MODELING THE SPREAD OF SUDDEN OAK DEATH ACROSS A HETEROGENEOUS LANDSCAPE IN REDWOOD NATIONAL PARK USING A SPATIALLY-EXPLICIT EPIDEMIOLOGICAL MODEL

By

Laura A. Morgan

A Thesis Presented to

The Faculty of Humboldt State University

In Partial Fulfillment of the Requirements for the Degree

Master of Science in Biology

Committee Membership

Dr. Erik S. Jules, Committee Chair

Leonel A. Arguello, Committee Member

Dr. Richard C. Cobb, Committee Member

Dr. Christopher J. Dugaw, Committee Member

Dr. Jeffrey M. Kane, Committee Member

Dr. Erik S. Jules, Graduate Coordinator

May 2017

ABSTRACT

MODELING THE SPREAD OF SUDDEN OAK DEATH ACROSS A HETEROGENEOUS LANDSCAPE IN REDWOOD NATIONAL PARK USING A SPATIALLY-EXPLICIT EPIDEMIOLOGICAL MODEL

Laura A. Morgan

The pathogen *Phytophthora ramorum*, the causal agent of Sudden Oak Death (SOD), is responsible for the deaths of millions of oak (*Quercus* spp.) and tanoak (*Notholithocarpus densiflorus*) trees in California and Oregon (USA). A recent infection in Redwood National Park (RNP) in California (USA) provided an opportunity to adapt an existing SOD model to assess the efficacy of current and proposed management strategies. A common method of SOD treatment includes killing both infected and uninfected hosts in the area of infection, as well as the area surrounding the infection to create buffers to account for undetected or cryptic infections. I used the existing SOD model for a larger spatial area (380 ha) and included host density data. Using this model, I show that buffers of plausible width are not effective methods for managing SOD infections in RNP because they do not control spread of the pathogen. Additionally, I ran each model with two dispersal kernels (exponential and power-law) with equal mean spread distances and showed that the shape of the distribution kernel used can significantly alter the outcome of the model. For example, models using 300 m and 400 m buffers with an exponential dispersal kernel predicted containment of *P. ramorum*, but

spread beyond these buffers was predicted with a power-law distribution kernel. Lastly, my work provides the first evidence of significant stream-to-land spread of *P. ramorum*. I show laboratory-confirmed *P. ramorum* infections along a creek in RNP, which included low-hanging branches with cankers on host trees concealed by debris. I also used the adapted SOD model to compare two scenarios, one including and one excluding stream-to-land transmission, and found that the model that included stream transmission predicted future spread significantly better than the model that did not include stream transmission. This work not only highlights the problems associated with treating SOD infections by removing hosts in buffer zones surrounding infections, but also demonstrates how precise knowledge about the dispersal distance and dispersal frequency is required to derive accurate model predictions. Additionally, my work points to a novel transmission pathway for an important forest pathogen and highlights the need to determine the prevalence of this dispersal mechanism across the range of the pathogen.

ACKNOWLEDGEMENTS

I would first like to thank Leonel Arguello at Redwood National Park for repeatedly hiring me as the Sudden Oak Death technician and ultimately guiding me into graduate school. Without his support this project would not have been possible. Next, I would like to thank my graduate advisor Dr. Erik Jules for his tremendous effort assisting me through my graduate career. He is a great scientist, mentor, and person, and I am honored to have worked with him. I offer my sincere gratitude to Dr. Christopher Dugaw for his help and patience during many statistical and mathematical nightmares. Additional thanks to Dr. Richard Cobb and Dr. Noam Ross for creating and sharing the model framework I used in my thesis, and to Dr. Jeffrey Kane for his assistance and guidance on my proposal and thesis. Many thanks to my reliable field partners Kayla Conover, Salvador Amador, Annie Allen, and Brendan Twieg. Thank you to Jason Teraoka and Scott Powell for providing data and invaluable GIS support. Thank you Rachel McCain for helping me get my foot in the door with the NPS. Finally, I'd like to thank my family for their unconditional love and encouragement throughout this endeavor.

Funding for this research was awarded through a cooperative agreement between Humboldt Sponsored Programs Foundation and Redwood National Park, in collaboration with Leonel Arguello and Dr. Erik Jules.

TABLE OF CONTENTS

ABSTRACT ii

ACKNOWLEDGEMENTS iv

TABLE OF CONTENTS v

LIST OF TABLES vii

LIST OF FIGURES viii

INTRODUCTION 1

References 4

Chapter 1: Assessing management efforts to control the invasive forest pathogen, *Phytophthora ramorum*, across a variable landscape of hosts 6

Introduction 6

Methods 12

Study area 12

Model adaptation 13

Results 17

Discussion 19

References 28

Chapter 2: Spatial vegetation modeling suggests a novel transmission pathway of the invasive forest pathogen, *Phytophthora ramorum* 43

Introduction 43

Reported Information 45

Discussion 48

References 49

Appendix A: Spatial vegetation modeling suggests a novel transmission pathway of the invasive forest pathogen, *Phytophthora ramorum* supplemental information 55

Model Description 55

References 58

LIST OF TABLES

Table 1. Parameter values for the epidemiological model. Infection and pathogen-caused mortality rates are the weighted average of the reciprocal of the average time to the event (infection or mortality) for multiple size classes from the survival model in Cobb et al. 2012..... 35

Table 2. Comparison of four logistic regression models to determine the best predictor of *P. ramorum* infections in 2016 in the study area in Redwood National Park, California, USA. All models had 2 degrees of freedom and were computed using binomial generalized linear models with a logit link in R. 54

LIST OF FIGURES

Figure 1. Map of the study area along Redwood Creek in Redwood National Park (USA). Gray areas represent 2014/2015 treatment areas and black triangles represent *Phytophthora ramorum* positive tree locations found in 2016. Bold black line represents Redwood Creek and thin black lines represent major tributaries of Redwood Creek. Red box indicates area included in the epidemiological model. Inset map shows Redwood National Park in Humboldt County, CA, USA. 34

Figure 2. Exponential (black line) and power-law (dashed red line) distributions describing the dispersal of *P. ramorum* spores across the model area (distance shown in units of cells, 1 cell = 28.5 m). Distribution equations are described by Eqn 1 and Eqn 2 and parameter values are listed in Table 1. Values were chosen so the mean dispersal distance was the same (80 m) for both distributions. This value was chosen so that spores disperse beyond the nearest-neighbor in the grid while not spreading so far that the distribution becomes uniform. Both distributions allow most dispersal to occur locally but also account for long distance dispersal. The power-law (red line) distribution has a slightly fatter tail than the exponential distribution, however this is not readily apparent in the figure. 37

Figure 3. Control model figure. a) 380 ha model area at Bridge Creek in Redwood National Park (RNP), California, USA, showing tanoak (*Notholithocarpus densiflorus*) density prior to SOD infection and treatment. Color gradient darkens with increasing absolute host cover, ranging from 0 (white) to 117 (darkest green). Redwood Creek is shown in white through the middle of the figure. b) predicted proportion of model area infected after 55 years without any management action in RNP using exponential (dashed-line) and power-law (solid line) dispersal kernel distributions. c) spread, defined as cells with at least 1% infected tanoak during the 55 year model run, using an exponential dispersal kernel distribution. d) spread, defined as cells with at least 1% infected tanoak during the 55 year model run, using a power-law dispersal kernel distribution. 38

Figure 4. SOD spread over time (shown in pink) and the total area impacted by implementing treatment buffers (shown in black) for the 380 ha model area at Bridge Creek in Redwood National Park, California, USA. Black arrows indicate when treatment was initiated (I assumed this was 5 years after pathogen introduction). Treatment consists of injecting herbicide into tanoak (*Notholithocarpus densiflorus*) and bay (*Umbellularia californica*) hosts. A 100 m buffer increases the area impacted by 20% (76 ha), without stopping spread from escaping the buffer (a, e). Spread was predicted to cover approximately 35% (133 ha; power-law) and 15% (57 ha; exponential) of the model area with a 200 m (122 ha) buffer. Considering the removal of hosts in the buffered area, the total area impacted increases to approximately 67% (255 ha) and 47% (179 ha) of the

model area in these scenarios, and does not contain the pathogen (b, f). The exponential dispersal kernel in the 300 m and 400 m buffer scenarios predicts pathogen containment (c, d). However, the pathogen was predicted to spread beyond both the 300 and 400 m buffers when using the power-law dispersal kernel (g, h). In these scenarios, *P. ramorum* was predicted to infect approximately 17% (65 ha) and 7% (27 ha) of the model area, respectively. However, the 300 m and 400 m buffers themselves take up approximately 50% (190 ha) and 65% (247 ha) of the entire model area. While large buffers do not eradicate the pathogen over time, treatment would be expected to reduce inoculum by removing hosts, effectively slowing the spread when compared to control (no treatment) scenarios..... 39

Figure 5. Variable buffer treatment model figure for the 380 ha model area at Bridge Creek in Redwood National Park, California, USA under various buffer management scenarios. Panel shows spread (defined as cells that had 1% or greater infected tanoak) in pink over 55 years using an exponential dispersal kernel distribution on the left panel (a, b, c, d) and power-law dispersal kernel distribution on the right panel (e, f, g, h). Rows show spread 50 years post-treatment with a 100 m buffer (a, e), 200 m buffer (b, f), 300 m buffer (c, g), and 400 m buffer (d, h). Spread is predicted to be contained in the model with 300 m buffers with the exponential kernel, but not with a 300 m or 400 m buffer with the power-law kernel. Host removal does not eliminate spread due to the 5 years of uncontrolled disease spread before treatment and also because of an assumed 99% host removal efficiency rate. 41

Figure 6. A) Infected creekside tanoak (*Notholithocarpus densiflorus*) with low hanging branches over the infected Redwood Creek during low flow season. B) Clump of debris caught on creekside tanoak twig indicating submersion during high flow season. C) The same twig as in B but with the debris removed to reveal a blackened canker, a common symptom of Sudden Oak Death. This twig was collected, plated in PARP (pimaricin-ampicillin-rifampicin-PCNB) agar, and confirmed positive for *Phytophthora ramorum* in 2014..... 52

Figure 7. Model output for two hypotheses of Sudden Oak Death (SOD) spread in a 2.1 × 1.8 km area in Redwood National Park. The yellow area shows predicted SOD spread after ten years if the infection had originated at one location only. Each cell that was infected in 2014 and 2015 in the model area was modeled individually as the origin of infection and I chose the cell that exhibited the greatest amount of spread after ten years, as to maximize the potential spread under this hypothesis. The purple area shows predicted SOD spread after ten years if every infected creekside location discovered in 2014 and 2015 was treated as an independent stream-to-land transmission event. Darkened cells indicate infection initializations. Black triangles represent infected trees discovered in 2016. The green-scale cells in the background represent tanoak (*Notholithocarpus densiflorus*) density, with lighter cells containing less hosts and darker cells containing more hosts. See Table 1 for details of models and model parameters.... 53

INTRODUCTION

The pathogen that causes Sudden Oak Death (SOD), *Phytophthora ramorum*, is a nonnative oomycete which has caused extensive mortality of oak (*Quercus* spp.) and tanoak (*Notholithocarpus densiflorus*) trees in California and Oregon since the 1990s (Rizzo & Garbelotto 2003). Sixteen counties in California and one county in southwest Oregon are currently infected with *P. ramorum* (Cunniffe et al. 2016). Once the pathogen becomes established in a wildland environment, the pathogen is known to spread via warm, wind-driven spring rains while shedding its inoculum onto nearby susceptible trees such as California bay (*Umbellularia californica*) and tanoak. Only trees within a few meters of already infected trees are likely to receive enough spores to become infected (Davidson et al. 2005), however, infrequent long-distance dispersal events can dramatically accelerate spread in regions with high host density and suitable weather conditions (Meentemeyer et al. 2011). Many native plants host the pathogen with varying degrees of damage, however outright mortality is most common in tanoak. While California bay trees don't die from the disease, the leaves produce higher levels of inoculum than other host taxa (Davidson et al. 2005).

Redwood National Park (RNP) participates in a SOD early detection and monitoring program in Humboldt and Del Norte counties. In 2010, when *P. ramorum* was first detected in the water of Redwood Creek, a subsequent aerial survey demonstrated the infection was located approximately 8 km upstream of the RNP boundary. An eradication program was quickly developed and implemented at the site of

the infestation, however, the pathogen continued to spread. Unfortunately, the continued spread of SOD despite management actions is common. Managers in southern Oregon have defined a "generally infested area" around an infected area where management actions have ceased, even though management actions were implemented shortly after discovery of the disease in Curry County in 2001 (Peterson et al. 2015). In practice, successful eradication of the pathogen has not been possible. Consequently, the goal of management in southern Oregon has shifted from an eradication focus to a "slow-the-spread" campaign.

In July 2014 the first detections of *P. ramorum* were confirmed within RNP along the banks of Redwood Creek in two separate locations (near Bridge and Bond Creeks). RNP managers have determined an initial course of action in response to this discovery, which included the following strategies:

- Identify and establish a treatment area consisting of a core infestation zone within which the infection occurs, and also establish a 100 m buffer zone surrounding the core infestation zone to be treated along with the core zone.
- Within the entire management area (core zone and buffer) inject all diseased and healthy tanoak and bay trees with herbicides using a hypo-hatchet.

After subsequent ground-based monitoring in 2014, the sizes of the RNP infestations were determined to be approximately 23.5 and 3.2 hectares (ha). Buffers of 100 m were added around the infection foci to create 39.2 and 23.4 ha treatment zones, respectively. The Bridge Creek infestation was treated in August of 2014 and the Bond Creek infestation was treated in June of 2015. In the summer of 2015, an additional 28.3

ha were treated surrounding the original Bridge Creek infestation and an additional 24.6 ha were treated around the original Bond Creek infestation. Park managers believed the additional treated area in 2015 were likely also infected in 2014, but did not yield positive results in the laboratory due to the time of year the samples were taken (mid-summer) and the associated decrease in pathogen viability at that time (L. Arguello, personal communication).

