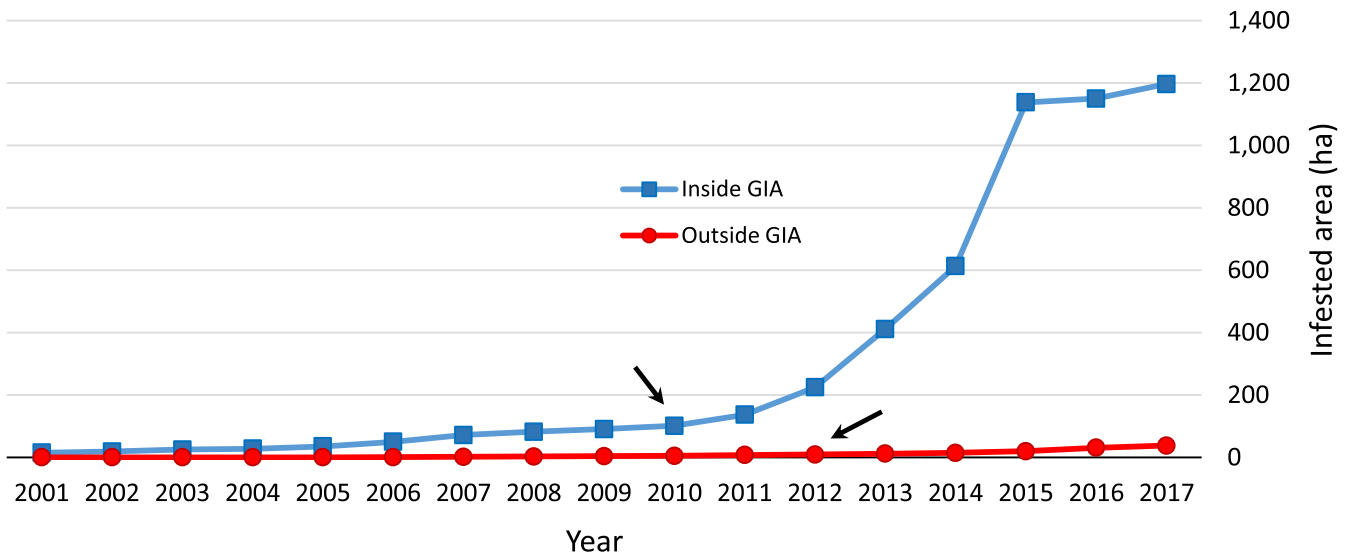

FIGURE 5 Cumulative SOD infested area within the GIA and outside the GIA. SOD eradication treatments stopped on non-Federal lands inside what became the GIA in 2010. The GIA was officially established in 2012. NA1 lineage only—EU1 infestations are excluded

2017), is also present in Oregon forests with tanoak, but often without symptoms.

Douglas-fir and alder are not providing appreciable inoculum; *P. ramorum* recovery rates from baited rain traps beneath these species are comparable to the very sporadic recovery from healthy tanoak stands. Oregon myrtle, on the other hand, is a sporulating host in Oregon as well as California, although infection appears to be quite variable and spore recovery rates are much lower than from tanoak. Oregon myrtle trees with visible confirmed foliage infections are removed in treatment, but individual symptomless trees may be left depending on local conditions. Diagnosis in the field is complicated by the common occurrence of other foliar *Phytophthora* species on Oregon myrtle leaves (Hansen et al., 2016).

It is significant that the pathogen was seldom recovered from rain traps on the perimeter of treatment areas. Local eradication treatments, including cutting a 100 m buffer of symptomless trees surrounding known infected trees, dramatically reduced local inoculum production and lateral tree-to-tree spread through splash dispersal.

Phytophthora ramorum can persist in soil for at least 11 years after elimination of the overhead inoculum source, and susceptible plants recolonizing the site from sprouts or seed are sometimes infected, although at a low and declining rate (Goheen et al., 2017). These observations led to restating the goal of the Oregon SOD management programme from "eradication" to "containment" or "slow the spread." Recovery rate from soil declines with years after treatment and shorter time intervals between detection and elimination of the inoculum source. We observed no evidence of epidemic spread in the regrowth vegetation. There have been no reports of disease in the forest originating from soil, although in horticultural situations, roots may be infected from soil-borne inoculum (Parke & Lewis, 2007). Plants are apparently not killed, and sporulating lesions typical of the nursery expression of SOD are not observed from this source. For practical purposes, soil in the forest may be a dead end reservoir for the pathogen.

