Biodiversity Conservation in the Face of Dramatic Forest Disease: An Integrated Conservation Strategy for Tanoak (*Notholithocarpus densiflorus*) Threatened by Sudden Oak Death

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BIODIVERSITY CONSERVATION IN THE FACE OF DRAMATIC FOREST DISEASE: AN INTEGRATED CONSERVATION STRATEGY FOR TANOAK (*NOTHOLITHOCARPUS DENSIFLORUS*) THREATENED BY SUDDEN OAK DEATH

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ABSTRACT

Non-native diseases of dominant tree species have diminished North American forest biodiversity, structure, and ecosystem function over the last 150 years. Since the mid-1990s, coastal California forests have suffered extensive decline of the endemic overstory tree tanoak, *Notholithocarpus densiflorus* (Hook. & Arn.) Manos, Cannon & S. H. Oh (Fagaceae), following the emergence of the exotic pathogen *Phythophthora ramorum* and the resulting disease sudden oak death. There are two central challenges to protecting tanoak: 1) the pathogen *P. ramorum* has multiple pathways of spread and is thus very difficult to eradicate, and 2) the low economic valuation of tanoak obscures the cultural and ecological importance of this species. However, both modeling and field studies have shown that pathogen-centric management and host-centric preventative treatments are effective methods to reduce rates of spread, local pathogen prevalence, and to increase protection of individual trees. These management strategies are not mutually exclusive, but we lack precise understanding of the timing and extent to apply each strategy in order to minimize disease and the subsequent accumulation of fuels, loss of obligate flora and fauna, or destruction of culturally important stands. Recent work identifying heritable disease resistance traits, ameliorative treatments that reduce pathogen populations, and silvicultural treatments that shift stand composition hold promise for increasing the resiliency of tanoak populations. We suggest distinct strategies for pathogen invaded and uninvaded areas, place these in the context of local management goals, and suggest a management strategy and associated research priorities to retain the biodiversity and cultural values associated with tanoak.

Key Words: California Floristic Provence, disease ecology, genetic diversity, pathogen-caused extinction, pathogen management, *Phytophthora ramorum*, restoration, tanoak population decline.

To the detriment of cultural, economic, and silvicultural interests, North America has experienced multiple declines of dominant tree species following the introduction and spread of exotic pathogens, many of which are innocuous microbes or weak pathogens in their native ranges (Loo 2009). The quarantine of known pathogens and other techniques to avoid introduction of pathogens into novel environments are important actions to reduce pathogen spread on international and inter-continental scales (Brasier 2008). However, the host range, invasion history, and persistence in the environment of many forest pathogens suggest these invasive organisms have, or will become, naturalized in their new ranges. This challenging natural resource problem demands well-crafted management efforts to avoid the most extensive or severe impacts of forest pathogens.

The pathogen *Phytophthora ramorum* S. Werres, A.W.A.M. de Cock (Werres et al. 2001) exemplifies the destructiveness, epidemiological complexity, and difficulty in managing the most problematic invasive microorganisms (de Castro and Bolker 2005; Smith et al. 2006). *Phytophthora ramorum* was first introduced into California and spread into wildlands via infected nursery stock in Santa Cruz and Marin Counties from a yet unknown native range (Garbelotto and Hayden 2012; Grünwald et al. 2012). The pathogen subsequently spread to Big Sur (Monterey County) and Humboldt County, probably on infected plants, and has since become established in central-California coastal forests. Establishment of *P. ramorum* has been rapid during the ~20 years since its arrival; spread of the pathogen is aerial with extensive local dispersal (20–50 m) and less frequent but consistent long-distance events of 1–3 km (Hansen et al. 2008; Meentemeyer et al. 2008a; Garbelotto and Hayden 2012). *Phytophthora ramorum* causes the disease sudden oak death (Rizzo et al. 2002) which has been responsible for a regional-scale population decline of tanoak, *Notholithocarpus densiflorus* (Hook. & Arn.) Manos, Cannon & S. H. Oh (Fagaceae); formerly *Lithocarpus densiflorus* (Manos et al. 2008), as well as the mortality of hundreds of thousands of oak trees (*Quercus* spp.) from the red oak clade, primarily coast live oak (*Q. agrifolia* Née) throughout the invaded areas of California (Meentemeyer et al 2008b; Davis et al. 2010; Metz et al. 2012).

Phytophthora ramorum has a broad host range, with over 140 known native and non-native plants including many trees, shrubs, forbs, grasses, and ferns common to the California floristic province (Grünwald et al. 2012). In California, sporulation occurs on multiple hosts, the most important of which is the common forest tree California bay laurel (*Umbellularia californica*, Hook. and Arn.). Sporulation also occurs on tanoak twig lesions at levels sufficient to spread the pathogen as well as cause bole-lesions which are the principal infections that lead to mortality in tanoak and oak. In California forests, sporulation can be an order of magnitude greater on bay laurel and peak amounts for both species occur during warm spring rain events (Davidson et al. 2005, 2008; Mascheretti et al. 2008). In California, both tanoak and bay laurel are central to pathogen spread within stands and across landscapes although risk of pathogen establishment and spread rates are more strongly affected by bay laurel density (Meentemeyer et al. 2008a; Davidson et al. 2011). Host impacts are strikingly different across species: *P. ramorum* infection often leads to tanoak mortality, but has no known deleterious impacts on bay laurel at either the individual or the population level (DiLeo et al. 2009; Cobb et al. 2010). In California, the stand-level densities of tanoak and bay laurel greatly influence tanoak mortality rate and population decline (Cobb et al. 2012b). Pathogen dynamics are somewhat different in Oregon forests where sporulation occurs year-round and bay laurel does not play a significant role in spread at the landscape scale (Hansen et al. 2008). However, in both Oregon and California forests, tanoak population decline is expected across much of its endemic range (Fig. 1; Meentemeyer et al. 2004; Václavík et al. 2010; Lamsal et al. 2011; Meentemeyer et al. 2011; Dillon et al. this volume).