Due to the impressive ability of *P. ramorum* to spread cryptically over long distances and the unsettling track record of previous land managers battling SOD, park managers are in need of localized spread models that incorporate various management strategies to understand and inform the optimal management strategy to employ (Filipe et al. 2012). The impacts of failing to slow the spread of *P. ramorum* in the park will likely result in stark changes to forest species composition and structure. Tanoak is the most abundant hardwood species in RNP and most creeks and drainages are densely populated by California bay. Although areas of dense alders (*Alnus rubra*) provide some host-breaks along Redwood and Bridge Creeks, along with areas of old growth redwood (*Sequoia sempervirens*) and mixed evergreen forests, none of these areas are completely void of these two hosts. Not only is tanoak a cultural resource considered sacred by native peoples (Bowcutt 2011), but loss of this species will have an indirect impact on other species at multiple trophic levels. Tanoak provides food for a variety of wildlife including birds and mammals, as well as habitat for a variety of wildlife and arthropods. It is an important tree resource that has no analog in the assemblage of species that currently exist in the redwood forests of RNP (L. Arguello, personal communication).

The main objective of my thesis was to evaluate the current SOD management protocol by modeling localized disease spread under various management scenarios. The information gained will feed directly into management practices by allowing park managers to focus early detection and monitoring efforts in the areas most likely to become infected, determine if current management protocols are sufficient, and explore alternative management options. I present my findings in two chapters. The first chapter focuses on the efficacy of buffer management and comparing models using two different dispersal kernels with the same mean spread distance. The second chapter presents evidence suggesting a novel transmission pathway (stream-to-land), a pathway previously considered epidemiologically insignificant in the origination of new infections (Grünwald et al. 2012, Peterson et al. 2014). The results of my study should allow park managers to understand spread potential in the context of the specific conditions occurring in RNP, model the impact of various management scenarios available to park managers, and help guide the management approach that is efficient and cost-effective for the park over the next several decades.

References

Cunniffe, N. J., Cobb, R. C., Meentemeyer, R. K., Rizzo, D. M., & Gilligan, C. A. (2016). Modeling when, where, and how to manage a forest epidemic, motivated by Sudden Oak Death in California. *Proceedings of the National Academy of Sciences*, *113*(20), 5640–5645. doi:10.1073/pnas.1602153113

Davidson, J. M., Wickland, A.C., Patterson, H.A., Falk, K.R., & Rizzo, D.M. (2005). Transmission of *Phytophthora ramorum* in mixed-evergreen forest in California. *Phytopathology 95*, 587–596 doi:10.1094/PHYTO-95-0587

Filipe, J. A. N., Cobb, R.C., Meentemeyer, R.K., Lee, C.A., Valachovic, Y.S., Cook, A.R., Rizzo, D.M., & Gilligan, C.A. (2012). Landscape epidemiology and control of pathogens with cryptic and long-distance dispersal: Sudden oak death in northern Californian forests. *PLoS Comput Biol 8*(1) doi:10.1371/journal.pcbi.1002328

Grünwald, N. J., Garbelotto, M., Goss, E. M., Heungens, K. & Prospero, S. (2012). Emergence of the sudden oak death pathogen *Phytophthora ramorum*. *Trends in Microbiology*, *20*(*3*), 131–138. doi: 10.1016/j.tim.2011.12.006

Meentemeyer, R. K., Cunniffe, N.J., Cook, A.R., Filipe, J.A.N., Huner, R.D., Rizzo, D.M., & Gilligan, C.A. (2011). Epidemiological modeling of invasion in heterogeneous landscapes: spread of sudden oak death in California (1990–2030) *Ecosphere 2*(2) doi:10.1890/ES10-00192.1

Peterson, E. K., Hansen, E.M., & Hulbert, J. (2014). Source or sink? The role of soil and water borne inoculum in the dispersal of *Phytophthora ramorum* in Oregon tanoak forests. *Forest Ecology and Management, 322,* 48–57. doi: 10.1016/j.foreco.2014.02.031

Peterson, E. K., Hansen, E. M., & Kanaskie, A. (2015). Temporal epidemiology of sudden oak death in Oregon. *Phytopathology 105*, 937-946 doi:10.1094/PHYTO-12-14-0348-FI

Rizzo, D. M. & Garbelotto, M. (2003). Sudden oak death: endangering California and Oregon forest ecosystems. *Frontier in Ecology and the Environment. 1*, 197-204 doi:10.1890/1540-9295(2003)001[0197:SODECA]2.0.CO;2

CHAPTER 1: ASSESSING MANAGEMENT EFFORTS TO CONTROL THE INVASIVE FOREST PATHOGEN, *PHYTOPHTHORA RAMORUM*, ACROSS A VARIABLE LANDSCAPE OF HOSTS

Introduction

Invasive forest pathogens have caused dramatic changes in many ecosystems worldwide and effective management of these pathogens requires an understanding of the factors that control their rate of spread (Byers et al. 2002, Aukema et al. 2010, Liebhold et al. 2012). The rate of spread of invasive forest pathogens is governed, in part, by its dispersal mode (Hastings 1996, Hastings et al. 2004, Carrasco et al. 2010). Some of the most impactful pathogens demonstrate rapid dispersal in air and water, often making management strategies aimed at slowing spread or eradicating new infestations impossible. For example, *Cronartium ribicola*, the causal agent of white pine blister rust, is a fungal pathogen dispersed by wind that has drastically reduced whitebark pine (*Pinus albicaulis*) populations in western North America (Kinloch 2003). In contrast, pathogens that spread through slower, or more predictable pathways allow for easier control of the invasion. Forests in northern California and southwestern Oregon (USA) have seen the rapid decline of Port Orford cedar (*Chamaecyparis lawsoniana*) due to the spread of *Phytophthora lateralis*, an invasive pathogen thought to be native to parts of Asia (Hansen et al. 2000). Unlike many forest pathogens, *P. lateralis* spores spread either by moving water or through transportation of mud carried by vehicles that can accidentally be deposited in riparian corridors inhabited by the host (Jules et al. 2002, 2014). The

absence of aerial dispersal in *P. lateralis* makes management of its spread far easier than for other pathogens such as *C. ribicola* (Hansen et al. 2000).

Management efforts have been employed to control and/or eradicate numerous pathogens with a wide range of dispersal modes. For example, large-scale efforts have been employed for the aerially-dispersed American chestnut blight (*Cryphonectria parasitica*), coffee leaf rust (*Hemileia vastatrix*), and white pine blister rust, as well as the water-dispersed jarrah dieback (*Phytophthora cinnamomi*), *P. lateralis*, and the water-dependent witch's broom rust (*Moniliophthora perniciosa*) on cocoa (Freinkel 2007, Arneson 2000, Kinloch 2003, McDougall et al. 2002, Hansen et al. 2000, Purdy & Schmidt 1996). However, when the epidemiology of the system is poorly understood, seemingly appropriate management actions can be insufficient or even increase pathogen spread and disease intensity (Mbah & Gilligan 2010, Filipe et al. 2012, Cobb et al. 2013). For example, a consequence of extensive cutting of dead and dying chestnut was the opening of the overstory of these forests, which served to increase spread of the pathogen (Freinkel 2007). Control efforts for whitebark pine blister rust were focused on breaking the disease cycle with the pathogen's alternate host (*Ribes* spp.) through eradication efforts, but with many *Ribes* spp. widely spread, this impossible and ineffective task was later referred to as "ecological warfare on a biblical scale" (Kinloch 2003).

Unfortunately, it is rare that the efficacy of control measures can be assessed in a meaningful way to inform managers, and few examples are available to serve as methodological templates for other systems (Hastings et al. 2006, Carrasco et al. 2009, Filipe et al. 2012).

The current epidemics of Sudden Oak Death (SOD) are similar in extent and severity to historical outbreaks of several well-known pathogens, including chestnut blight, Dutch elm disease, and white pine blister rust (Filipe et al. 2012; see also Brasier 1991, Kinloch 2003, Elliott & Swank 2008). SOD (a disease caused by the nonnative oomycete *Phytophthora ramorum*) and was discovered in California in 1995 and in Oregon in 2001 (Goheen et al. 2002, Rizzo et al. 2002) and has since spread to 16 counties in California and one county in Oregon via natural (wind, stream, and rain-splash) and human-mediated (nursery trade) transport. SOD has far-reaching ecological, economic, and cultural impacts (Meentemeyer et al. 2008, Cobb et al. 2013). The loss of tanoak has resulted in changes to forest composition and structure, and has had an indirect impact on other species at multiple trophic levels. SOD can also increase fuel loads and decrease foliar moisture content of tanoak, thereby increasing the possibility of crown fire in affected areas (Kuljian & Varner 2010, Valachovic et al. 2011, Metz et al. 2012). Additionally, tanoak is a cultural resource, considered sacred by native peoples (Bowcutt 2011). Tanoak is an important tree resource that has no analog in the assemblage of species that currently exist in redwood forests (L. Arguello, personal communication).

In Oregon, efforts to eradicate *P. ramorum* have been underway since its discovery over 15 years ago. Eradication protocol consisted of removing all main hosts and was usually accomplished by cutting and burning, within a minimum buffer of 100 m from the outermost infected sample (Goheen et al. 2004, Peterson et al. 2015). However, the emergence of new infections at the periphery of known sites and the occurrence of

long and unpredictable jumps between sites have continued to confound eradication goals (Hansen et al. 2008, Peterson et al. 2015). A dramatic increase in SOD-infected forests was detected in Oregon in 2011, forcing the eradication program to be replaced with a "slow-the-spread" management regime in the area now considered "generally infested" (Peterson et al. 2015). Although the eradication program did not eliminate *P. ramorum* from Oregon forests, it likely reduced inoculum build-up and slowed the epidemic significantly (Hansen et al. 2008).

Similar to the protocol implemented in Oregon (but on a much smaller scale) management of isolated outbreaks of *P. ramorum* in California have largely relied on removal of infected hosts to reduce inoculum. These treatments are also often combined with removal of susceptible hosts in the surrounding area (buffers) in an attempt to account for cryptic, undetected infections. While these efforts are likely effective in reducing local spread (Hansen et al. 2008, Goheen et al. 2004, Filipe et al. 2012), in each case the pathogen has not been eradicated by the treatments and has continued to spread throughout the landscape (Cobb et al. 2013). This is attributable to the biology of the pathogen and its ability to withstand adverse conditions and spread asymptomatically over long distances, and to the suitable climate and host availability in northern California and southern Oregon (Filipe et al. 2012, Cobb et al. 2012). For example, Meentemeyer et al. (2011) found that models predicted a ten-fold increase in disease spread between 2010 and 2030, with infection concentrated specifically along the north coast between San Francisco and Oregon.

Epidemiological models can help guide management by evaluating the efficacy of treatments and forecasting the spread and impacts of pathogens; in several cases, the dynamics of invasive species spread have been successfully captured in population models (Hastings 1996, Higgins & Richardson 1996, Madden et al. 2007). However, precisely determining the distances that propagules move and their frequency becomes increasingly difficult when a pathogen has the ability to disperse over long distances (Rodrigues et al. 2015, García & Borda-de-Água 2016). It is now well understood that long distance dispersal is a strategy used by many species (Kot et al. 1996, Kelly et al. 2014, Rodrigues et al. 2015).

A dispersal kernel with a lower rate of decay (a fat-tailed kernel) accounts for long-distance dispersal (Kot et al. 1996, Rodrigues et al. 2015). While it is natural that the kurtosis would have an impact on containment of dispersed propagules, fat-tailed kernels tend to give accelerating invasion spread rates while other kernels yielded constant invasion rates (Kot et al. 1996, Garnier 2011, Rodrigues et al. 2015). Although challenging, determining the appropriate dispersal kernel for the organism of interest is crucial, as the conclusions drawn from using different distribution kernels may be drastically different. For example, it was demonstrated that rates of seed spread can increase by an order of magnitude even when the frequency of long distance dispersal is extremely low (Higgins & Richardson 1999). Interestingly, Meentemeyer et al. (2011) and Filipe et al. (2012) concluded that a power-law function fit their observed *P. ramorum* spread data significantly better than a negative-exponential function, indicating dispersal over longer distances. Because of the importance of dispersal mode in

controlling spread rates, any attempt to assess potential management efforts must incorporate dispersal modes explicitly, including rates of long-distance dispersal (García & Borda-de-Água 2016).

The initial risk models created to describe SOD spread in California and Oregon identified high priority locations for early detection that were optimal for pathogen invasion (Meentemeyer et al. 2004, Václavík et al. 2010). More recent models have incorporated local spread following establishment in specific locations (Meentemeyer et al. 2011, Filipe et al. 2012, Cobb et al. 2013). One such model, the stand-scale SODDr model, (Cobb et al. 2012; packaged into R under the title "SODDr" [Sudden Oak Death Dynamics in R]) is a spatially explicit, density dependent, stage structured model that was used to predict tanoak population decline, changes in canopy structure, and stand composition on a 20-ha lattice. Model parameters were estimated using plot-level measurements from over 200 long-term study plots in northern and central California. The SODDr model simulates the terrestrial spread of disease and resulting tree mortality and stand dynamics in a mixed system of tanoak, bay, and redwood (*Sequoia sempervirens*), and outputs a matrix of population densities by species, infection status, and location. With this methodology, Cobb et al. (2012) were able to determine that larger, overstory trees are likely to be greatly reduced or eliminated in California forests.

The work presented here had five objectives: (1) adapt the SODDr model based on empirical measures of host density in a 380 ha forested landscape in Redwood National Park (RNP), (2) use the model to predict spread over the course of 55 years with no control measures, (3) assess the potential of using 100 m, 200 m, 300 m, and 400 m

buffers to contain spread for 50 years post-treatment, (4) assess the differences between exponential and power-law dispersal kernels, and (5) discuss the management implications of my findings. The results of this study will allow park managers to understand the spread potential in the context of the specific conditions occurring in RNP by modeling the impacts of various management scenarios. This information will help guide a management approach that is efficient and in alignment with RNP's mission to conserve natural resources. Additionally, this work provides a basic methodology for adapting existing models to include site-specific details to simplify modeling efforts and make predictions about other forested landscapes.