Demonstrating the overall efficacy of local eradication treatments in slowing the spread of SOD on the southern Oregon landscape is frustratingly difficult. It has not been possible to maintain controlled, replicated experiments on a landscape scale due to the eradication requirements of Oregon law. The exigencies of high cost and sporadically available financial support, coupled with a few temporarily uncooperative private landowners, have ensured that inoculum levels remain high in some areas. The generally conducive climate in SW Oregon and the one- or two-year period between initial cryptic infection of a tree and its visible death (Peterson et al., 2014) mean that there is a prolonged period of inoculum production before local treatments are initiated. Still, the demonstration that untreated areas surrounding initially isolated infection sites remained disease-free an average of 3.2 years is encouraging.

Twenty-one of the 45 sites analysed had nearby infections one or two years after treatments were completed. Many of these "failed" sites probably already had infected trees in the surrounding stand that were not detected at the time of treatment. In those cases, it is often evident that initial treatment areas were not large enough. This was particularly true in the early years of the programme, before aerial dispersal of *P. ramorum* was recognized, and treated buffer distances were often only about 15 m.

In nearly all cases where appearance of SOD in the surrounding stand was delayed 3 or more years after treatment, examination of the maps suggested that the source of new infection did not appear to be the original treated area but rather the general advance of the epidemic front. In most cases (81%), prompt, complete treatment was effective in preventing outlier long-distance infections from becoming new epidemic centres. This is evident in a comparison of treated areas that were inside what in 2012 became the GIA and areas outside the GIA (Table 1) and in the difference in the cumulative infested area inside and outside the GIA (Figure 5). This conclusion validates epidemiological modelling that predicted this result (Václavik et al., 2010).

The lack of comparable "no treatment" control sites remains worrisome. Interpretation of high-resolution colour imagery each year from 2012 through 2017 clearly shows that the infested area within the GIA has expanded considerably in the absence of treatment (Figure 1). Goheen et al. (2017) used the same imagery to follow disease increase on 1 ha plots around single initially isolated killed trees; mortality went from <5% to >80% in 4 years. Similarly, Hansen et al. (2016), using historical Google Earth images, reported that 16 initially isolated tanoaks killed by *P. ramorum* in areas without local treatments had an average of 17 dead trees within 50 m 2–4 years later.

The reasons behind success or failure of local eradication attempts can be illustrated with four "case studies": (a) Emily Creek (site 179), on Rogue Siskiyou National Forest land, where early detection and prompt treatment led to a successful local eradication; (b) Cape Sebastian State Park (site 372), representing perhaps the optimum performance of the "slow the spread" programme; (c) the original infested sites detected in 2001, representing the limitations of actions taken with inadequate epidemiological knowledge; and (d) the GIA, where the realities of conducive weather and financial limitations compromised the programme.

a Emily Creek. In 2008, a single recently dead tanoak was detected near Emily Creek by aerial survey. Cause of death was confirmed by ground sampling, and the infested area was delimited that year and full treatment of the surrounding 7 acres (herbicide, cut and burn), along with an additional 21 acres of herbicide treatment only, were completed in 2009. Annual surveys detected no new infections in the surrounding tanoak stands until 2016.

b Cape Sebastian Park. Until 2011, the northernmost SOD infestation was about 31 km north of the original 2001 infestation. The 2011 aerial survey detected two recently dead tanoaks at Cape Sebastian, 19 km north of the nearest previously known infection. Another 14 symptomatic trees were detected by follow-up ground survey, and by 30 August that year *P. ramorum* was confirmed in culture and by PCR. The indigenous *Phytophthora*

nemorosa (Hansen et al., 2016) was also isolated from some symptomatic trees, justifying the culture effort instead of relying on symptoms alone. Because of the "long jump," 10 km beyond the established quarantine boundary, and because of the sensitivity of the site on State Park land, delimitation and eradication protocols were immediately initiated. Rain traps were deployed by October beneath symptomatic trees and beyond the apparent perimeter of the infestation, and hack and squirt and tree felling began in the core area. Treatments were completed by March 2012, and no further *P. ramorum* was detected in the rain traps after 28 March 2012. Although *P. ramorum* was isolated from soil samples collected in 2012 around the stumps of the infected trees, live tanoaks beyond the treatment area remained free of SOD through 2016. In 2017, aerial survey and follow-up ground checking mapped and confirmed 14 new symptomatic trees in the area. The original Cape Sebastian *P. ramorum* isolates were all NA1 genetic lineage (data not shown) (Ivors et al., 2006). In 2017, the new cultures were all EU1 lineage (Grünwald et al., 2016), evidently spreading northward from a recently discovered nursery infestation in Pistol River—5.5 km south. The new forest infestation of the EU1 lineage clearly originated from a new introduction to Curry County. In the case of Cape Sebastian, the local eradication effort was successful in blocking further local spread of the established, NA1, pathogen. It could not protect against a new, independent introduction of *P. ramorum*, however. That represented a failure of the state quarantine, not of the local "slow the spread" effort.