Independent studies using repeated measurements of individual tanoak show that mortality rate of infected trees increases with size (McPherson et al. 2010; Cobb et al. 2012b). Although the mechanism for this increase is not known, the pathogen's selective removal of large tanoak leads to rapid, stand-level loss of tanoak biomass in *P. ramorum* invaded stands (Ramage et al. 2011; Cobb et al. 2012b; Metz et al. 2012). Basic biodiversity information such as acorn production rates, knowledge of species obligate to tanoak, and other ecological functions provisioned by mature tanoak trees have not been reported in the peer-reviewed literature. However, ecological functions of individual trees often increase with tree biomass, which scales exponentially with tree size (Lamsal et al. 2011; Cobb et al. 2012a). Selective removal of large trees is likely to accelerate loss of ecosystem characteristics directly associated with tanoak, such as ectomycorrhizal associations or provisioning of habitat for other native flora and fauna which rely on or are obligate to tanoak (Rizzo et al. 2005; Bergemann and Garbelotto 2006; Cobb et al. 2012b; Wright and Dodd this volume).

The distribution of tanoak varies across the landscapes of the California floristic province (Fig. 1), increasing in prevalence with latitude up to the northern range limit of the species in

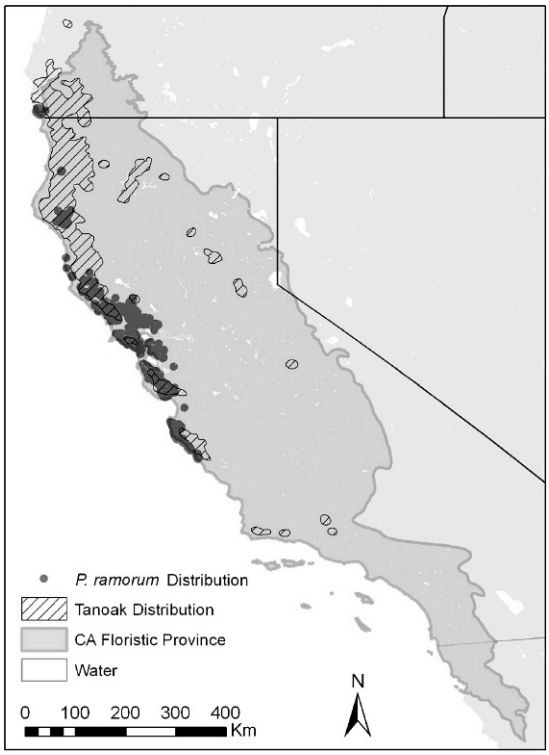

FIG. 1. The California floristic province with approximate extent of *P. ramorum* invasion and tanoak distribution. *Phytophthora ramorum* points are confirmed infected trees; the map was generated with publically available data accessed in January 25, 2013 from oakmapper.org and suddenoakdeath.org.

southwestern Oregon. Several notable disjunct populations are located in the northern Sierra Nevada foothills and in small, isolated populations in Santa Barbara County, CA (Fig. 1; Lamsal et al. 2011; Meentemeyer et al. 2011; Dillon et al. this volume). While *P. ramorum* establishment and disease emergence have been extensive in central coastal California, *P. ramorum* has not yet invaded the majority of the tanoak range (Meentemeyer et al. 2011). However, the pathogen is expected to invade culturally important, highly susceptible North Coast forests over the next several decades reflecting the opportunity and importance of planning and employing a proactive strategy to reduce disease impacts.

Phytophthora ramorum populations in newly invaded areas are often below thresholds where the pathogen can be detected using landscape-scale surveys. These cryptic invasions play an important role in landscape-level spread, and represent a critical challenge to eradication attempts and other pathogen-centric disease management strategies (Filipe et al. 2012). For many invasive pathogens, rapid responses that match the scale of the outbreak increase the likelihood of successful eradication or the efficacy of slowing pathogen spread (Gilligan and van den Bosch 2008; Filipe et al. 2012). This requires rigorous study to identify effective management actions and minimize unintended impacts that are counter to the overall goal. Our intent here is not to review individual management techniques and their efficacy, rather we seek to bring together different management actions and place them in the context of local disease conditions and management goals. We begin by highlighting several historical and contemporary mistakes in forest disease management and emphasize the importance of incorporating historical lessons into sudden oak death management. We suggest how judicious use of established techniques can alter disease conditions and increase tanoak population resiliency across different forest types and community assemblages. We use the term 'resiliency' to mean retention of tanoak and the ecological functions provisioned by this tree and recognize that specific management objectives will vary among ownerships, agencies and situations. Part of our goal is to aid in the selection of when, where, and how to increase tanoak resiliency with existing management actions. We stress that management resources, land-owner cooperation, and chemical treatments will often be insufficient to control pathogen spread and disease emergence. Rather, when management actions aim to retain tanoak as a component of forest structure and composition, we advocate for integration of host-centric actions into management strategies. These strategies rely on increasing tanoak resiliency through stand manipulation to reduce sporulation, increasing distance among tanoak to reduce the probability of transmission, and identification of resistance in tanoak populations. Lastly, we stress that continuous feedback among sudden oak death research, management, and pathogen monitoring is needed to maintain tanoak as a component of biodiversity in many forests and to reduce the spread of *P. ramorum*.

LEARNING FROM FAILURE

The consequences of invasion of *Cryphonectria parasitica* (Murrill) Barr and the resulting disease, chestnut blight, is a notorious example of disease causing North American overstory tree loss (Anagnostakis 1987). Experience with chestnut blight can inform present-day challenges posed by exotic pathogens, especially *P. ramorum*. *Cryphonectria parasitica* is a generalist pathogen, selectively removes American chestnut (*Castanea dentata* Marsh.) from forest stands, and readily disperses across local and regional scales (Loo 2009). Within 30 years of introduction by the pathogen, these characteristics caused a widespread

decline of American chestnut and loss of the considerable ecological, cultural, and economic value that was associated with this species (Anagnostakis 1987; Freinkel 2007). The rate and extent of chestnut decline was unprecedented when the disease emerged circa 1900, and remains as one of the most infamous diseases in North American forest history because of the significant economic and cultural impacts caused by the epidemic (Freinkel 2007).