Methods

Study area

In 2010, an aerial survey discovered a new *P. ramorum* infection along a Redwood Creek in northern California approximately 8 km upstream of the RNP boundary. The University of California Cooperative Extension quickly developed and implemented an eradication program at the site of the infestation; however, the pathogen continued to spread. In July 2014, the first detections of *P. ramorum* were confirmed downstream, within RNP, along the banks of Redwood Creek in two separate locations near the confluences of Bridge and Bond Creeks with Redwood Creek (Figure 1). These infections were separated by approximately 3 km and were approximately 15 km from the upstream infestation discovered in 2010. Although not considered a common dispersal mode, both infections are thought to have transferred from Redwood Creek

water to hosts via debris caught on the stems as the critical medium for transmission (see Chapter 2). RNP managers established treatment areas with the use of 100 m buffers surrounding infected areas and either cut or injected all diseased and healthy tanoak and bay trees with herbicide. As of 2015, approximately 68 ha and 48 ha were treated at the Bridge Creek and Bond Creek infection sites, respectively. I chose to model 380 ha of remote, backcountry, redwood/Douglas fir/tanoak forest at the confluence of Bridge Creek and Redwood Creek, where SOD was originally discovered in RNP. Using geospatial software, areas extending 400 m away from the 2014 and 2015 Bridge Creek treatment zones were mapped to create the 380 ha model area. The model area was then divided into a lattice of 4,725 812.25 m^2 cells (each 28.5 × 28.5 m). The cell size was chosen to be congruent with the cell size of the vegetation data which was used to determine the initial spatial distribution of hosts in RNP. These changes did not impact the model predictions made by the original SODDr model.

Model adaptation

I modified the SODDr model to create the RNP model (here on called "RNP model"). The RNP model is a version of the SODDr model that has been modified for a larger spatial area and includes host density data. For simplification, parameters for tanoak species were combined into a single size class. Therefore, the parameter values representing the transition rates of tanoaks from one size class to the next larger size class were omitted and recruitment rates were calculated for steady-state levels by dividing the mortality rate by the density-dependence coefficient at simulation start. Weighted averages of tanoak parameters from the SODDr size classes were used to represent the

single size class. The tanoak mortality rate parameter was the most sensitive, so I ran the model with a 20% increased and a 20% decreased from average tanoak mortality rate parameter. For a complete list of parameter values and origins, see Table 1. A significant addition in the RNP model from the SODDr model was the inclusion of empirical estimates of hosts, which exhibited spatial heterogeneity across the study landscape. To determine host densities, I used RNP's recently completed vegetation classification and vegetation land cover map, created from remotely sensed and ground-verified data, as a part of the National Park Service Vegetation Mapping Inventory (Stumpf et. al. 2017 *in review*). The map employed the U.S. National Vegetation Classification (USNVC) to map 57,491 ha of RNP. The proportions of tanoak, bay, and redwood were determined in each cell by dividing the species density by the total tree density.

The transmission of infection within and between cells was determined by the proportion of spores deposited within and between cells, and was calculated in a similar fashion as in the SODDr model. However, to model different management scenarios realistically, dispersal was not limited to adjacent-only cells. This was because the infection in RNP is quite young, and if spores could only reach their nearest neighbor in the model, simulations with buffers surrounding infected areas would appear to contain the infection in the model. Since this has not been the case in RNP or any other areas where buffer management protocols were implemented, I assumed spores are capable of spreading beyond the nearest neighbor cell with likelihood decreasing with distance. Two dispersal kernels (exponential and power-law, shown below respectively) were

considered to describe pathogen dispersal rates and fit the actual *P. ramorum* observations from RNP.

Eqn 1: $\gamma \times e^{-\lambda \times distance}$

Eqn 2: $\gamma \times \frac{1}{(distance)^{\beta}}$

The parameters λ and β are both used to estimate dispersal distance of *P. ramorum* spores. In the exponential distribution equation (Eqn 1), λ describes the rate of decay of dispersed spores with distance. Similarly, β, the exponent on distance for the power-law distribution equation (Eqn 2), describes the reduction of dispersed spores with distance. The values for λ (0.76) and β (2.91) in the equations above were chosen so the mean dispersal distances (in units of cells; approximately 2.8 [80 m]) were equal in both models. In both equations, γ is a normalization constant chosen so that the total amount of dispersal outside the infected cells is 1. In other words, this allows both dispersal kernels to disperse over the entire grid. Values for γ were calculated by either dividing by the total amount of dispersal outside of the center cell for exponential dispersal, or by subtracting 0.1 (i.e., proportion of spores that spread within a cell, explained below) from 1 and dividing by the total amount of dispersal outside the center cell for power-law dispersal. The mean dispersal distance (80 m) was chosen so that 1) long-distance dispersal could be accounted for, and 2) buffer scenarios could be modeled. A smaller mean dispersal distance would reduce spread to only the nearest-neighbor cell, and a larger mean dispersal distance would begin to resemble a uniform dispersal distribution. Both exponential and power-law distributions account for infrequent long-distance

dispersal, however the highest frequency of spread occurs locally. The main difference is the width of the tail – the fatter tail of the power-law distribution accounts for more long-distance dispersal than the exponential distribution (Figure 2).

The amount of spread from infected cells to susceptible cells was calculated by first setting a dispersal coefficient for a grid so that the total dispersal outside of infected cells was 0.90 (i.e., 0.10 falls within the center cell and 0.90 is spread out of the cell). As in the SODDr model, the rate of transmission of infection between host species was described by the "Who Acquires Infection From Whom" (WAIFW) matrix (Anderson & May 1985). Additionally, like many hardwood trees, disease-killed tanoak develop basal sprouts from below-ground tissues. This feature was accounted for in both the SODDr and RNP models. Due to the fine scale of the model, neither topography nor climate data were included in the SODDr or RNP models. These omissions and their possible implications are discussed further in later sections.

I began spread simulations by initializing each cell that contained an observed infected tree in 2014 or 2015 (26 cells total) to have 50% infected hosts. With these assumptions, several management scenarios were explored, including: (1) control with no removal of hosts in order to predict natural spread, (2) removal of hosts in the infected area plus 100 m buffer (current treatment protocol), and (3) removal of hosts in the infected area plus 200, 300, and 400 m away from the infected area (increased-treatment protocols). The control scenario, in which no management actions were simulated, was modeled first to represent how the pathogen may spread in the absence of treatment. It is likely that the pathogen had been at the site for more than two years prior to observation

because the estimated average time between tanoak infection and mortality is about 2.5 years (Filipe et al. 2012). To purposefully overestimate this period of initial infection, I chose to allow the infection to spread in the model for five years before removing hosts to account for treatment. To simulate treatment, host density values were decreased by 99% in the areas that were treated in 2014 and 2015, and the model was allowed to run for 50 years post-treatment.

Two additional models were run to examine differences between homogenous and heterogeneous host distributions. The average tanoak density for the entire 380 ha model area (47%) was applied homogenously in one model and heterogeneous host densities in the other. Otherwise these models were identical – no treatment was accounted for, no buffers were applied, and the same distribution kernel was used (power-law) over the same period of time (55 years). If heterogeneous host density was not significantly influencing the spread of the disease, while other parameters in Table 1 were held constant in both models, I expected the differences between the model outputs to be negligible.

Results

Host density affected the rate of *P. ramorum* spread across the 380 ha model area, visible by the greater incidence of spread in denser host areas (Figure 3a, 3c). Specifically, regions to the northeast of Redwood Creek have higher host density (approximately 70% tanoak) than the region to the southwest (approximately 20% tanoak). The model predicted greater spread into the higher host density region as shown

in Figure 3c. When all cells were divided into high and low tanoak density groups (<45% and >45%) at the end of the control (no treatment) power-law model scenario, the average percent infected tanoak for the low density group was 7% whereas the average percent infected tanoak for the high density group was 22%. Additionally, more cells became infected in the model when a homogenous tanoak density of 47% (the average over the entire model area) was applied to the entire grid. A total of 4,625 cells were predicted to be infected after 55 years with no treatment with a homogenous tanoak density grid. When host heterogeneity was applied, the same model predicted 4,149 infected cells over the same period of time, reinforcing the assumption that host heterogeneity plays a significant role in modeled disease spread (spread defined as 1% infected hosts/cell or greater).

In the no-treatment scenarios, the exponential dispersal model initially accelerated spread more quickly than the power-law dispersal model, but over time (55 years) the power-law kernel spread the pathogen further than the exponential kernel (Figure 3b). Without treatment, spread was predicted to cover approximately 90% (342 ha) and 75% (285 ha) of the entire study area over time using power-law and exponential dispersal kernels, respectively (Figure 3c, 3d). The models predicted that the pathogen would spread beyond the 100 m buffer after 50 years using either dispersal kernel, infecting approximately 52% (198 ha; power-law) and 40% (152 ha; exponential) of the model area. However, when considering the effect of treatment (removal of tanoak and bay in the treatment areas), the total area impacted in the 100 m buffer treatment scenario increases to approximately 72% (274 ha) and 60% (228 ha) of the model area (Figure 4a,

4e). While overall spread was reduced in the 100 m buffer scenarios compared to the no treatment (natural spread) scenarios, a 100 m buffer also increases the area impacted by 20% (76 ha), without stopping spread from escaping the buffer. The same result was observed with the 200 m buffer, but with less spread beyond the buffer than in the previous scenario. Spread was predicted to cover approximately 35% (133 ha; power-law) and 15% (57 ha; exponential) of the model area with a 200 m (122 ha) buffer. Considering the removal of hosts in the buffered area, the total area impacted increases to approximately 67% (255 ha) and 47% (179 ha) of the model area in 200 m buffer scenarios, and does not contain the pathogen (Figure 4b, 4f).

When using the exponential dispersal kernel, the pathogen was contained in the 300 m and 400 m buffer scenarios (Figures 5c, 5d). However, the pathogen was predicted to spread beyond both the 300 and 400 m buffers when using the power-law dispersal kernel (Figures 5g, 5h). In the 300 m and 400 m buffer scenarios using the power-law dispersal kernel, *P. ramorum* was predicted to infect approximately 17% (65 ha) and 7% (27 ha) of the model area, respectively. However, the 300 m and 400 m buffers themselves take up approximately 50% (190 ha) and 65% (247 ha) of the entire model area. Therefore, these methods would still impact 67% (255 ha) and 72% (274 ha) of the model area, effectively slowing the spread but not stopping it from moving across the buffers over time (Figures 4g, 4h).

Discussion

My model showed that as buffer sizes increased, overall spread 50 years post-treatment decreased (Figures 4, 5). However, spread, defined as cells with at least 1% of hosts infected, was only contained in the models that used buffers of 300 m or greater (Figure 4c, 4d). Overall spread across all treatment scenarios was reduced compared to the natural spread (no treatment) scenarios, where the majority of the area included in the model was predicted to be infected over time (Figure 3). Thus, while host removal buffer treatments do appear to slow the spread of *P. ramorum*, they do not appear capable of eradicating *P. ramorum* from forested ecosystems. Furthermore, the adapted SODDr models underestimated actual spread observed in the study area, as areas predicted to be infected 50 years following a 100 m host removal buffer treatment have become infected much faster in reality. I also found large differences in model outcomes when using exponential or power-law distribution kernels, and when using homogenous or heterogeneous initial host densities. These results agree with other recent SOD modeling efforts which claim that eradicating *P. ramorum* from California forests is not a possibility (Cunniffe et al. 2016).

P. ramorum spores are spread locally (<20 m) via rain splash but can be driven longer distances (up to 4 km/year) by wind and rain (Davidson et al. 2005, Filipe et al. 2012). Interestingly, *P. ramorum* is also commonly detected in rivers and streams downstream of infected areas by baiting host leaves in infected waterways (Davidson et al. 2005, Frankel 2008). This method of early detection is referred to as “stream baiting”, but SOD researchers have concluded that stream-to-land dispersal of *P. ramorum* (natural stream baiting) has not occurred in any epidemiologically significant situation (Rizzo et

al. 2005, Parke & Lucas 2008, Peterson et al. 2014, Grünwald et al. 2012). However, the conspicuous pattern of infection along Redwood Creek with large gaps between infected trees, coupled with the presence of SOD-positive cankers concealed by debris (indicating submersion in the infected water of Redwood Creek during the rainy season), suggests that *P. ramorum* is utilizing a stream-to-land transmission pathway not previously considered significant in *P. ramorum* epidemiology (Morgan et al. 2017, *in review*). For example, in 2015, spread was observed along Redwood Creek over 800 m downstream of the 2014 Bridge Creek treatment zone (Figure 1). The same trend was observed in 2016, with distances between infected, creekside tanoaks reaching over 1.5 km downstream in several locations (Figure 1). Evidence of this uncommon transmission pathway, along with *P. ramorum*'s already-impressive ability to disperse cryptically, over long distances and infect a diverse range of hosts, strongly suggests that the current 100 m buffer treatment scenario (as well as larger buffer treatment options) are ineffective in stopping the spread of SOD, especially with the favorable climate and high host density of northern California (Judelson & Blanco 2005, Grünwald et al. 2008, Meentemeyer et al. 2011, Filipe et al. 2012).

In the original SODDr model, initial host densities were applied homogenously to the entire model grid. However, the inclusion of host heterogeneity caused significant differences between otherwise identical models. The average host density across the model area at Bridge Creek in RNP was 47%. When this value was applied homogenously, an additional 476 cells became infected compared to the heterogeneous host density model. This is the result of a reduction in detail; assuming a homogenous

47% tanoak density is analogous to painting with a broad brush. Comparatively, terrestrial (creek cells not included) tanoak host density values range from almost zero (0.6%) to a dominant 76.2% when host heterogeneity is examined. In this case, the detail included by accounting for host heterogeneity caused the spread of the infection to decrease in the model. It is safe to assume that if a smaller homogenous initial value had been chosen (perhaps 20%), spread would be reduced compared to the heterogeneous model.

