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ORIGINAL ARTICLE

Changes to relative stand composition after almost 50 years of *Heterobasidion* root disease in California true fir and pine forests

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Abstract

The Agaricomycete *Heterobasidion annosum* sensu lato (s.l.), a species complex of fungal pathogens, causes root and butt rot on conifers throughout the northern hemisphere, thereby shaping structure, composition, and evolution of vast and diverse forest ecosystems. We analyze forest change 48–49 years following Heterobasidion root disease emergence in 63 permanent plots first established in 1970–1972 and measured at least once every decade through 2020. We use this dataset to infer the long-term consequences of *Heterobasidion* root disease in a set of common forest types that reflect some of the most important hosts for this set of diseases in western North America. We contrasted three host–pathogen systems located within the Sierra Nevada, southern Cascades Mountain Range and the Modoc Plateau: (1) *H. irregulare* in host communities dominated by several *Pinus* (pine) species and *Calocedrus decurrens* (incense cedar) in Yosemite Valley, on the Sierra Nevada Range western slope; (2) *H. irregulare* in a largely single species (*Pinus jeffreyi*) host disease system on the eastern slopes of the Sierra Nevada and southern Cascades as well as the Modoc Plateau; and (3) *H. occidentale*—also on the western slope of the Sierra Nevada—in stands with a mixture of susceptible *Abies* (true fir) and a diversity of non-hosts for this *Heterobasidion* species. Approximately 50 years after disease emergence, relative basal area and stem density were significantly reduced within disease centres in all three pathosystems, but changes to forest composition and relative species dominance were determined by pre-disease host and non-host diversity. In the western-slope *H. occidentale* system, the disease increased the dominance of non-susceptible species, *Pinus* species and *C. decurrens*. In the multi-genus Yosemite pathosystem, *H. irregulare* did not significantly shift species dominance, and as expected, species shifts did not occur in the largely single-genus eastern-slope forests. In these widespread California forest ecosystems, two factors appeared to determine forest conditions almost 50 years after Heterobasidion root disease monitoring: the size of the disease centre and the initial diversity of non-hosts. Along with pathogen species, these factors appear to affect the local severity of disease as well as the degree of species shifts relative to initial plot compositions, reaffirming host susceptibility classifications associated with these forest types.

1 | INTRODUCTION

The Agaricomycete *Heterobasidion annosum* sensu lato (s.l.) is a fungal species complex that includes pathogens that commonly cause root and butt rot on conifers throughout the northern hemisphere including western North American conifer forests (Barrett & Robertson, 2021; Garbelotto & Gonthier, 2013; Rizzo & Slaughter, 2001). Conifer forests of California are impacted by *H. irregulare* and *H. occidentale*, two closely related root pathogens with different host preferences and ranges. *Heterobasidion irregulare* attacks a wide range of species within California and worldwide (Garbelotto & Gonthier, 2013), and *H. occidentale* is particularly damaging to several species of *Abies* and attacks several non-*Pinus* coniferous species (Garbelotto & Gonthier, 2013). Here we focus on several widespread and often locally dominant *Heterobasidion* host species including several *Pinus* species and incense cedar (*Calocedrus decurrens*) which are impacted by *H. irregulare* and two *Abies* (*A. concolor* and *A. magnifica*) species impacted by *H. occidentale*. Both *Heterobasidion* pathogens appear to impact forests from the stand to landscape levels and at time scales spanning decades (Garbelotto et al., 1997; Rizzo et al., 2000; Slaughter & Parmeter, 1995). While the Heterobasidion root disease impacts are significant, the widespread distribution of these pathogens creates potential for interactions with other major disturbances shaping the region's forests including wildfire, bark beetles and drought, which increases their relevance in light of a broad set of management challenges (Cobb, 2022; Lalande et al., 2020; Poloni et al., 2021).