Chestnut blight is also notable as a case of failed management that stemmed from decisions with incomplete knowledge of pathogen biology and host relationships. For example, the state of Pennsylvania initiated an ambitious plan to remove infected chestnut trees and create a barrier zone with the intention of restricting pathogen spread into the western portion of the state (Freinkel 2007). However, understanding of *C. parasitica* transmission pathways, estimates of pathogen spread rate, and the capacity to detect cryptic infection were all inadequate and pathogen spread was unabated. A consequence of dedicated and extensive chestnut cutting was, at a minimum, an acceleration of chestnut removal from the overstory of these forests, an action that may have even expedited pathogen spread (Freinkel 2007). Today, American chestnut remains common in eastern forests as an understory shrub, but overstory trees and their associated ecological functions such as provisioning of large, nutritious, low-tannin nuts, have been lost from most of the species' former range (Paillet 2002). A full accounting of changes to native flora and fauna is hampered by the lack of natural history investigations of species associated with the tree prior to the epidemic. Although other species replaced American chestnut in the overstory, loss of chestnut probably impacted small mammal and invertebrate populations and may have resulted in the extinction of several insects obligate to this overstory tree (Orwig 2002; Ellison et al. 2005).

Regrettably, chestnut blight is not the sole example of misdirected or ineffective management actions. Eastern hemlock (*Tsuga canadensis* L.) forests are in decline across its native range from Georgia to southern Vermont and New Hampshire due to the invasion and subsequent regional-scale outbreak of hemlock woolly adelgid (*Adelges tsugae* Annand), an exotic insect pest (Fitzpatrick et al. 2012; Orwig et al. 2012). Eastern hemlock decline has been accelerated in southern New England by pre-emptive hemlock harvesting, regardless of stand invasion status (Orwig et al. 2002). From a conservation perspective, indiscriminant host tree removal will accelerate population declines of trees threatened by insect or disease outbreak as well as degrade the genetic diversity of remaining populations (Foster and Orwig 2006; Broadhurst et al. 2008).

Natural resource management relies on adaptive management to address imprecise knowledge and to avoid the field's own version of "the twin traps of overtreatment and therapeutic nihilism" (e.g., the Hippocratic Oath, Edelstein 1943). The backbone of this approach is feedback among researchers, managers, and policy makers who together establish experimental treatments, quantify their efficacy, and direct resources. An analogous framework was not in place to address either chestnut blight or much of the management response to the decline of eastern hemlock populations. In the case of sudden oak death, an adaptive management approach is more likely to identify treatments which judiciously use limited management resources (Filipe et al. 2012) and avoid loss of genetic diversity (Hayden et al. 2011; Kjær et al. 2012) in comparison to reactive actions such as the attempt to construct a barrier to arrest the spread of chestnut blight (Freinkel 2007). The benefits of this approach are already very clear; one of the most important initial breakthroughs regarding sudden oak death was the discovery that *P. ramorum* was the cause of widespread oak and tanoak mortality (Rizzo et al. 2002) which laid to rest spurious hypotheses that the mortality was caused by insects or native pathogens. Adaptive management further relies on clear articulation of management objectives while also recognizing the need to evaluate treatments to achieve these objectives. The extent of the *P. ramorum* invasion suggests management goals must be comprehensive enough to match the magnitude of the disease and recognize technical and practical limitations in order to identify where, when, and in what combination available techniques will be most effective in slowing pathogen spread and maintaining the cultural and ecological value of tanoak.

Tanoak Mortality: Patterns and Impacts

Tanoak has low timber value and the economic value is further lowered because of competition with commercial timber species, i.e., Douglas fir, *Pseudotsuga menziesii* (Mirb.) Franco (Harrington and Tappeinier 2009). Before the 1950s, tanoak-bark extracted tannins were important to California's leather industry, but the advent of synthetic tanning compounds has shifted the economic perspective on this tree (Alexander and Lee 2010; Bowcutt 2011). Prior to the emergence of sudden oak death, most recent research involving tanoak focused on quantifying and reducing competition with coniferous species central to commercial timber production often through the use of herbicides (Harrington and Tappeiner 2009; Bowcutt 2011). Little basic research has been conducted on tanoak and the measurement of growth characteristics, their relationship with disease resistance, and descrip-

tion of flora and fauna obligate to or associated with tanoak have only recently been undertaken (Bergemann and Garbelotto 2006; Cobb et al. 2012b; Wright and Dodd this volume). Although the risk of pathogen-driven, regional-scale removal of tanoak from the forest overstory is becoming increasingly clear, we face a critical lack of understanding as to what and how many species may be threatened by the widespread mortality of this common tree.

Even though *P. ramorum* has not yet invaded most of the tanoak range (Meentemeyer et al. 2011), the pathogen has significantly impacted tanoak from the Big Sur region (Monterey County, CA) north to Sonoma County, CA. In these regions, sudden oak death has caused widespread loss of overstory tanoak trees, significant shifts in overall forest composition, and outright extirpation of tanoak from some stands (Meentemeyer et al. 2008b; Ramage et al. 2011; Cobb et al. 2012b; Metz et al. 2012). These forests occur across topographically, environmentally, and biologically diverse landscapes where several different conifer and broad-leafed trees are likely to experience increases in density, biomass, and dominance as a result of the decline of tanoak populations. Metz et al. (2012) and Cobb et al. (2010) examined sudden oak death-impacted California stands ranging from relatively dry, mixed-evergreen forests to relatively cool and moist redwood-dominated forests; in both studies, tanoak mortality was associated with increased dominance of bay laurel. Given that *P. ramorum* establishment and disease emergence is associated with bay laurel abundance (Maloney et al. 2005; Meentemeyer et al. 2008a; Cobb et al. 2010), bay laurel is often situated to benefit from tanoak population decline. This shift in species composition has the additional impact of improving habitat for *P. ramorum*, suggesting that the pathogen will persist even if tanoak becomes locally extinct (Cobb et al. 2012b).