Different dispersal kernels also created significant differences between model outputs. The shape of the distribution used in the kernel is a representation of a pathogen's ability to spread over distances at certain frequencies, and the longer the distance of dispersal, the more widespread the epidemic (Zadoks & Schein 1979). While both distributions in my models follow leptokurtic dispersal curves, exponential dispersal curves still approach a constant invasion speed, whereas power-law (fat-tailed) dispersal curves are extremely leptokurtic and lead to accelerating invasion rates, even with short average dispersal distances (Kot et al. 1996, Clark 1998). The mean dispersal distance (in units of cells; 2.8, or 80 m) was the same for all models regardless of kernel distribution, but the conclusions drawn from the models with different kernels are significantly different. The exponential kernel models predict pathogen containment whereas the power-law kernel models do not. Judging by the exponential kernel models, managers may be inclined to create 200 m or 300 m buffers around infections with the expectation of containment. However, since power-law functions have better fit data in previous SOD modeling efforts (Meentemeyer et al. 2011, Filipe et al. 2012), it is reasonable to assume

that a 400 m buffer would not have contained the pathogen at Bridge Creek in RNP, as evidenced by the power-law model results. The discrepancies between the models using either exponential and power-law dispersal kernels suggests more information is needed about the long-distance dispersal capabilities of pathogens to ensure dispersal kernels in modeling accurately represent observed spread distances and their frequencies. Additionally, researchers using models should test multiple dispersal kernels before interpreting results, as model results may be sensitive to the chosen dispersal kernel.

Many models have been constructed to understand the spread of SOD, both at small and large spatial scales. These studies have resulted in a wide range of tools and information, including: risk maps at various scales; significant biotic and abiotic explanatory variables; probable time of invasion and decline of specific tanoak populations; changes in ecosystem structure and function; and insight as to how to most effectively manage the spread of SOD; (Meentemeyer et al. 2004, 2011, Václavík et al. 2010, Cobb et al. 2012, Filipe et al. 2012, Peterson et al. 2015, Cunniffe et al. 2016, Haas et al. 2016). The earlier models – developed since the pathogen's introduction into California in the mid-1990's – have become reliable tools for managers because they have repeatedly demonstrated the accuracy of many epidemiological assumptions (Meentemeyer et al. 2012). For example, the initial risk maps identified areas where invasion was likely, such as the stretch of forest between Sonoma county, California, and the Oregon border (Meentemeyer et al. 2004, 2011, Filipe et al. 2012). More recent SOD models have overlaid additional data to the initial risk models to aid management and conservation efforts. Meentemeyer et al. (2011) applied a stochastic, epidemiological

model to California landscapes over a 40 year period (1990-2030) and found that while most *P. ramorum* spread happens locally, long-distance dispersal in areas with suitable weather conditions and high host density can drastically increase the spread of the disease. By applying their model to all of California, they determined that northern coastal California was at a much higher risk of infection than the Sierra Nevada foothills and coastal southern California due to host availability and climate. Filipe et al. (2012) used a probabilistic, spatially-explicit metapopulation model to demonstrate that successful *P. ramorum* management depends on the spatial scale of invasion as well as the type of treatment applied. Their models showed that large-scale preventative treatments ahead of the infection, together with removal of infected hosts (if started in 2010 and repeated every 8-10 months) could have slowed the speed of spread from approximately 4 km/year to approximately 0.5 km/year, and could have contained spread for more than 6 years. If treatment started earlier (2005), a greater reduction in spread was predicted. However, as Filipe et al. (2012) stated, the large size of the area required to achieve this level of reduction in spread (>500 km by 2017) would be a major challenge for managers financially and logistically.

Using the SODDr model in California, Cobb et al. (2012) revealed how *P. ramorum* affects tanoaks of various sizes and how community-level epidemiology plays a role in disease intensity. They discovered that tanoak is likely to persist in many infected forests due to its prolific resprouting ability, but overstory trees are likely to be greatly reduced, if not eliminated. Additionally, they showed that host-density thresholds may exist in forests, as low densities of tanoak among other non-susceptible hosts resulted in

much slower transmission. Similarly, Haas et al. (2016) used survival analysis modeling framework to show how accounting for heterogeneity across multiple levels (individual, community, and landscape) better predicted infection than models that only focused on one level. Fortunately, with decades of SOD research and well-supported models, adapting the models for other specific locations is much more reasonable of a task than it would have been previously.

The RNP model presented here used the framework of the SODDr model – modified for a larger spatial area and included host density – to give managers at RNP insight regarding the efficacy of current and proposed SOD treatment scenarios in the specific context of RNP. However, the RNP model had several important assumptions and limitations, and these may be fruitful areas for future model development. For simplicity, parameters for climate or topography were not included in the RNP model. While including these parameters could more accurately represent how the pathogen moves in its environment, it was beyond the scope for this project. Stochasticity was not accounted for in any of the model parameters, however, a 20% increase and decrease of the tanoak mortality parameter was examined and outputs were insignificant. Seasonal variations in the life cycle of *P. ramorum* were not accounted for, nor did I include a scenario which included additional (i.e., annual, biennial) treatments. Conceivably, managers could slow the spread by monitoring and treating new areas annually. The predicted spread of *P. ramorum* would have decreased if I had accounted for repeated treatments in my models, however, continuing to treat new areas every year is an arguably unrealistic approach for managers over the long run.

The current use of buffer treatment protocols are controversial from two perspectives – removal of too many healthy and disease-free tanoak and bay trees that may never become diseased, thus reducing two hardwood species while minimizing the chances of discovering natural resistance; or not removing enough host species around an infestation zone to effectively corral and slow the spread of the disease. In some cases, removal of hosts from forests may be desired to create tanoak refugia or to slow the spread across property boundaries. In these situations, managers may choose to adopt practices such as thinning or injecting chemical protection to make stands more resistant to an inevitable SOD infection (Filipe et al. 2012, Cobb et al. 2013). These methods serve to reduce pathogen spread and minimize the loss of a significant hardwood tree, delaying wide scale loss of this tree while efforts are underway to identify disease resistant trees or new technologies to control the spread of the pathogen. Currently, however, the limited phenotypic and genetic structure of tanoak indicate insufficient evidence of disease resistance (Hayden et al. 2011).

Unfortunately, my modeling efforts, as well as other recent SOD modeling efforts, suggest that host-free barriers of plausible width are ineffective at containing long-distance dispersal. Significantly wider barriers (10 km, suggested by Filipe et al. 2012) can delay the epidemic front, but are unreasonable to apply due to cost effort, and controversy. Pathologists and resource managers have long understood that eradication of a new pathogen is practical only in the early stages of a given outbreak (Alexander & Lee 2010). However, for a pathogen like *P. ramorum* that can disperse asymptomatically over long distances, disease identification and control are extremely difficult (Filipe et al.

2012). Cunniffe et al. (2016) introduced a broad framework for quantifying the likelihood of success and risk of failure of management of invading pathogens and found that, despite extensive cryptic infection and frequent long-distance dispersal, eradication of *P. ramorum* in California could have been possible if treatments had been initiated in California by 2002. The financial and labor expenditures would have been very large, but sufficient knowledge of *P. ramorum* epidemiology was available at that time (Cunniffe et al. 2016).

While eradication of *P. ramorum* from western forests does not appear possible, by modeling various treatment buffer scenarios with differing dispersal kernels, while also accounting for host heterogeneity and other host-specific details (for example, tanoak basal sprouting), managers will have a broader understanding of the predicted efficacy of their management efforts. Ultimately, this work will inform and assist land managers making difficult decisions on how best to manage SOD-threatened and SOD-infected forests across heterogeneous landscapes, and contribute to the understanding of the effects of applying different dispersal kernels in epidemiological modeling. Additional work to understand the efficacy of SOD management strategies will require empirical evidence of dispersal distance and frequency of *P. ramorum* spores, including all transmission pathways at play. Further development of models that incorporate local, community, and landscape factors are needed not only to predict the extent of ecological, cultural, and economic losses expected by the invasion of *P. ramorum*, but also to better identify appropriate locations for future tanoak conservation efforts (Cobb et al. 2013).

References

Alexander, J. & Lee, C. A. (2010). Lessons learned from a decade of sudden oak death in California: evaluating local management. *Environmental Management, 46*(3), 315-328. doi:10.1007/s00267-010-9512-4

Arneson, P. (2000). Coffee rust. *The Plant Health Instructor*. doi:10.1094/phi-i-2000-0718-02

Aukema, J. E., McCullough, D. G., Von Holle, B., Liebhold, A. M., Britton, K., & Frankel, S. J. (2010). Historical accumulation of nonindigenous forest pests in the Continental United States. *Bioscience*, *60*(11), 886-897.

Anderson, R. M., & May, R. M. (1985). Vaccination and herd immunity to infectious diseases. *Nature*, *318*(6044), 323–329. doi:10.1038/318323a0

Bowcutt, F. (2011). Tanoak target: the rise and fall of herbicide use on a common native tree. *Environmental History*, *16*(2), 197–225. doi:10.1093/envhis/emr032

Brasier, C. M. (1991). *Ophiostoma novo-ulmi* sp. nov., causative agent of current Dutch elm disease pandemics. *Mycopathologia*, *115*(3), 151–161. doi:10.1007/BF00462219

Byers, J. E., Reichard, S., Randall, J. M., Parker, I. M., Smith, C. S., Lonsdale, W. M., Atkinson, I. A. E., Seastedt, T. R., Williamson, M., Chornesky, E. & Hayes, D. (2002). Directing research to reduce the impacts of nonindigenous species. *Conservation Biology, 16*(3), 630–640. doi:10.1046/j.1523-1739.2002.01057.

Carrasco, L. R., Baker, R., MacLeod, A., Knight, J. D. & Mumford, J. D. (2010). Optimal and robust control of invasive alien species spreading in homogeneous landscapes. *J. R. Soc. Interface, 7*, 529-540. doi:10.1098/rsif.2009.0266.

Clark, J. S. (1998). Why trees migrate so fast: confronting theory with dispersal biology and the paleorecord. *The American Naturalist*, *152*(2), 204-224.

Cobb, R. C., Filipe, J. A. N., Meentemeyer, R. K., Gilligan, C. A., & Rizzo, D. M. (2012). Ecosystem transformation by emerging infectious disease: loss of large tanoak from California forests. *Journal of Ecology*, *100*(3), 712–722. doi:10.1111/j.1365-2745.2012.01960.x

Cobb, R. C., Rizzo, D. M., Hayden, K. J., Garbelotto, M., Filipe, J. A. N., Gilligan, C. A., Dillon, W.W., Meentemeyer, R.K., Valachovic, Y.S., Goheen, E., Swiecki, T.J., Hansen, E.M., Frankel, S. J. (2013). Biodiversity conservation in the face of

dramatic forest disease: an integrated conservation strategy for tanoak (*Notholithocarpus densiflorus*) threatened by Sudden Oak Death. *Madroño*, *60*(2), 151–164. doi:10.3120/0024-9637-60.2.151

Cunniffe, N. J., Cobb, R. C., Meentemeyer, R. K., Rizzo, D. M., & Gilligan, C. A. (2016). Modeling when, where, and how to manage a forest epidemic, motivated by Sudden Oak Death in California. *Proceedings of the National Academy of Sciences*, *113*(20), 5640–5645. doi:10.1073/pnas.1602153113

Davidson, J. M., Wickland, A. C., Patterson, H. A., Falk, K. R., & Rizzo, D. M. (2005). Transmission of *Phytophthora ramorum* in mixed-evergreen forest in California. *Phytopathology*, *95*(5), 587–596. doi:10.1094/PHYTO-95-0587

Elliott, K. J., & Swank, W. T. (2008). Long-term changes in forest composition and diversity following early logging (1919–1923) and the decline of American chestnut (*Castanea dentata*). *Plant Ecology*, *197*(2), 155–172. doi:10.1007/s11258-007-9352-3

Filipe, J. A. N., Cobb, R. C., Meentemeyer, R. K., Lee, C. A., Valachovic, Y. S., Cook, A. R., Rizzo, D.M., Gilligan, C. A. (2012). Landscape epidemiology and control of pathogens with cryptic and long-distance dispersal: Sudden Oak Death in northern Californian forests. *PLoS Computational Biology*, *8*(1). doi:10.1371/journal.pcbi.1002328

Frankel, S. J. (2008). Sudden oak death and *Phytophthora ramorum* in the USA: a management challenge. *Australasian Plant Pathology*, *37*(1), 19. doi: 10.1071/AP07088

Freinkel, S. (2007). *American Chestnut: The Life, Death, and Rebirth of a Perfect Tree*. Berkeley: University of California Press.

García, C. & Borda-de-Água, L. (2016). Extended dispersal kernels in a changing world: insights from statistics of extremes. *Journal of Ecology*, *105*, 63–74. doi:10.1111/1365-2745.12685

Garnier, J. (2011). Accelerating solutions in integro-differential equations. *SIAM J Math Anal 43*, 1955–1974.

Goheen, E. M., Hansen, E. M., Kanaskie, A., McWilliams, M. G., Osterbauer, N., & Sutton, W. (2002). Sudden Oak Death caused by *Phytophthora ramorum* in Oregon. *Plant Disease*, *86*(4), 441–441. doi:10.1094/PDIS.2002.86.4.441C

Goheen, E. M., Hansen E. M., Kanaskie, A., McWilliams, M. G., Osterbauer, N., Sutton, W., & Rehms, L. (2004). An eradication strategy for *Phytophthora ramorum* in Oregon coastal forests. *Phytopathology 94*(S35).