Canopy gaps caused by Heterobasidion root disease can reach up to 5% of the total area in some landscapes and persist and gradually expand for decades (Rizzo & Slaughter, 2001; Rizzo et al., 2000). Therefore, *Heterobasidion* pathogens, along with other root diseases, may be contributing to increased tree mortality across the western United States (Cobb et al., 2017; Hartmann et al., 2015; Millar & Stephenson, 2015). Although these disease centres are persistent, if, how, and on what time scales *Heterobasidion* alters forest structure and composition in California are not well quantified. In this study, we compare three examples of Heterobasidion root disease in terms of their changes in relative species composition and stand structure ~50 years (48 and 49 years) after the disease first emerged. We seek to understand if these pathogens cause persistent shifts in species composition and forest structure on a time span significantly longer than similar studies in the region and contrasting multiple *Heterobasidion* pathogens (cf. Garbelotto et al., 1997; Rizzo & Slaughter, 2001; Rizzo et al., 2000; Slaughter & Parmeter, 1995). Addressing these goals could inform a range of land management actions from fuel mitigation to timber production.

Our measurements span a region including the Sierra Nevada, southern Cascade mountains and the Modoc Plateau, which encompass much of the epidemiological variability of root diseases caused by *H. occidentale* and *H. irregulare* in California (Garbelotto & Gonthier, 2013). The study region is experiencing climate-driven increases in tree mortality and wildfire (Fettig et al., 2019; Preisler et al., 2017; Williams et al., 2019); thus, understanding biological drivers of mortality and any resulting species changes may inform these other forest health issues as they clearly overlap spatially and may interact ecologically (Cobb, 2022; Johnstone et al., 2016; Millar & Stephenson, 2015). Within the Sierra Nevada range, *H. occidentale* is a pathogen of the relatively productive western-slope forests, often dominated by *Abies concolor* and *A. magnifica* (white and red fir, respectively, see Dolanc et al., 2014). *Heterobasidion occidentale* is pathogenic on both *Abies* species, but saprotrophic on abundant co-occurring species including incense cedar and pines, notably *Pinus ponderosa* and *P. jeffreyi* (ponderosa and Jeffrey pine, respectively). *Heterobasidion occidentale* becomes established within stands via direct infection of standing trees likely on tree wounds and secondarily on freshly cut stumps (Garbelotto et al., 1997; Otrosina & Garbelotto, 2010; Poloni et al., 2021). In contrast, *H. irregulare* is pathogenic and saprotrophic on incense cedar and western juniper, as well as ponderosa, Jeffrey, and lodgepole pines. *Heterobasidion irregulare* is a widespread pathogen, causing root disease in eastern slope forests of the Sierra Nevada, southern Cascades, as well as the Modoc Plateau (Slaughter & Parmeter, 1995). Freshly cut stumps are an example of management activities that foster the spread of *H. irregulare*, contributing to within-stand development of this disease (Otrosina & Garbelotto, 2010; Rizzo et al., 2000; Shaw et al., 1995). For both *Heterobasidion* species, disease centres commonly originate from a single infected tree, often a stump, and spread secondarily via root-to-root contact (Garbelotto et al., 1997; Rizzo et al., 2000). Previous studies have demonstrated these disease centres to be persistent and active over decades suggesting changes to stand composition and structure compared to pre-monitoring conditions will also be long-lasting (Rizzo et al., 2000; Slaughter & Parmeter, 1995).

We resurveyed three plot networks representing the range of *Heterobasidion* pathogens in California, and the range of host communities that they impact, using a set of repeated surveys of tree mortality in a dataset spanning 48–49 years after disease emergence. Collectively, these community and pathogen combinations represent variable levels of initial host or non-host canopy dominance and diversity ranging from a relatively simple system where all trees are a single susceptible host species to a system with multiple hosts and hon-hosts. In epidemiological terms, this variability encompasses an almost exclusively single-host disease epidemic with a minimal non-host component (Eastside pine), multi-host epidemic with a minimal non-host component (Yosemite), and a single-genus-host epidemic with a relatively diverse non-host component (true fir). In this dataset, we lack a true reference set of measurements and inferences must be made using a pre–post epidemic comparison. Furthermore, this approach can only be made when other disturbances common to the region such as timber harvest or wildfire have not overlapped with the disease. The potential for disturbance overlap and interaction is a common challenge in long-term monitoring of Heterobasidion root disease in California and is mitigated here, and in past studies, by regular and repeated surveys over the study period (here, every 4–8 years; see also Preisler & Slaughter, 1997; Rizzo et al., 2000; Slaughter & Parmeter, 1995) as well as a careful transfer of data and training in survey approaches by former-lead surveyors