Additional shifts in species composition due to sudden oak death are also likely in North Coast forests from Mendocino County to Curry County, OR. While the current distribution and prevalence of *P. ramorum* in the North Coast is lower compared to the central coast where the pathogen was initially introduced (Fig. 1), pathogen invasion and associated tanoak mortality have mobilized management actions by state, private, and federal land managers (Rizzo et al. 2005; Valachovic et al. 2008; Goheen et al. 2009). In North Coast forests, Douglas-fir and redwood (*Sequoia sempervirens* [Lamb. ex D. Don] Endl.) have greater importance in terms of density, biomass, and basis in local economies; these characteristics are likely to favor an increase in dominance by these species in areas following tanoak mortality. Notably, forests in Del Norte County, CA and Curry County, OR are notable for their lower abundance of bay laurel especially compared to forests in the central coast, especially Big Sur (Monterey County) and Sonoma County (Lamsal et al. 2011). *Phytophthora ramorum* is likely to invade and cause disease even when tanoak is the sole sporulation-supporting species (Rizzo et al. 2005; Goheen et al. 2009; Meentemeyer et al. 2011) although mortality rates are likely to eventually slow because of a negative feedback on pathogen populations when hosts die (Cobb et al. 2012b). Additionally, pathogen-killed trees or cut tanoak often develop prolific basal sprouts (Harrington and Tappeiner 2009; Cobb et al. 2010; Ramage et al. 2011), which may be sufficient to perpetuate pathogen populations and maintain spread in stands that do not include bay laurel (Cobb et al. 2012b).

Sudden oak death also has far-reaching economic, cultural, and ecological impacts. Spread of *P. ramorum* via ornamental nursery plants was responsible for the initial introduction to California and continues to play a role in spread within the United States and internationally (Garbelotto and Hayden 2012; Grünwald et al. 2012). Regulation of nursery stock, a costly but necessary management action, has been initiated to address this pathway of pathogen spread (see also Alexander and Lee 2010). For individual property owners, the loss of oak and tanoak directly impacts property values (Kovacs et al. 2011), and increased disease-caused fuel loadings at wildland-urban interfaces could augment the risk of property loss during wildfire (Metz et al. 2011; Valachovic et al. 2011). Similarly to American chestnut, tanoak has historically been an important component of human nutrition and local culture; tanoak resources retain significant cultural importance to many Native American communities in California (Bowcutt 2011). From a functional ecology perspective, tanoak is the sole ectomycorrhizal host in many forests (Rizzo et al. 2005) and supports ectomycorrhizal diversity on par with *Quercus* species (Bergemann and Garbelotto 2006). Tanoak-wildlife associations are poorly described, but the large nutritious acorns are likely used by many species and a recently described insect pollination pathway suggests yet undocumented insect communities and ecological interactions that may be impacted by the loss of tanoak (Wright and Dodd this volume).

Disease Treatments: Models and Experiments

A central problem of managing emergent pathogens is that the available biological or epidemiological understanding is usually insufficient to control disease at the earliest stages of

outbreak. When epidemiological understanding is flawed, seemingly appropriate management actions may actually increase pathogen spread and disease intensity (Ndeffo Mabah and Gilligan 2010), or simply be insufficient to control pathogen spread despite large investments of management resources (Filipe et al. 2012). Research is essential to addressing these inefficiencies and maximizing the benefits of disease management. Epidemiological models can help guide management by estimating the efficacy of treatments and forecasting the spread and impacts of pathogens based on current data. Of equal importance are field experiments designed to test treatment efficacy and model structure. Field experiments are also essential in establishing a feedback among treatment application, monitoring, and research efforts, including modeling, which comprise adaptive management. For example, field studies improve the understanding of pathogen biology and epidemiology, which can be applied in models to identify the most appropriate treatments thus generating hypotheses that are testable with further field experiments (Sniezko 2006; Gilligan and van den Bosch 2008).

Models of disease risk based on host distribution, environmental conditions, and pathogen spread are useful tools for understanding where and when tanoak mortality will occur. The initial risk models for sudden oak death in California and Oregon (Meentemeyer et al. 2004; Václavík et al. 2010) identified locations that are optimal for pathogen establishment. These models have been reliable tools because the underlying epidemiological assumptions have subsequently been demonstrated to be biologically accurate (Meentemeyer et al. 2012). More recent models incorporate spread following establishment in a given set of locations (Meentemeyer et al. 2011; Filipe et al. 2012). These dynamical models enable estimation of a probable time of invasion for specific tanoak populations and suggest that the majority of the tanoak range is at risk of pathogen invasion and disease emergence over the next several decades. In conjunction with tanoak distribution models (Lamsal et al. 2011), these *P. ramorum* spread models hold promise to predict the timing of disease emergence, risk of tanoak population decline (Dillon et al. this volume), and changes in ecosystem functions such as carbon cycling (Cobb et al. 2012a). Further development of models and datasets to identify levels of biodiversity, cultural, or ecological value would be useful as an overlay with risk models (e.g., Meentemeyer et al. 2004; Václavík et al. 2010) to aid in identification of specific stands where resources for tanoak conservation should be focused.