Grünwald, N. J., Garbelotto, M., Goss, E. M., Heungens, K. & Prospero, S. (2012). Emergence of the sudden oak death pathogen *Phytophthora ramorum*. *Trends in Microbiology*, *20*(*3*), 131–138. doi: 10.1016/j.tim.2011.12.006

Grünwald, N. J., Goss, E. M., & Press, C. M. (2008). *Phytophthora ramorum*: a pathogen with a remarkably wide host-range causing sudden oak death on oaks and ramorum blight on woody ornamentals. *Mol. Plant Pathol. 9*, 729–740. doi:10.1111/J.1364-3703.2008.00500.X

Hansen, E. M., Goheen, D. J., Jules, E. S., & Ullian, B. (2000). Managing Port Orford cedar and the introduced pathogen, *Phytophthora lateralis*. *Plant Disease 84*, 4-10.

Hansen, E. M., Kanaskie, A., Prospero, S., McWilliams, M., Goheen, E. M., Osterbauer, N., Reeser, P., & Sutton, W. (2008). Epidemiology of *Phytophthora ramorum* in Oregon tanoak forests. *Canadian Journal of Forest Research, 38*(5), 1133–1143. doi:10.1139/X07-217

Haas, S. E., Cushman, J. H., Dillon, W. W., Rank, N. E., Rizzo, D. M., & Meentemeyer, R. K. (2016). Effects of individual, community, and landscape drivers on the dynamics of a wildland forest epidemic. *Ecology, 97*: 649–660. doi:10.1890/15-0767.1

Hastings, A. (1996). Models of spatial spread: is the theory complete? *Ecology*, *77*(6), 1675–1679. doi:10.2307/2265772

Hastings, A., Cuddington, K., Davies, K. F., Dugaw, C. J., Elmendorf, S., Freestone, A., Harrison, S., Holland, M., Lambrinos, J., Malvadkar, U., Melbourne, B. A., Moore, K., Taylor, C. and Thomson, D. (2004). The spatial spread of invasions: new developments in theory and evidence. *Ecology Letters, 8*, 91–101. doi:10.1111/j.1461-0248.2004.00687.x

Hastings, A., Hall, R. J., & Taylor, C. M. (2006). A simple approach to optimal control of invasive species. *Theoretical Population Biology, 70*(4): 431-35.

Hayden, K. J., Nettel, A., Dodd, R. S., Garbelotto, M. (2011). Will all the trees fall? Variable resistance to an introduced forest disease in a highly susceptible host. *Forest Ecology and Management 261*(11): 1781-1791. doi:10.1016/j.foreco.2011.01.042

Higgins, S. I., & Richardson, D. M. (1996). A review of models of alien plant spread. *Ecological Modelling*, *87*(1–3), 249–265. doi:10.1016/0304-3800(95)00022-4

Judelson, H. S. & Blanco, F. A. (2005). The Spores of *Phytophthora*: weapons of the plant destroyer. Microbiology. *Nature Reviews, 3*(1): 47-58.

Jules, E.S., Carroll, A.L., Garcia, A.M., Steenbock, C.M., & Kauffman, M.J. (2014). Host heterogeneity influences the impact of a non-native disease invasion on populations of a foundation tree species. *Ecosphere, 5*(9), 105. doi:10.1890/ES14-00043.1

Jules, E. S., Kauffman, M. J., Ritts, W. D., & Carroll, A. L. (2002). Spread of an invasive pathogen over a variable landscape: a nonnative root rot on Port Orford cedar. *Ecology*, *83*(11), 3167–3181. doi:10.1890/0012-9658(2002)083[3167:SOAIPO]2.0.CO;2

Kelly, R., Lundy, M. G., Mineur, F., Harrod, C., Maggs, C. A., Humphries, N. E., Sims, D. W. & Reid, N. (2014). Historical data reveal power-law dispersal patterns of invasive aquatic species. *Ecography*, *37*(6), 581–590. doi:10.1111/j.1600-0587.2013.00296.x

Kinloch, B. B. (2003). White pine blister rust in North America: past and prognosis. *Phytopathology*, *93*(8), 1044–1047. doi:10.1094/PHYTO.2003.93.8.1044

Kot, M., Lewis, M. A., & van den Driessche, P. (1996). Dispersal data and the spread of invading organisms. *Ecology*, *77*(7), 2027–2042. doi:10.2307/2265698

Kuljian, H., & Varner, J. M. (2010). The effects of sudden oak death on foliar moisture content and crown fire potential in tanoak. *Forest Ecology and Management*, *259*(10), 2103–2110. doi: 10.1016/j.foreco.2010.02.022

Liebhold, A. M., Brockerhoff, E. G., Garrett, L. J., Parke, J. L. & Britton, K. O. (2012). Live plant imports: the major pathway for forest insect and pathogen invasions of the US. *Frontiers in Ecology and the Environment 10*, 135–143. doi:10.1890/110198

Madden, L. V., Hughes, G., & Bosch, F. van den. (2007). *Study of plant disease epidemics*. St. Paul, Minneapolis: American Phytopathological Society.

Meentemeyer, R. K., Cunniffe, N. J., Cook, A. R., Filipe, J. A. N., Hunter, R. D., Rizzo, D. M., & Gilligan, C. A. (2011). Epidemiological modeling of invasion in heterogeneous landscapes: spread of Sudden Oak Death in California (1990–2030). *Ecosphere*, *2*(2). doi:10.1890/ES10-00192.1

Meentemeyer, R. K., Rank, N. E., Shoemaker, D. A., Oneal, C. B., Wickland, A. C., Frangioso, K. M., & Rizzo, D. M. (2008). Impact of Sudden Oak Death on tree mortality in the Big Sur ecoregion of California. *Biological Invasions*, *10*(8), 1243–1255. doi:10.1007/s10530-007-9199-5

Meentemeyer, R. K., Rizzo, D. M., Mark, W., & Lotz, E. (2004). Mapping the risk of establishment and spread of Sudden Oak Death in California. *Forest Ecology and Management*, *200*(1–3), 195–214. doi:10.1016/j.foreco.2004.06.021

Metz, M. R., Frangioso, K. M., Wickland, A. C., Meentemeyer, R. K., & Rizzo, D. M. (2012). An emergent disease causes directional changes in forest species composition in coastal California. *Ecosphere*, *3*(10). doi:10.1890/ES12-00107.1

Mbah, M. L., & Gilligan, C. A. (2010). Optimization of control strategies for epidemics in heterogeneous populations with symmetric and asymmetric transmission. *Journal of Theoretical Biology*, *262*(4), 757–763. doi:10.1016/j.jtbi.2009.11.001

McDougall, K. L., Hardy, G. E. St J. & Hobbs, R. J. (2002). Distribution of *Phytophthora cinnamomi* in the northern jarrah (*Eucalyptus marginata*) forest of Western Australia in relation to dieback age and topography. *Australian Journal of Botany*, *50*, 107-114. doi: 10.1071/BT01040

Parke, J. L. & Lucas, S. (2008). Sudden oak death and ramorum blight. *The Plant Health Instructor*. doi: 10.1094/PHI-I-2008-0227-01

Peterson, E., Hansen, E. M., & Hulbert, J. (2014). Source or sink? The role of soil and water borne inoculum in the dispersal of *Phytophthora ramorum* in Oregon tanoak forests. *Forest Ecology and Management*, *322*, 48–57. doi: 10.1016/j.foreco.2014.02.031

Peterson, E. K., Hansen, E. M., & Kanaskie, A. (2015). Temporal epidemiology of Sudden Oak Death in Oregon. *Phytopathology*, *105*(7), 937–946. doi:10.1094/PHYTO-12-14-0348-FI

Purdy, L.H. & Schmidt, R.A (1996). Status of cacao witches' broom: biology, epidemiology, and management. *Annual Review of Phytopathology*, *34.* doi: 10.1146/annurev.phyto.34.1.573

Rizzo, D. M., & Garbelotto, M. (2003). Sudden Oak Death: endangering California and Oregon forest ecosystems. *Frontiers in Ecology and the Environment*, *1*(4), 197–204. doi:10.1890/1540-9295(2003)001[0197:SODECA]2.0.CO;2

Rizzo, D. M., Garbelotto, M., Davidson, J. M., Slaughter, G. W., & Koike, S. T. (2002). *Phytophthora ramorum* as the cause of extensive mortality of *Quercus* spp. and

Lithocarpus densiflorus in California. *Plant Disease, 86*(3), 205–214. doi:10.1094/PDIS.2002.86.3.205

Rizzo, D. M., Garbelotto, M., & Hansen, E. M. (2005). *Phytophthora ramorum*: integrative research and management of an emerging pathogen in California and Oregon forests. *Annual Review of Phytopathology, 43*(1), 309–335. doi: 10.1146/annurev.phyto.42.040803.140418

Rodrigues, L. A. D., Mistro, D. C., Cara, E. R., Petrovskaya, N., & Petrovskii, S. (2015). Patchy invasion of stage-structured alien species with short-distance and long-distance dispersal. *Bulletin of Mathematical Biology*, *77*(8), 1583–1619. doi: 10.1007/s11538-015-0097-1

Stumpf, K. A., Cogan Technology, and Kier Associates. (2017). Vegetation mapping and classification project: Redwood National and State Parks, California. Natural Resource Report NPS/XXXX/NRR—2017/XXX. National Park Service, Fort Collins, Colorado.

Václavík, T., Kanaskie, A., Hansen, E., Ohmann, J. & Meentemeyer, R. (2010). Predicting potential and actual distribution of sudden oak death in Oregon: Prioritizing landscape contexts for early detection and eradication of disease outbreaks. *Forest Ecology And Management, 260*(6), 1026-1035. doi:10.1016/j.foreco.2010.06.026

Valachovic, Y. S., Lee, C. A., Scanlon, H., Varner, J. M., Glebocki, R., Graham, B. D., & Rizzo, D. M. (2011). Sudden Oak Death-caused changes to surface fuel loading and potential fire behavior in Douglas-fir-tanoak forests. *Forest Ecology and Management, 261*(11), 1973–1986. doi:10.1016/j.foreco.2011.02.024

Zadoks, J. C., & Schein, R. D. (1979). Epidemiology and plant disease management. *Epidemiology and plant disease management.*

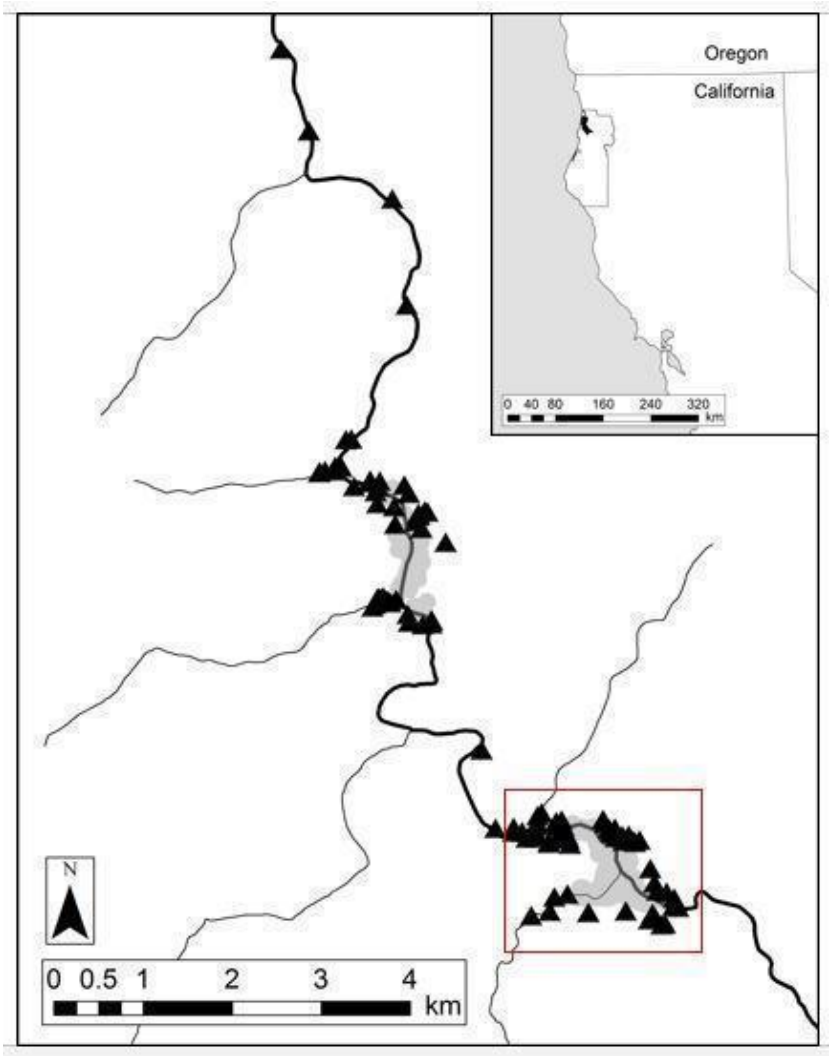

Figure 1. Map of the study area along Redwood Creek in Redwood National Park (USA). Gray areas represent 2014/2015 treatment areas and black triangles represent *Phytophthora ramorum* positive tree locations found in 2016. Bold black line represents Redwood Creek and thin black lines represent major tributaries of Redwood Creek. Red box indicates area included in the epidemiological model. Inset map shows Redwood National Park in Humboldt County, CA, USA.

Table 1. Parameter values for the epidemiological model. Infection and pathogen-caused mortality rates are the weighted average of the reciprocal of the average time to the event (infection or mortality) for multiple size classes from the survival model in Cobb et al. 2012. Sources: (1) Survival model for infection from Cobb et al. 2012; (2) Ndeffo Mbah and Gilligan 2010; (3) Cobb et al. 2010; (4) Survival model for mortality from Cobb et al. 2012; (5) Equations (8-11) from Cobb et al. 2012; (6) Davidson et al. 2011; (7) Chosen so mean dispersal distance was 2.8 cells = 80 m; (8) Chosen so total dispersal over model area = 1.