(G. Slaughter). No formal comparison of Heterobasidion root disease impacts has been made across these epidemiological and host–pathogen systems in this region, although previous work has demonstrated root disease centres can range from relatively small to large areas (~250 m^2 to >1 ha area) and persist for at least 25 years (Rizzo & Slaughter, 2001; Rizzo et al., 2000; Slaughter & Parmeter, 1995). Leveraging a high quality of monitoring with the variability in *Heterobasidion* species, host and non-host communities, allows us to specifically address the following: (1) after an approximately 50-year timespan since initial disease emergence, do pathogen impacts differ among these host and pathogen systems within California in terms of total disease area and dominance measures including relative changes in basal area, stand density and importance value? (2) Does disease result in relative changes to host or non-host canopy tree species dominance within these long-term disturbance sites? The previous work left unclear whether regeneration and canopy infilling would diminish pathogen impacts in the following decades, and whether disease is associated with changes in overstory species composition and dominance, as has been demonstrated in other forest disease systems (Cobb et al., 2010; Gómez-Aparicio et al., 2012; Reinhart et al., 2010). Irrespective of these uncertainties, we can expect changes in species dominance could only occur via the establishment of a new canopy dominant species in the Eastside pine plot network or ecosystem conversion as these forests are typified by low canopy diversity. In contrast, changes in species dominance could occur in the Yosemite and Fir epidemiological systems as higher canopy diversity of host and non-hosts species typify each system (respectively). In the Yosemite system, these species shifts could occur due to differences in survival times within disease centres between the two primary hosts, ponderosa pine and incense cedar (Rizzo & Slaughter, 2001; Rizzo et al., 2000). In the true fir monitoring network, shifts in species composition could occur due to selective mortality of hosts and competitive release of non-hosts (Cobb et al., 2010; Hawkins & Henkel, 2011; Holt & Lawton, 1994).

2 | METHODS

2.1 | Geographic area and pathosystems

The three study pathosystems are distributed within a geographic area spanning the Sierra Nevada, southern Cascades and Modoc Plateau; however, the respective hosts and pathogens do not exist uniformly across this area (Figure 1). For each epidemiological system (here synonymous with plot network), *Heterobasidion* presence was initially documented through direct sampling of symptomatic but living trees and stumps during plot establishment. At the time of plot establishment, our two focal tree species (pine and true fir) were thought to represent variations adapted to different hosts with *H. irregulare* and *H. occidentale*, respectively. Plots with confirmed infections became the basis for an ongoing survey of tree mortality and disease centre dynamics. Although direct sampling of