Management of isolated outbreaks of *P. ramorum* has largely relied on removal of infected hosts to reduce inoculum. These treatments are often combined with removal of susceptible hosts in the surrounding area in an attempt to account for undetected infections (treatment buffers) and have been implemented in southern Humboldt County, Redwood Valley (northern Humboldt County), and Curry County, OR (Hansen et al. 2008; Valachovic et al. 2008). These treatments, when done at an early disease stage, are clearly effective in reducing local pathogen prevalence and the likelihood of spread within treated stands (Hansen et al. 2008; Goheen et al. 2009), suggesting overall rates of spread in the landscape are slowed by aggressive and rapid interventions (Filipe et al. 2012). However, in each case the pathogen has not been eradicated by the treatments and spread throughout the landscape has continued. Undetected, or cryptic, infections are an important source of this continued spread into uninfected stands (Filipe et al. 2012). The broad *P. ramorum* host range, asymmetric impacts on hosts (some hosts support sporulation but are not killed by infections), cryptic infections, and survival in habitats such as soil and watercourses make eradication from a region exceedingly difficult (Hansen et al. 2008; Goheen et al. 2009). However, disease management need not attain 100% pathogen reduction to be effective. Slowing pathogen spread within the landscape is valuable, as it provides time to plan and proactively manage for ecosystem impacts, such as increased fuel loads, stress to biodiversity associated with tanoak, and the accompanying loss of acorn production.

As new isolated outbreaks emerge in northern California and southern Oregon it will become necessary to focus limited management resources to achieve specific cultural and biological conservation goals. Inoculum-reduction treatments such as attempts to locally eradicate the pathogen are expensive to apply and demand significant investments from land managers, researchers, and field staff (e.g., Valachovic et al. 2008) suggesting a shift from pathogen-centric to host-centric management will be more effective to reaching conservation goals when an individual outbreak reaches some yet undefined threshold. However, limiting spread into specific uninvaded landscapes will be aided by attempting eradication of isolated outbreaks as these treatments are likely to reduce inoculum (Filipe et al. 2012). For example, the Redwood Valley outbreak is strategically important given its proximity to culturally and ecologically valuable tanoak stands on tribal lands, within Redwood National Park, and an other nearby public and private lands with large tanoak populations. Pathogen-centric treatments in Redwood Valley have likely slowed *P. ramorum* spread into these valuable nearby areas by reducing inoculum loads. However, further removal of isolated infections outside of the treated area may be needed to attain maximum inoculum reduction or as part of

host-centric management that increases the resiliency of these tanoak populations and protects their associated biodiversity, ecological function, and cultural value. When to shift from pathogen-centric to host-centric management or identifying the optimal balance of each approach is an outstanding question for land managers working to address
P. ramorum and other forest disease outbreaks.

Epidemiological models can inform both pathogen-centric and host-centric management actions (Fig. 2). Often, treatments for each management type will use the same techniques (stand thinning, infected host removal), but the timing and location of treatments may differ. Some insights into the design of these treatments can be gleaned from stand-level epidemiological models which often describe the amount of inoculum that can reach a given individual host through the concept of force of infection (FOI) (for examples, see Meentemeyer et al. 2011; Cobb et al 2012a; Filipe et al. 2012). FOI often has two components: the number of infected hosts weighted by a dispersal kernel (a function of relative distance between hosts), and the rate at which each infected individual transmits the pathogen to susceptible individuals. Sporulation rates vary across host species and are dependent on environmental conditions (rainfall, temperature), therefore formulations of FOI may include species specific pathogen spread factors as well as environmental variation. In addition, the length of time an individual remains infectious, known as the infectious period, also determines the number of individuals which spread a pathogen. Infectious period is poorly quantified for bay laurel and tanoak, but models demonstrate that this parameter can influence rates of pathogen spread at stand-to-landscape scales as well as the persistence of the pathogen within invaded stands (Cobb et al. 2012b; Filipe et al. 2012). The rate of spread and tanoak mortality within a population or landscape increases with FOI and management actions can be directed at one or more of its components. Therefore, the achievement of specific management goals will be improved by understanding which components of FOI are being acted upon by a given treatment.

In general, eradication treatments reduce the number of hosts spreading the pathogen but do not change the rate of spread from each infected host. This is important because infected individuals that remain untreated support continued local and regional pathogen spread (Hansen et al. 2008; Filipe et al. 2012). Prophylactic chemical treatments, such as phosphonate injections, have been shown to reduce the susceptibility of individual tanoak against *P. ramorum* (Garbelotto and Schmidt 2009); while further research is needed on the efficacy, longevity, phytotoxicity and environmental interactions with phosphonate, in general protection of individual trees has been shown to be effective in slowing spread within some populations. Genetic variation within host species may also reduce susceptibility and subsequent pathogen spread. Bay laurel populations in Oregon (known locally as ''myrtlewood'') are not as abundant, susceptible, and consequently not as important in spreading *P. ramorum* as they are in much of California (Hansen et al. 2005; Hüberli et al. 2011), suggesting genetically based differences among host populations may influence disease severity in different regions. Similarly, Hayden et al. (2010) found evidence of resistance to *P. ramorum* infection within tanoak populations, indicating that patterns of resistance could influence tanoak susceptibility or mortality within stands. Similarly to chemical protection treatments, further work is needed to understand how resistance influences pathogen spread rates.

Recent modeling results suggest the degree to which management actions reduce *P. ramorum* infection rates can directly influence the number of tanoak that maintain an overstory canopy position in *P. ramorum* invaded forests (Fig. 3). Following a line of tested epidemiological models (Filipe and Gibson 2001; Gilligan and van den Bosch 2008; Ndeffo Mabah and Gilligan 2010; Filipe et al. 2012), Cobb et al. (2012a) developed a stand level model of disease outbreak with dynamic vegetation composition and tanoak size structure. These models demonstrate that a desired outcome of host-centric management may be feasible: retention of tanoak, including large tanoak, even though *P. ramorum* has invaded the stand. The model shows a critical level of tanoak density (tanoak stems ha^{-1}) below which the pathogen is not able to maintain spread from host to host and consequently, does not diminish tanoak populations or remove overstory tanoak. These results assume bay laurel is not present in, or has been removed from the stand and that remaining tanoak trees are evenly distributed across the stand. The models also suggest that slowed host-to-host infection rates result in better retention of the habitat and ecosystem function provided by tanoak (Fig. 2). Slower infection rates could be achieved by increasing tanoak resistance or stand-level application of effective chemical protection. However, identification of tanoak resistance prior to treatments is currently limited by the lack of molecular-resistance markers; this technical limitation must be overcome to use resistance measurements in broader-scale preemptive treatments. Regardless, the epidemic threshold predictions of the model (e.g., Fig. 3) should still be tested with field experiments, including current phosphonate applications (Garbelotto and Schmidt 2009) and tanoak thinning treatments to understand under which conditions these