Parameter	Symbol	Value	Source
Infection rates		(yr^{-1})	
Tanoak to tanoak	β_1	0.31	(1)
Bay laurel to bay laurel	β_2	1.33	(2)
Bay laurel to tanoak	β_{12}	1.46	(3)
Tanoak to bay laurel	β_{21}	0.30	assumed
Tanoak pathogen-caused mortality rate	$\alpha 1$	0.04543(± 20%)	(4)
Tanoak natural mortality rate	d1	0.007354	(5)
Tanoak recruitment rate	b1	0.02017596	(5)
Additional rate parameters		(yr^{-1})	
Tanoak recovery rate	$\mu 1$	0.01	assumed
Bay laurel recovery rate	$\mu 2$	0.1	(6)
Bay laurel rate of recruitment from seed	b2	0.05487072	(5)
Redwood rate of recruitment from seed	b3	0.01371768	(5)
Bay laurel natural mortality rate	d2	0.02	(5)
Redwood natural mortality rate	d3	0.005	(5)
Additional probability parameters		(yr^{-1})	
Proportion of pathogen spores deposited within cells	f_{wth}	0.10	assumed
Proportion of pathogen spores deposited between cells	f_{btw}	0.90	assumed
Tanoak probability of resprouting	r	0.5	(3)
Relative measure of per-capita space used per plot by species s	W_s	1	assumed
Exponential kernel parameters		cells($^{-1}$)	
Lambda	λ	0.07	(7)
Gamma	γ	0.76	(8)
Power-law kernel parameters		cells($^{-1}$)	
Beta	β	0.09	(7)

Parameter	Symbol	Value	Source
Gamma	γ	2.91	(8)

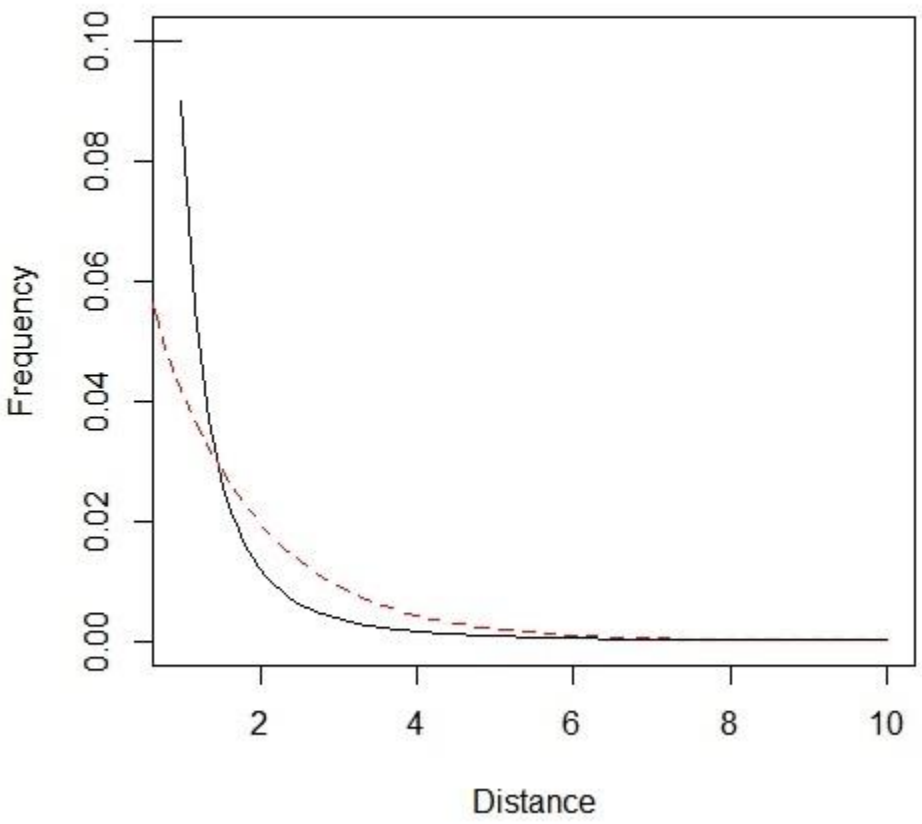

Figure 2. Exponential (black line) and power-law (red dashed line) distributions describing the dispersal of *P. ramorum* spores across the model area (distance shown in units of cells, 1 cell = 28.5 m). Distribution equations are described by Eqn 1 and Eqn 2 and parameter values are listed in Table 1. Values were chosen so the mean dispersal distance was the same (80 m) for both distributions. This value was chosen so that spores disperse beyond the nearest-neighbor in the grid while not spreading so far that the distribution becomes uniform. Both distributions allow most dispersal to occur locally but also account for long distance dispersal. The power-law (red line) distribution has a slightly fatter tail than the exponential distribution, however this is not readily apparent in the figure.

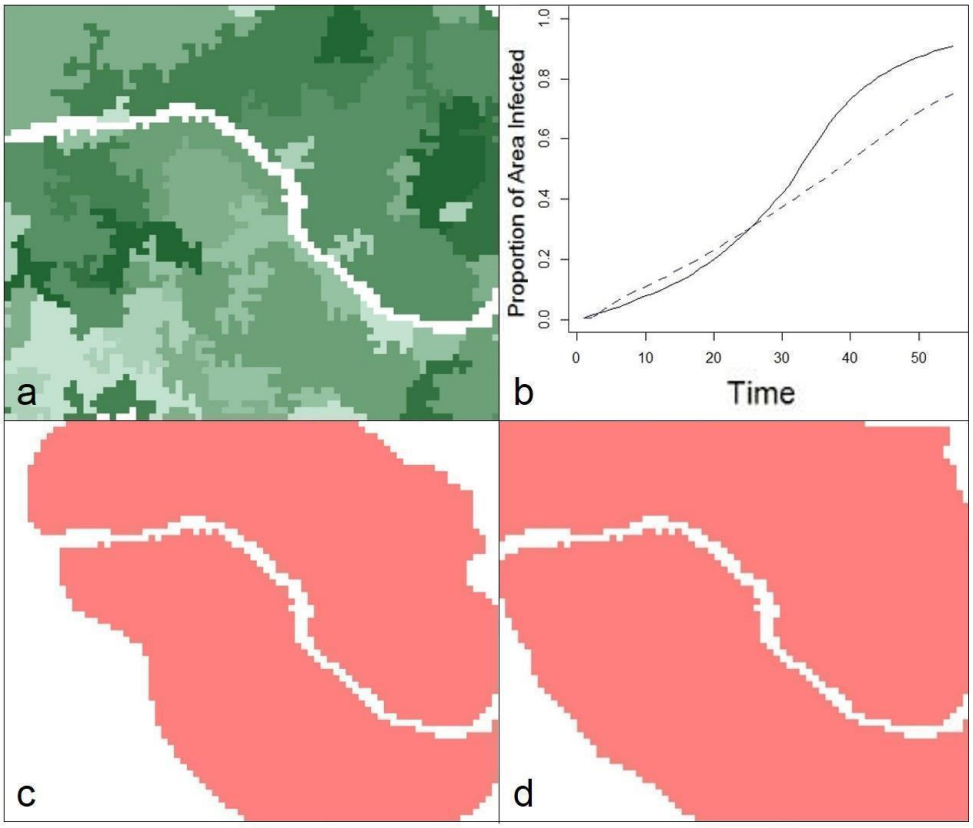

Figure 3. Control model figure. a) 380 ha model area at Bridge Creek in Redwood National Park (RNP), California, USA, showing tanoak (*Notholithocarpus densiflorus*) density prior to SOD infection and treatment. Color gradient darkens with increasing absolute host cover, ranging from 0 (white) to 117 (darkest green). Redwood Creek is shown in white through the middle of the figure. b) predicted proportion of model area infected after 55 years without any management action in RNP using exponential (dashed-line) and power-law (solid line) dispersal kernel distributions. c) spread, defined as cells with at least 1% infected tanoak during the 55 year model run, using an exponential dispersal kernel distribution. d) spread, defined as cells with at least 1% infected tanoak during the 55 year model run, using a power-law dispersal kernel distribution.

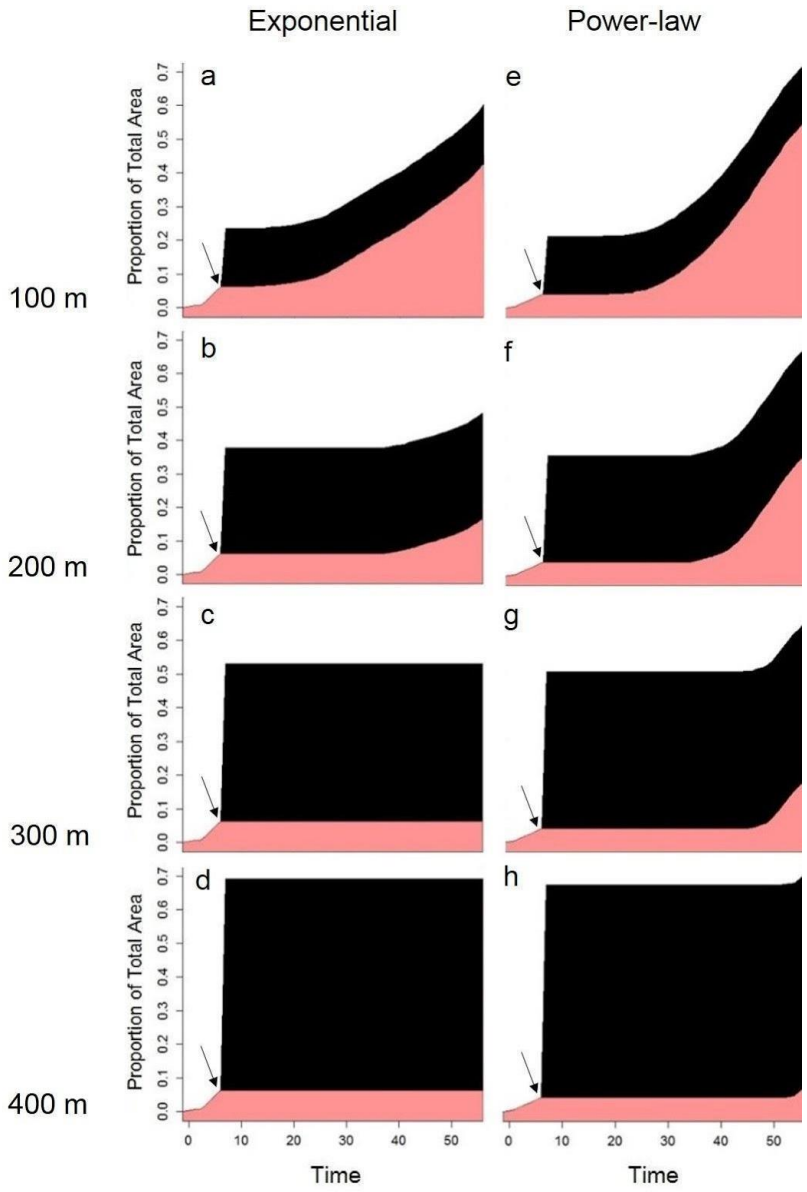

Figure 4. SOD spread over time (shown in pink) and the total area impacted by implementing treatment buffers (shown in black) for the 380 ha model area at Bridge Creek in Redwood National Park, California, USA. Black arrows indicate when treatment was initiated (I assumed this was 5 years after pathogen introduction). Treatment consists of injecting herbicide into tanoak (*Notholithocarpus densiflorus*) and bay (*Umbellularia californica*) hosts. A 100 m buffer increases the area impacted by 20% (76 ha), without stopping spread from escaping the buffer (a, e). Spread was predicted to cover approximately 35% (133 ha; power-law) and 15% (57 ha; exponential) of the model area with a 200 m (122

ha) buffer. Considering the removal of hosts in the buffered area, the total area impacted increases to approximately 67% (255 ha) and 47% (179 ha) of the model area in these scenarios, and does not contain the pathogen (b, f). The exponential dispersal kernel in the 300 m and 400 m buffer scenarios predicts pathogen containment (c, d). However, the pathogen was predicted to spread beyond both the 300 and 400 m buffers when using the power-law dispersal kernel (g, h). In these scenarios, *P. ramorum* was predicted to infect approximately 17% (65 ha) and 7% (27 ha) of the model area, respectively. However, the 300 m and 400 m buffers themselves take up approximately 50% (190 ha) and 65% (247 ha) of the entire model area. While large buffers do not eradicate the pathogen over time, treatment would be expected to reduce inoculum by removing hosts, effectively slowing the spread when compared to control (no treatment) scenarios.

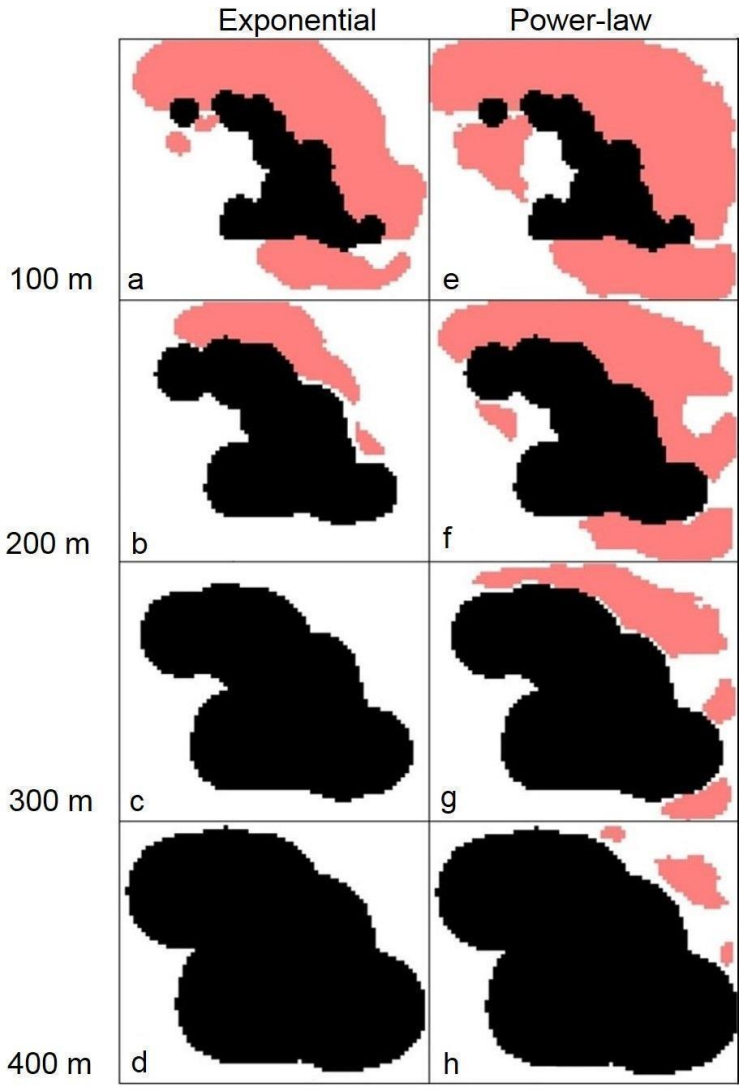

Figure 5. Variable buffer treatment model figure for the 380 ha model area at Bridge Creek in Redwood National Park, California, USA under various buffer management scenarios. Panel shows spread (defined as cells that had 1% or greater infected tanoak) in pink over 55 years using an exponential dispersal kernel distribution on the left panel (a, b, c, d) and power-law dispersal kernel distribution on the right panel (e, f, g, h). Rows show spread 50 years post-treatment with a 100 m buffer (a, e), 200 m buffer (b, f), 300 m buffer (c, g), and 400 m buffer (d, h). Spread is predicted to be contained in the model with 300 m buffers with the exponential kernel, but not with a 300 m or 400 m buffer with the power-law kernel. Host removal does not eliminate spread due to the 5 years of

uncontrolled disease spread before treatment and also because of an assumed 99% host removal efficiency rate.