c First detections. In 2001, the first Oregon surveys for SOD started shortly after the causal agent was identified in California as an undescribed *Phytophthora* species. In that year, 9 clusters of dead tanoaks within 5 km of each other, each with 5–40 trees, were identified and confirmed with *Phytophthora* and it was quickly resolved to attempt eradication. A quarantine was established under state law, banning movement of soil or host plant material from the designated area and destruction of infected plants within the area. Intensive ground surveys delimited the treatment areas and cutting and burning tanoaks within a buffer distance of 15–30 m was begun. Four of the treatment areas (Areas 8, 11, 17 and 33) were sufficiently isolated to be included in the current analysis. In succeeding years, additional trees were diagnosed around the treated areas, however, and the infested area continued to expand. In retrospect, the initial treatment areas were too small, and the perimeters of the initial infections were not clearly delimited. The importance of aerial dispersal was not yet realized. The disease was already more widely established than initially apparent. Treatment appeared to halt further spread in only 1 of the 4 selected areas. Treatment area 8 was the furthest south, about 1 km across the Chetco River from the other areas. No new infections were found near area 8 for 7 years. Success in this case seemed to result from the tanoak free barrier formed by the river, and location of the site upwind (south) of the other infested areas. Prevailing winds are from the SW.

d The GIA. Establishment of the GIA in 2012 and cessation of control activities within its boundaries highlights the necessity of sustained financial support for the success of a long-term disease management programme. In 2008 and 2009, we were forced to suspend eradication treatments on private land several times because of State budget cuts. The first stoppage was from January to May 2008, on the western part of the quarantine area. When treatments resumed, we gave highest priority to treating new sites near the quarantine boundary and many older sites remained untreated for months. We resumed regular treatments in April 2010, but by this time we were dealing with a large backlog of untreated or partially treated sites accumulated from 2008 and 2009. As funds again became limiting in late 2010 and 2011, we gave priority to treating sites nearest the quarantine boundary. In 2012, the Oregon quarantine regulations were changed to reflect the new reality. The official goal was restated from "eradication" to "slow the spread" of SOD, and the GIA was legally established. Within its boundaries, treatment was no longer required (although treatments did continue on federal land) and restrictions on tanoak movement were eased. Available resources were redirected to treatments of new, outlier infestations.

In conclusion, eradication of SOD from individual infested sites is difficult but not impossible. The disease usually does not persist after cutting and burning known infected trees, but the pathogen may survive in soil for several years, although with minimal impact on regrowth vegetation (Goheen, Hansen, Kanaskie, Sutton, & Reeser, 2008; Goheen et al., 2017). Spread on the landscape continues because the pathogen may be present and sporulating from new infections on leaves and in twig lesions for a year or two before whole tree symptoms are visible. This limits early detection and coupled with delays in completing eradication treatments from funding challenges or operational constraints associated with summer fire seasons and availability of contract crews, prolongs the period of inoculum production and the chances for long-distance aerial dispersal. The wet, mild climate of SW Oregon allows sporangium production year-round, intensifying the need for prompt treatment.

Local treatments demonstrably reduce local inoculum levels. Financial realities forced establishment of the GIA, however, leaving an increasing number of infected tanoaks to produce sporangia for longer periods of time, thereby increasing inoculum pressure on healthy stands beyond the GIA and the disease continues to spread, although at a reduced rate.

The economic benefits of slowing the spread of SOD, in contrast to eliminating it, were recently analysed for the Oregon Department of Forestry (https://www.oregon.gov/ODF/ForestBenefits/Pages/ForestHealth.aspx)

("Sudden Oak Death: Economic Impact Assessment." Prepared by: Highland Economics, | Mason, Bruce & Girard, Inc., 2/15/2019).

The report concluded that over the next 20 years, the potential benefits of slowing the spread outweighed the costs of control

operations at a ratio of 19:1. Measured benefits were primarily derived from reduced risk of international sanctions that would severely disrupt timber exports from the major deep-sea port at Coos Bay, Oregon, north of the current range of sudden oak death. Importantly, the ecological and sociological costs and benefits of controlling the disease were discussed including cultural impacts to tribes, adverse impacts on aesthetics and related private property values, increased public safety risk and increased wildfire risk. These impacts could not be measured in economic terms in this analysis, however, because there is as yet no consensus on how to convert these values to monetary terms.

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