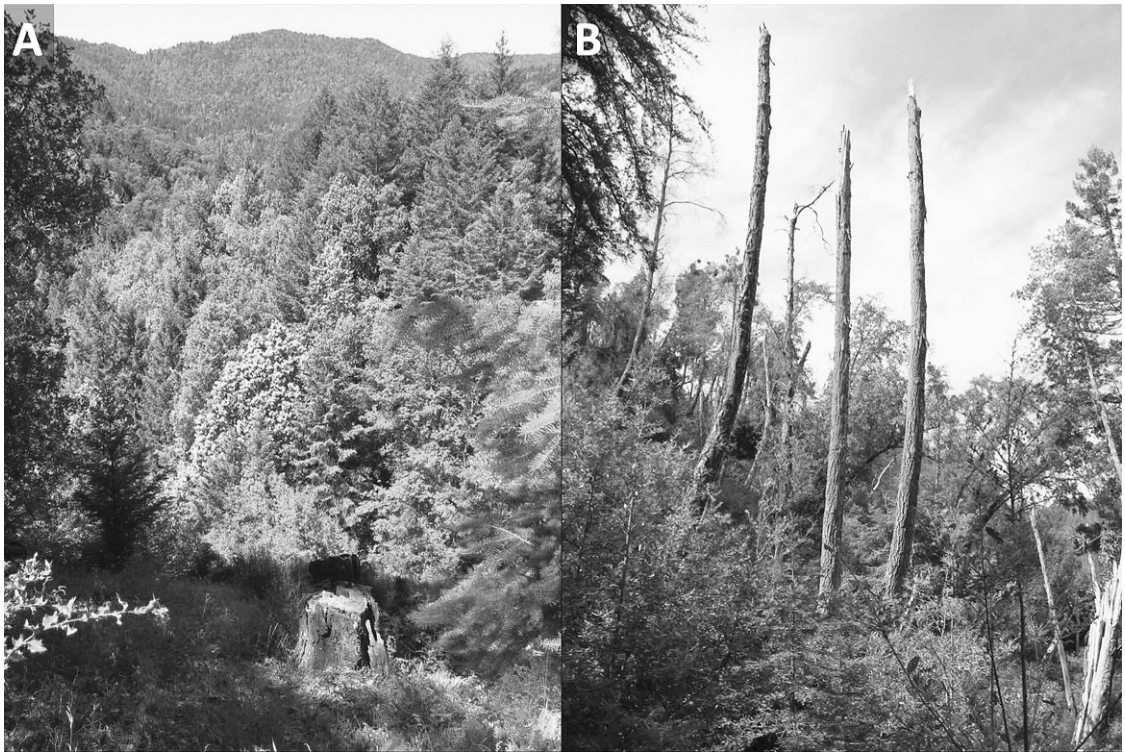

FIG. 2. Landscapes and stands that have not been invaded by *Phytophthora ramorum* (A) offer different opportunities and require different management approaches compared to invaded forests (B) when the goal is to retain tanoak and its associated flora and fauna. Variation in the likelihood of pathogen invasion into pathogen-free landscapes and stands is largely driven by proximity to pathogen invaded stands and community composition. Community composition can be manipulated to slow invasion and disease impacts. Disease impacted stands (e.g., B) require a host-centric management approach that avoids removal of surviving tanoak, protects potential genetically based resistance, and retains ecological functions and species obligate to tanoak. In both cases, successful conservation of tanoak will require continuous feedback between research and management to establish baselines, test hypotheses, and determine the efficacy of management actions.

density thresholds are overcome through long-distance dispersal events (Filipe and Gibson 2001; Gilligan and van den Bosch 2008), sporulation from species not thought to be epidemiologically significant, or human-mediated transport of infected plants (Cobb et al. 2012b; Filipe et al. 2012).

MANAGEMENT GOALS DEFINED: CONSERVE BIODIVERSITY ASSOCIATED WITH TANOAK

Phytophthora ramorum has, and will continue to alter the distribution, prevalence, and biomass of tanoak across California and Oregon. A difficult reality of this disease is that much of this tanoak loss has and will occur regardless of the amount and efficacy of management actions. However, the available management tools and current understanding of the disease may be sufficient to avoid the worst possible outcomes such as the removal of large trees and consequent loss of ecological function (cf., Orwig 2002; Ellison et al. 2005). Management efficacy should be measured at least in part by the success in retaining biodiversity and function associated with tanoak; retention of tanoak is likely to be easier in community or environmental conditions where pathogen spread is lower. Currently, many culturally and ecologically significant tanoak populations have yet to be invaded by *P. ramorum* (Lamsal et al. 2011; Meentemeyer et al. 2011), and timely actions have the potential to reduce future impacts of this disease.