CHAPTER 2: SPATIAL VEGETATION MODELING SUGGESTS A NOVEL TRANSMISSION PATHWAY OF THE INVASIVE FOREST PATHOGEN, *PHYTOPHTHORA RAMORUM*

Introduction

Phytophthora ramorum, the pathogen that causes Sudden Oak Death (SOD), has caused extensive mortality of oak (*Quercus* spp.) and tanoak (*Notholithocarpus densiflorus*) trees in California and Oregon since the 1990s (Rizzo & Garbelotto 2003). The pathogen has caused significant ecological changes to forests across large areas of California and Oregon; the death of tanoak has changed forest composition, reduced a key food source for wildlife (acorns), and increased the potential for extreme fire behavior in infected forests (Valachovic et al. 2011, Metz et al. 2012). Attributable to favorable climate and high host densities, an alarming number of models suggest uninfected forest ecosystems in northern California and Oregon are at high risk of *P. ramorum* infection (Meentemeyer et al. 2004, Meentemeyer et al. 2011, Filipe et al. 2012). Transportation of *P. ramorum* inoculum is thought to typically occur via rain splash, stream and river currents, wind and mist, and human-mediated transport (Davidson et al. 2005, Filipe et al. 2012). To date, no study has suggested soil or stream borne inoculum to be important in the primary establishment of *P. ramorum* in new wildland areas (Rizzo et al. 2005, Parke & Lucas 2008, Peterson et al. 2014). Yet, *P. ramorum* can be recovered from waterways downstream of areas of known infection by deploying host leaves as bait for SOD spores in water, a common monitoring technique called “stream baiting” (Davidson et al. 2005, Hansen 2008, Peterson et al. 2014). It has

been speculated that terrestrial infections can originate when host leaves are submerged during periods of heavy rainfall, however, evidence has suggested stream-borne inoculum to be an unimportant dispersal pathway in natural ecosystems (Grünwald et al. 2012, Peterson et al. 2014).

A 2010 aerial survey discovered a new *P. ramorum* infection along a Redwood Creek in northern California approximately 8 km upstream of the RNP boundary. The University of California Cooperative Extension (UCCE) quickly developed and implemented an eradication program at the site of the infestation; however, the pathogen continued to spread. In July 2014, the first detections of *P. ramorum* were confirmed downstream of the UCCE infection site, within RNP, along the banks of Redwood Creek in two separate locations near the confluences of Bridge and Bond Creeks with Redwood Creek. *P. ramorum* was discovered near Bridge Creek during a ground-based survey from the UCCE infection site to the mouth of Redwood Creek, covering the entirety of Redwood Creek within RNP. Fifteen and nineteen tanoaks near Bridge Creek were confirmed to be infected with *P. ramorum* after being plated in PARP (pimaricin-ampicillin-rifampicin-PCNB) agar in RNP and University of California at Davis laboratories in 2014 and 2015, respectively. The Bond Creek infection site was discovered during a 2014 aerial survey of the Redwood Creek corridor. After extensive ground-based monitoring, fourteen and seventeen tanoaks were confirmed positive near Bond Creek in 2014 and 2015, respectively, using the same methodology as described above. These locations were separated by approximately 3 km and were approximately 15 km from the infection discovered outside the RNP boundary in 2010 (Figure 1). In

2014 and 2015, six tributaries of Redwood Creek were stream-baited up and downstream of the confirmed *P. ramorum* infections at Bridge and Bond Creeks and yielded negative results each time. Typical treatment protocols used in Oregon and other regions of California were applied to both infections in 2014 and 2015, which consisted of either cutting or injecting all hosts with herbicide within 100 m of infected trees (Frankel 2008, Peterson et al. 2015).

Reported Information

Since the initial discovery of SOD in RNP in 2014, a conspicuous pattern of spread has been observed that strongly indicates stream-to-land spread of the *P. ramorum*. Diseased tanoaks have been primarily located either within 100 m of the previous year's treatment boundary, along tributaries of Redwood Creek near known infections, or downstream along Redwood Creek. Localized, terrestrial spread is assumed to account for the newly infected trees near the previous treatment zone and along nearby tributaries, however, the pattern of long distance spread along Redwood Creek indicates a different means of transmission. For example, in 2015, spread was observed along Redwood Creek over 800 m downstream of the 2014 Bridge Creek treatment zone (Figure 1). The same trend was observed in 2016, with distances between infected, creekside tanoaks reaching over 1.5 km downstream in several locations (Figure 1). In 2016, ground-based monitoring efforts were concentrated around the previous year's treatment zones, along Redwood Creek, and up tributaries of Redwood Creek until symptoms were no longer observable.

While topography and spring wind currents likely favor downstream spread (and prevailing winds likely favor upstream spread), aerial long-distance spread would not be expected to infect only creekside hosts. Furthermore, all of the originally infected creekside trees from 2014-2016 were tanoak and many of these trees had low-hanging branches which extended over the creek channel (Figure 6). During stream monitoring efforts in 2014, dried clumps of debris caught between twigs were frequently observed, indicating submersion during winter months. Blackened cankers (a common symptom of SOD and other *Phytophthora* species), were revealed on the twigs on multiple occasions when the debris was removed (Figure 6). These twigs were later confirmed positive for *P. ramorum* after being plated in PARP agar in RNP and University of California at Davis laboratories. It has been previously assumed that high winter flows in the creek channel were too vigorous to allow for transmission of SOD. However, debris in the low-hanging branches may act as a buffer between the flowing water and the susceptible host, making transmission possible.

To further evaluate the possibility of stream-to-land spread I implemented an epidemiological model to compare the viability of two hypotheses: SOD was introduced to RNP via a rare long-distance dispersal event that originally infected a single host tree (i.e., no stream transmission), or SOD was spread to the region via Redwood Creek and multiple creekside tanoaks were originally infected (i.e., stream transmission). I modified a spatially-explicit, density-dependent, stage-structured epidemiological model created by Cobb et al. (2012; packaged into R under the title “SODDr” [Sudden Oak Death Dynamics in R]). The model simulates the terrestrial spread of disease and resulting tree

mortality and stand dynamics in a mixed system of tanoak, bay (*Umbellularia californica*), and redwood (*Sequoia sempervirens*), and outputs a matrix of population densities by species, infection status, and location. I did not model stage-structure of the hosts, but I included an exponential dispersal kernel for the pathogen (rather than nearest neighbor dispersal). The RNP model was parameterized as in Cobb et al. (2012), except for parameters that depended on host stage. For these parameters I computed a weighted average of the stage-based parameters from Cobb et al. (2012) using the disease-free stage distribution predicted by the original model. To increase the accuracy of the model, I included site-specific host heterogeneity data obtained from a vegetation classification and land cover map of RNP that was created as a part of the National Park Service Vegetation Mapping Inventory (NVMI) (Stumpf et al. 2017 *in review*) (see: Table 1 for parameter values and Appendix A for complete model description).

The same model was used to test the two hypotheses; the only difference was how the infection was initialized. If the infections in RNP are the result of a rare, long-distance, aerial dispersal event and not the result of stream-to-land transmission, one would expect that a single, original infected host would be responsible for the spread observed. To assess the no stream transmission hypothesis, I initiated the model with each cell that was discovered infected in 2014 and 2015 individually and allowed it to run for 10 years. I then selected the cell that demonstrated the greatest amount of spread. Although Park managers estimate that the terrestrial SOD infections originated 4-6 years ago, I chose 10 years to purposefully overestimate this time. The resulting pattern of spread from a single infected cell was not consistent with the observed spread in the Park

(Figure 7). Next, I assessed the stream transmission hypothesis by initializing every creekside cell that contained an infected tree in 2014 and 2015 (to represent multiple stream-to-land transmission events) and found the pattern of spread was more consistent with field observations (Figure 7).

To compare the efficacy of the two models, I used logistic regression where the response variable was the presence or absence of 2016 field-observed infections for each model cell location in the study area, excluding the regions treated in 2014 and 2015. Explanatory factors included in the regressions were model output for each cell taken from the two model scenarios: stream transmission, and no stream transmission. Four logistic regression models were compared using Akaike Information Criteria (AIC) (Anderson 2008), each model used one of the following explanatory variables shown in Table 2. The logistic regression models using stream transmission output accounted for essentially all the AIC weight in my analysis, providing convincing evidence to support the hypothesis of the presence of stream-to-land transmission over the hypothesis of no stream-to-land transmission. Between the two regression models that used output from the stream transmission scenario, there was considerably more evidence for regression model that used the infected fraction as a predictor than the one that used the infected host density.

Discussion

The extreme distances between infections along Redwood Creek (Figure 1), coupled with the presence of cankers under debris on creekside hosts strongly suggests

that *P. ramorum* is capable of spreading several kilometers downstream of an active infection via stream-to-land transmission. Additionally, the data and models presented here provide strong evidence that stream-to-land transmission of SOD played a significant role in the spread of the pathogen in RNP. It now seems clear that *P. ramorum* shares a transmission mode (water-borne dispersal) with other members of the genus *Phytophthora*, including the important forest pathogens *P. lateralis* and *P. cinnamomi* (Erwin et al. 1983, Jules et al. 2002, 2014). Land managers have previously considered the possibility of stream mediated spread negligible, and this work calls attention to the need for a revised SOD monitoring protocol. The focus of monitoring protocols should be expanded from predominantly terrestrial monitoring to include nearby waterways, similar to monitoring for *P. lateralis* (Hansen et al. 2000, Jules et al. 2014). These monitoring efforts will help evaluate whether waterborne dispersal of *P. ramorum* is common throughout California and Oregon, or is restricted to the area studied.

References

Anderson, D. R. (2008). Model Based Inference in the Life Sciences: A Primer on Evidence. (2008). *Springer-Verlag New York.* doi: 10.1007/978-0-387-74075-1

Cobb, R. C., Filipe, J. A. N., Meentemeyer, R. K., Gilligan, C. A., & Rizzo, D. M. (2012). Ecosystem transformation by emerging infectious disease: loss of large tanoak from California forests. *Journal of Ecology*, *100*(3), 712–722. doi:10.1111/j.1365-2745.2012.01960.x

Davidson, J. M., Wickland, A. C., Patterson, H. A., Falk, K. R., & Rizzo, D. M. (2005). Transmission of *Phytophthora ramorum* in mixed-evergreen forest in California. *Phytopathology*, *95*(5), 587–596.

Erwin, D., Bartnicki-Garcia, S., & Tsao, P. (1983). *Phytophthora*. St. Paul, Minn.: American Phytopathological Society.

Filipe, J. A. N., Cobb, R. C., Meentemeyer, R. K., Lee, C. A., Valachovic, Y. S., Cook, A. R., Rizzo, D.M., Gilligan, C. A. (2012). Landscape epidemiology and control of pathogens with cryptic and long-distance dispersal: Sudden Oak Death in northern Californian forests. *PLoS Computational Biology*, *8*(1). doi:10.1371/journal.pcbi.1002328

Frankel, S. J. (2008). Sudden oak death and *Phytophthora ramorum* in the USA: a management challenge. *Australasian Plant Pathology*, *37*(1), 19. doi: 10.1071/AP07088

Grünwald, N. J., Garbelotto, M., Goss, E. M., Heungens, K. & Prospero, S. (2012). Emergence of the sudden oak death pathogen *Phytophthora ramorum*. *Trends in Microbiology*, *20*(*3*), 131–138. doi: 10.1016/j.tim.2011.12.006

Hansen, E. M. (2008). Alien forest pathogens: Phytophthora species are changing world forests. *Boreal Environmental Research*, *13*, 33–41.

Hansen, E.M., Goheen, D.J., Jules, E.S., & Ullian, B. (2000). Managing Port Orford cedar and the introduced pathogen, *Phytophthora lateralis. Plant Disease, 84*, 4-10.

Jules, E. S., Kauffman, M. J., Ritts, W. D. & Carroll, A.L. (2002). Spread of an invasive pathogen over a variable landscape: a nonnative root rot on Port Orford cedar. *Ecology, 83*, 3167–3181.