Invaded Stands and Landscapes

Invasion status is a useful first distinction for management efforts and techniques as *P. ramorum* invasion of a stand eliminates the usefulness of many treatments (Fig. 2). For example, prophylactic treatments are ineffective on trees that are already infected (Garbelotto and Schmidt 2009). Similarly, limiting inoculum influx into stands where the pathogen is already established may have a minimal effect in reducing disease because most inoculum is produced

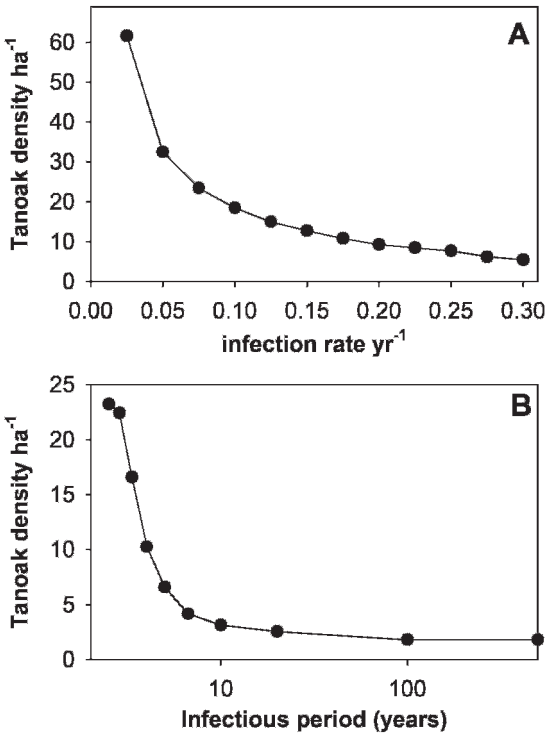

FIG. 3. The epidemiological characteristics of *Phytophthora ramorum* affect the threshold density of large tanoak trees (all trees > 30 cm diameter at 1.3 m) below which *P. ramorum* outbreak cannot be sustained in a parameterized epidemiological model (Cobb et al. 2012b). (A) The epidemic thresholds for tanoak density decreases with increasing infection rate. (B) Similarly, the density of large tanoak decreases with increasing infectious period (log scale; infection rate was kept constant at 0.3).

locally (Davidson et al. 2005; Mascheretti et al. 2008; Cobb et al. 2012b). *Phytophthora ramorum*-invaded stands are potential pathways of spread and the quarantine of infected, sporulation-supporting plants from these stands is a judicious action to reduce, though not eliminate, this pathway of long-distance pathogen spread.

Managers increasingly face isolated outbreaks that threaten to spread into the wider landscape. In Oregon, emphasis has been placed on monitoring and rapid implementation of eradication-type treatments within a designated quarantine zone (Hansen et al. 2008). In northern Humboldt County, the isolated outbreak in Redwood Valley was also addressed with a rapid eradication attempt response, but discovery of the pathogen outside of the treatment area raises the question of what follow-up treatments are needed to maximize the benefit of the initial intervention. The model designed by Filipe et al. (2012) was used to examine a number of landscape-level treatments including inoculum-reduction treatments (such as eradication attempts) at the initial invasion or preemptive host removal ahead of the infection, and construction of a host-free barrier at the landscape-scale. Among these treatments, preemptive removal of hosts ahead of the initial invasion and removal of infected hosts at the initial invasion, similar to
the strategy in Oregon, were found to be most effective in slowing *P. ramorum* spread as long as the treatments were applied early during the epidemic and the scale of treatments matched the scale of the invaded area. The landscape-barrier treatments were not effective in slowing the spread or protecting particular areas because continued spread from undetected infections, or long-distance dispersal across the barrier overcame these treatments.

In regions with the most extensive *P. ramorum* invasion, eradication attempts are a poor choice of management. In highly invaded stands from Big Sur to Sonoma County, *P. ramorum* populations are so large and widely established that any benefit from local removal of infected trees would be overcome by reinvasion from adjacent stands. Eradication attempts always represent a tradeoff between causing tree mortality through cutting and herbicides vs. allowing the pathogen to cause tree mortality. In highly invaded landscapes, management should be aimed at reducing disease impacts and protecting high-value individual stands or trees. For example, in the fire-prone Big Sur landscape, high-density tanoak stands are problematic because sudden oak death generates significant amounts of dead fuels (Metz et al. 2011; Cobb et al. 2012a). Stands with accumulated dead fuels also suffered greater amounts of soil damage during the 2008 Basin Fire (Metz et al. 2011; Big Sur). These patterns suggest treatments which increase resiliency of tanoak populations to *P. ramorum* while also reducing dead fuels may have substantial benefits in terms of reducing ecological costs caused by wildfire in disease impacted forests. Disease-generated fuels are dependent on the rate of mortality and the amount of tanoak biomass present before invasion. This suggests preemptive reduction of tanoak density combined with bay laurel removal is likely to slow disease-driven dead fuel accumulation and reduce maximum amounts (Valachovic et al. 2011; Cobb et al. 2012a). These treatments could easily be structured to retain specific trees such as larger or more resistant individuals and increase spacing between individual trees in order to reduce within-stand spread. However, the effects of increasing stand openness are unresolved; pathogen dispersal may be less impeded in more open stands or, conversely, microclimate conditions could be less suitable to sporulation and infection (Rizzo et al. 2005). This uncertainty reflects the need for further experimental treatments to

understand the interactions of management actions with the epidemiology of *P. ramorum*. The Big Sur region is a strong candidate for experimental fuel and disease reduction treatments given the frequency of fire and the extent of sudden oak death in this region (Rizzo et al. 2005; Meentemeyer et al. 2008b; Metz et al. 2011).

Uninvaded Stands and Landscapes

The most effective strategy to prevent impacts of sudden oak death is to prevent *P. ramorum* invasions into new, at risk environments altogether. However, the history of *P. ramorum* is notable for numerous and often surprising invasions (Werres et al. 2001; Rizzo et al. 2005; Hansen et al. 2008; Brasier and Webber 2010) and current models suggest many stands and landscapes will be invaded in the coming decades (Meentemeyer et al. 2011). Proactive management of uninvaded stands may be much easier to implement where manipulation of stand and community structure are already planned to achieve management goals apart from disease. Where ever possible, host-centric management should aim to achieve multiple goals (Fig. 2).