Jules, E.S., Carroll, A. L., Garcia, A. M., Steenbock, C. M., & Kauffman, M. J. (2014). Host heterogeneity influences the impact of a non-native disease invasion on populations of a foundation tree species. *Ecosphere, 5*(9):105. doi: 10.1890/ES14-00043.1

Meentemeyer, R. K., Rizzo, D.M., Mark, W., & Lotz, E. (2004). Mapping the risk of establishment and spread of sudden oak death in California. *Forest Ecology and Management, 200*(1–3), 195–214. doi: 10.1016/j.foreco.2004.06.021

Meentemeyer, R. K., Cunniffe, N. J., Cook, A. R., Filipe, J. A. N., Hunter, R. D., Rizzo, D. M., & Gilligan, C. A. (2011). Epidemiological modeling of invasion in heterogeneous landscapes: spread of Sudden Oak Death in California (1990–2030). *Ecosphere, 2*(2). doi:10.1890/ES10-00192.1

Metz, M. R., Frangioso, K. M., Wickland, A. C., Meentemeyer, R. K., & Rizzo, D. M. (2012). An emergent disease causes directional changes in forest species composition in coastal California. *Ecosphere, 3*(10). doi:10.1890/ES12-00107.1

Parke, J. L. & Lucas, S. (2008). Sudden oak death and ramorum blight. *The Plant Health Instructor*. doi: 10.1094/PHI-I-2008-0227-01

Peterson, E., Hansen, E. M., & Hulbert, J. (2014). Source or sink? The role of soil and water borne inoculum in the dispersal of *Phytophthora ramorum* in Oregon tanoak forests. *Forest Ecology and Management, 322*, 48–57. doi: 10.1016/j.foreco.2014.02.031

Peterson, E. K., Hansen, E. M., & Kanaskie, A. (2015). Temporal epidemiology of sudden oak death in Oregon. *Phytopathology, 105*(7), 937–946. doi: 10.1094/PHYTO-12-14-0348-FI

Rizzo, D. M., & Garbelotto, M. (2003). Sudden oak death: endangering California and Oregon forest ecosystems. *Frontiers in Ecology and the Environment, 1*(4), 197–204. doi: 10.1890/1540-9295(2003)001[0197:SODECA]2.0.CO;2

Rizzo, D. M., Garbelotto, M., & Hansen, E. M. (2005). *Phytophthora ramorum*: integrative research and management of an emerging pathogen in California and Oregon forests. *Annual Review of Phytopathology, 43*(1), 309–335. doi: 10.1146/annurev.phyto.42.040803.140418

Stumpf, K. A., Cogan Technology, and Kier Associates. (2017). Vegetation mapping and classification project: Redwood National and State Parks, California. Natural Resource Report NPS/XXXX/NRR—2017/XXX. National Park Service, Fort Collins, Colorado.

Valachovic, Y. S., Lee, C. A., Scanlon, H., Varner, J. M., Glebocki, R., Graham, B. D., & Rizzo, D. M. (2011). Sudden Oak Death-caused changes to surface fuel loading and potential fire behavior in Douglas-fir-tanoak forests. *Forest Ecology and Management, 261*(11), 1973–1986. doi:10.1016/j.foreco.2011.02.024

Figure 6. A) Infected creekside tanoak (*Notholithocarpus densiflorus*) with low hanging branches over the infected Redwood Creek during low flow season. B) Clump of debris caught on creekside tanoak twig indicating submersion during high flow season. C) The same twig as in B but with the debris removed to reveal a blackened canker, a common symptom of Sudden Oak Death. This twig was collected, plated in PARP (pimaricin-ampicillin-rifampicin-PCNB) agar, and confirmed positive for *Phytophthora ramorum* in 2014.

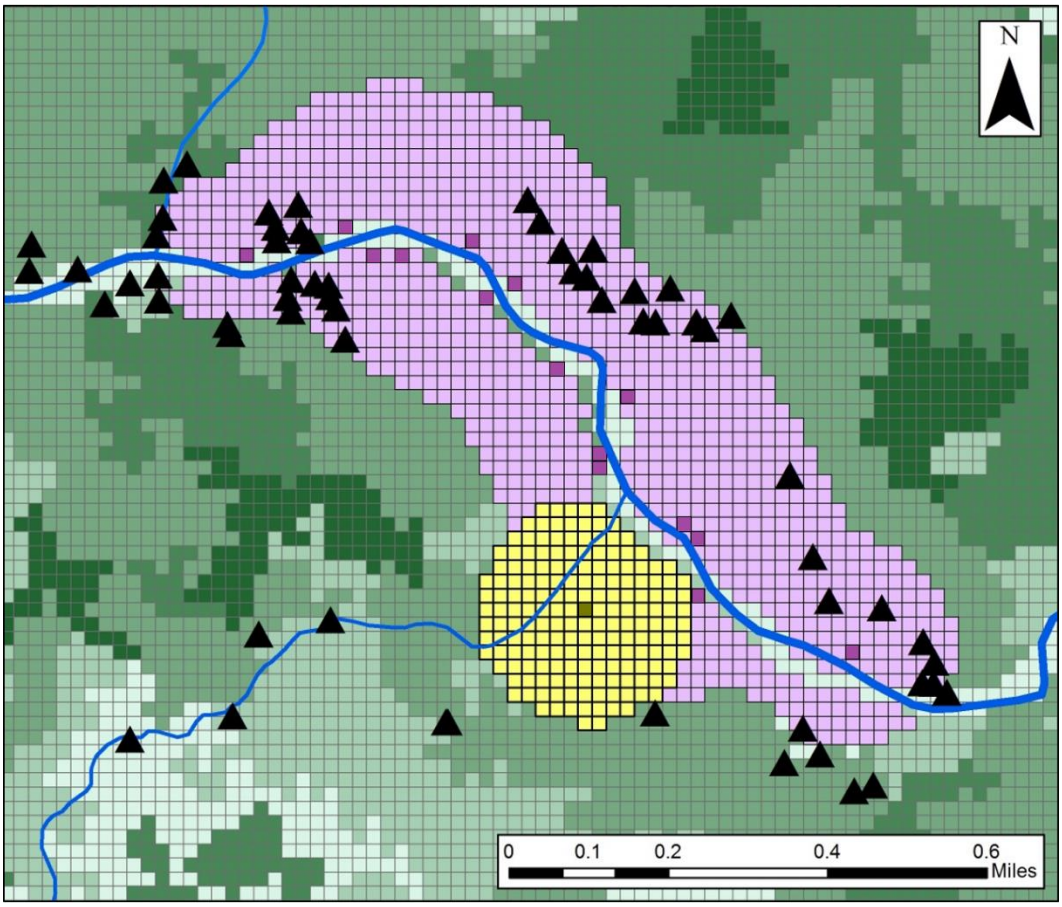

Figure 7. Model output for two hypotheses of Sudden Oak Death (SOD) spread in a 2.1 × 1.8 km area in Redwood National Park. The yellow area shows predicted SOD spread after ten years if the infection had originated at one location only. Each cell that was infected in 2014 and 2015 in the model area was modeled individually as the origin of infection and I chose the cell that exhibited the greatest amount of spread after ten years, as to maximize the potential spread under this hypothesis. The purple area shows predicted SOD spread after ten years if every infected creekside location discovered in 2014 and 2015 was treated as an independent stream-to-land transmission event. Darkened cells indicate infection initializations. Black triangles represent infected trees discovered in 2016. The green-scale cells in the background represent tanoak (*Notholithocarpus densiflorus*) density, with lighter cells containing less hosts and darker cells containing more hosts. See Table 1 for details of models and model parameters.

Table 2. Comparison of four logistic regression models to determine the best predictor of *P. ramorum* infections in 2016 in the study area in Redwood National Park, California, USA. All models had 2 degrees of freedom and were computed using binomial generalized linear models with a logit link in R.

Logistic Regression	deltaAIC	AIC Weight
Stream transmission: Fraction of infected hosts	0	0.824
Stream transmission: Infected host density	3.098	0.175
No stream transmission: Fraction of infected hosts	19.944	< 0.001
No stream transmission: Infected host density	19.972	< 0.001

APPENDIX A: SPATIAL VEGETATION MODELING SUGGESTS A NOVEL TRANSMISSION PATHWAY OF THE INVASIVE FOREST PATHOGEN, *PHYTOPHTHORA RAMORUM* SUPPLEMENTAL INFORMATION

Model Description

The stand-scale epidemiological model created by Cobb et al. (2012; packaged into R under the title "SODDr" [Sudden Oak Death Dynamics in R]) was used as framework and modified for the conditions in RNP. The SODDr model is a spatially explicit, density dependent, stage structured, epidemiological model that was originally used to predict the mortality of tanoak in a 20 ha area in California. The model simulates the spread of disease and resulting tree mortality and stand dynamics in a mixed system of tanoak, bay, and redwood. In this system, only tanoak and bay laurel carry the disease, and it mostly kills tanoak, but all three species compete for space in the forest. The model outputs a large matrix of population densities by species, age class, and location. Subsequent graphical outputs show the disease as it progresses through the stand, with tanoak population as intensity and the fraction of trees diseases as hue. Much of the SODDr model was parameterized using detailed plot-level measurements and performing survival analyses. Spanning over a decade, the depth and quality of this data is exceptional. For this reason, parameter values for infection rates, disease-dependent and independent mortality rates, and recovery from disease were used in the model I created (from hereon referred to as the "RNP" model).

The RNP model is a version of the SODDr model that has been modified for a larger spatial area. I removed the stage-structure component, added host heterogeneity,

and used a distribution kernel rather than a nearest neighbor kernel. As in the SODDr model, the dynamics governing the proportions of each species, in each epidemiological state, and in each cell location were described by a series of differential equations and defined by a series of parameters. The RNP model covers approximately 380 ha of remote, backcountry, redwood/douglas fir/tanoak forest at the confluence of Bridge Creek and Redwood Creek, where SOD was originally discovered in RNP. Using geospatial software, a 380 ha model area was created extending 400 m from the 2014 and 2015 treatment zone at Bridge Creek. The model area was then divided into a lattice of 4,725 812.25 m^2 (28.5 × 28.5 m) cells. The increased temporal scale did not impact the model predictions made by the original SODDr model.

For simplification, parameters for tanoak species were combined into a single size class, as the bay and redwood species' parameters. For this reason, the parameter values representing the transition rates of tanoaks from one size class to the next larger size class were omitted and recruitment rates were calculated for steady-state levels by dividing the mortality rate by the density-dependence coefficient at simulation start (again, the same as bay and redwood). Weighted averages of the borrowed tanoak parameters in differing size classes were used to represent the single size class. For a complete list of parameter values and origins, see Table 1.

A significant addition to the SODDr model was the inclusion of host heterogeneity in the RNP model. To determine host densities, I used RNP's Geographic Resource Solutions (GRS) vegetation data, a vegetation classification and vegetation land cover map of RNP created from remotely sensed and ground-verified data, as a part of

the National Park Service Vegetation Mapping Inventory (NVMI). The map employed the U.S. National Vegetation Classification (USNVC) to map 142,062 acres (57,491 ha) of RNP. Forty-five (45) vegetated map classes at the level of USNVC alliance and four non-vegetated land cover classes were mapped. GRS conducted a thematic accuracy assessment on the 30 most abundant vegetation classes, employing 512 observations throughout RNSP. The overall accuracy at the alliance level vegetation classes was found to be 65.5%. When the classes are aggregated to thematically coarser levels, the accuracy was 86.2% at the USNVC group level and 95.4% at the level of USNVC macrogroup (Stumpf et al. 2017 *in review*). The proportion of tanoak, bay, and redwood was determined in each cell by dividing the alliance-level trees per acre (TPA) of a certain species by the total TPA in the cell, and adjusting to represent trees per cell (TPC). To simplify calculations, the cell size in the RNP model exactly matched the cell size in the GRS data (812.25 m^2).

The transmission of infection within and between cells was determined by the proportion of spores deposited within and between cells, and was calculated in a similar fashion as in the SODDr model. However, dispersal was not limited to adjacent-only cells. We assume spores are capable of spreading beyond the nearest neighbor cell with likelihood decreasing with distance. An exponential dispersal kernel was used to describe pathogen dispersal rates in RNP. The value for λ (0.7) in the equation below was chosen so the mean dispersal distance was 2.8 cells (approximately 80 m). The value for γ was calculated by dividing 1 by the total amount of dispersal outside of the center cell.

Eqn 1: $\gamma \times \frac{1}{(distance)^{\beta}}$

The amount of spread from infected cells to susceptible cells was calculated by first setting the dispersal coefficient for a grid so that the total dispersal outside of infected cell was 0.90 (0.10 falls within center cell, 0.90 is spread out of the cell). Next, we used the reciprocal of the sum of the total amount of dispersal outside of center cell with $\gamma = 1$ and exponential distribution (as a function of distance, model parameters, and λ or β, respectively). As in the SODDr model, the vulnerability of each class of spores originating from others was described by the "Who Acquires Infection From Whom" (WAIFW) matrix (Anderson and May 1985). Additionally, like many hardwood trees, disease-killed tanoak develop basal sprouts from below-ground tissues. This feature was accounted for in both the SODDr and RNP models. When a cell was switched from "susceptible" to "infected", we assume 50% of the hosts in the cell become infected. Due to the fine scale of the model, neither climate data nor long-distance dispersal were included in the SODDr or RNP models.

To examine the sensitivity of the tanoak mortality parameter, the model was run with weighted averages, a 20% reduction of the weighted average parameter, and a 20% increase of the weighted average parameter, and alternating weighted average and increased/decreased parameters. These variations did not produce significant changes in the model so weighted average parameters were used.

References

Anderson, R. M., & May, R. M. (1985). Vaccination and herd immunity to infectious diseases. *Nature, 318*(6044), 323–329. doi: 10.1038/318323a0

Cobb, R. C., Filipe, J. A. N., Meentemeyer, R. K., Gilligan, C. A., & Rizzo, D. M. (2012). Ecosystem transformation by emerging infectious disease: loss of large tanoak from California forests: *Ecosystem transformation by disease*. *Journal of Ecology*, *100*(3), 712–722. doi: 10.1111/j.1365-2745.2012.01960.x

Stumpf, K. A., Cogan Technology, and Kier Associates. (2017). Vegetation mapping and classification project: Redwood National and State Parks, California. Natural Resource Report NPS/XXXX/NRR—2017/XXX. National Park Service, Fort Collins, Colorado.