The design, implementation, and evaluation of preemptive disease treatments are difficult from a research perspective because invasion dynamics are idiosyncratic. Without pathogen invasion of a stand–which is never guaranteed–the efficacy of a preemptive treatment cannot be evaluated. At the same time, intentional pathogen introduction to uninvaded areas must be ruled out on ethical grounds. We suggest two actions that help overcome this practical problem. First, disease management should be put in the context of long-term management goals, such as the reduction of fuel loads, growth of timber species, desired community composition or canopy structure, and/or enhancement of biodiversity. Second, further effort should be allocated to developing models for management evaluation. Models provide insight into the consequences of landscape-level management when experiments at this spatial and/or temporal scale are not possible (Meentemeyer et al. 2012). Lastly, to implement treatments for emerging outbreaks, we caution that management goals must be consistent with the priorities and level of cooperation within the local community (Alexander and Lee 2010).

Genetically-Based Resistance

Genetically-based resistance to *P. ramorum* in tanoak has the potential to be of great practical value. Stands with greater innate resistance are likely to experience lower rates of pathogen spread, fewer disease impacts, and have larger tanoak populations following challenge by *P. ramorum* (Fig. 3). In a study quantifying patterns of tanoak resistance, Hayden et al. (2010; 2011) found variation in susceptibility within tanoak populations, but fairly equivalent susceptibility among populations. If this pattern is broadly representative of tanoak genetic patterns, individual stands are unlikely to resist invasion by *P. ramorum* on the basis of genetic composition alone. However, resources from within stands could be used to maximize tanoak population resiliency in conjunction with other treatments, or to develop less susceptible tanoak growing stock suitable for restoration in *P. ramorum*-invaded areas and preemptive planting treatments in uninvaded landscapes. Further work is needed to increase the ease and rate of identifying tanoak resistance, to determine if rates of sporulation differ in less susceptible trees, and to develop tanoak suitable for restoration.

Where the management goal is to retain biodiversity and function associated with tanoak, treatments need not conserve every living tanoak. Rather, these treatments must retain tanoak populations large enough to maintain both the species and its valued ecological functions. Moreover, sampling and conservation efforts should be targeted to retain genetic diversity across regions of known tanoak differentiation. A genetic analysis of neutral markers by Nettel et al. (2009) has shown deep divisions in chloroplast markers between central coastal California tanoak populations and northern coastal-California/Klamath/Sierra tanoak populations, along with evidence of considerable pollen flow among populations. The variance structure in neutral nuclear genetic markers was similar to the variance in resistance measured on leaves cut from wild trees, with more variance within populations than there was among populations (Hayden et al. 2011). In pathogen-invaded landscapes, disease may be a useful force to select for more resistant tanoak. Hayden et al. (2011) suggested that greater prevalence of resistance at some sites could reflect a selection effect of the pathogen, and McPherson et al. (2010) reported the survival of several mature tanoaks after nearly a decade of monitoring in a *P. ramorum*-invaded forest. These results argue for retention of tanoak where *P. ramorum* has already killed a large portion of the tanoak population. In highly disease impacted stands, treatments which remove surviving trees would clearly exacerbate the loss of tanoak and may reduce genetic resources important for developing molecular markers of resistance and restoration planting stock.

Protect High Conservation Value Stands

Targeted conservation acquisitions, such as land purchases or conservation easements, are

often used to protect rare species or habitat and could be effective in some cases for retaining high-value tanoak populations, such as uniquely located or isolated stands threatened by development. For example, the most southern tanoak populations occur in Santa Barbara County in small and relatively high-elevation stands and a larger geographically-separated tanoak population occurs in the foothills of the central to northern Sierra Nevada range (Fig. 1). In both cases, these tanoak populations hold important genetic diversity, and have lower likelihood of *P. ramorum* invasion because of their isolation, poorer environmental conditions for pathogen sporulation, and low spread risk from adjacent stands (Nettel et al. 2009; Dodd et al. 2010; Meentemeyer et al. 2011). However easements can restrict available management tools needed to slow pathogen spread or protect individual trees and will not be appropriate for many sites or as a method to protect large tanoak populations.

Conclusions–Guidelines for Tanoak Conservation

The goal of tanoak conservation requires an adaptive management approach where management actions and research are designed and conducted with synergistic feedback. We suggest a set of overarching management and research guidelines to achieve this objective.

1. Define the management goals for *P. ramorum* and integrate tanoak conservation as part of a broader vision of landscape management melding treatments for fire, wildlife, aesthetics, and other stand-level goals.
2. Continue efforts to develop field, lab, and molecular tools to identify resistance to *P. ramorum* and develop methodologies to identify and map resistance in the field. Combine this information with stand-to-landscape level epidemiological models to identify tanoak stands with the greatest resiliency to disease.
3. Conduct field experiments to evaluate the efficacy of host-centric management treatments to retain tanoak in *P. ramorum* invaded stands. In combination, conduct epidemiological and bio-economic analysis to identify the optimal timing to shift from pathogen-centric management (eradication and similar slow-the-spread treatments) to host-centric management aimed at retaining tanoak in invaded lands.
4. In uninvaded stands or landscapes where tanoak is highly valued, increase the resiliency of these populations by reducing the potential for pathogen spread (i.e., reduce bay and tanoak densities) while retaining the most resistant tanoak individuals.
5. Develop host-centric, ameliorative and proactive treatments that reduce the ecological costs of disease for: fire prone forests, regions with the greatest potential ecological impact of disease (i.e., areas with high tanoak importance and biomass), and lands where tanoak has the greatest cultural value.
6. Work with the public to increase appreciation and interest in tanoak and to encourage further participation in pathogen monitoring and disease management (cf., Alexander and Lee 2010).
7. Increase collaboration among state and federal agencies, and public-private partnerships to support management in tanoak stands.
8. Expect the unexpected. *Phytophthora ramorum* is a remarkably well-adapted pathogen for which the native host or geographic range has not yet been identified. The importance of pathogen characteristics often becomes clear only after a new management problem has emerged.

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