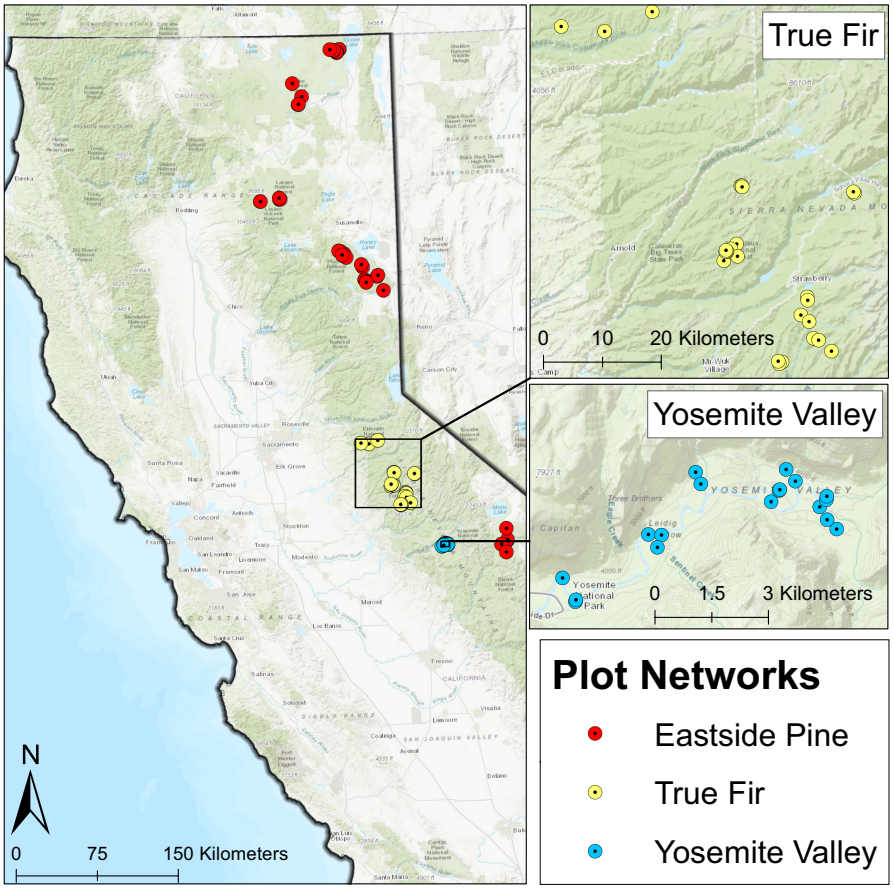

FIGURE 1 Location of our three Heterobasidion root disease monitoring networks in northern California with national forest lands in light grey. The Eastside pine and Yosemite plot networks are impacted by *H. irregulare* while the fir plot network is impacted by *H. occidentale*. See text for descriptions of the respective host communities.

each *Heterobasidion* species was not part of regular plot resampling, signs and symptoms of *Heterobasidion* pathogen activity including root rot, delamination of root tissue, and reduction in tree health were common within and on the disease centre bordering trees during the subsequent decades of plot surveys (Rizzo et al., 2000; Slaughter & Parmeter, 1989, 1995). Over the course of almost 50 years, other disturbances including timber harvest, wildfire, and Armillaria root disease occurred on particular plots. As a result, these were dropped from the ongoing monitoring effort and are not included in our analysis.

We investigated the impacts of *H. irregulare* in low-canopy diversity, *Pinus* dominated forests which occur on the eastern-slopes of the Sierra Nevada, southern Cascade Range and Modoc Plateau ('Eastside pine'; Figure 1). In these forests, stand composition is typically uniform, dominated by ponderosa or Jeffrey pines which are epidemiologically indistinguishable (Slaughter & Parmeter, 1995). *Heterobasidion irregulare* also impacts *Juniperus occidentalis* (western juniper), which is a dominant canopy tree in many forests of the Modoc Plateau, but was not adequately represented in our dataset to allow inference on its role in disease dynamics or to contrast disease among hosts in these forests. Therefore, we excluded four plots with a significant canopy component of *J. occidentalis* (>10% density and/or basal area) and grouped *J. occidentalis* into a single host class; in total this included 39 live *J. occidentalis* stems or <1% of all live host stems and total basal area within all *H. irregulare* plots. *Heterobasidion irregulare* is common in these forests with infections associated with past logging (Slaughter & Parmeter, 1995). *Heterobasidion occidentale* is also present in these forests although its primary host genus (*Abies*) is virtually absent. This plot network was originally established in 1972 by the USDA Forest Service—Forest Pest Management (currently Forest Health Protection), in response to landscape-scale Heterobasidion root disease emergence associated with past timber harvest; these monitoring plots are located across five National Forests from Modoc to Inyo Counties (Slaughter & Parmeter, 1995; Figure 1).

We contrasted this Eastside pine system with an isolated, but damaging *H. irregulare* epidemic in Yosemite Valley, where ponderosa pine and incense cedar co-occur at the stand level, and each species transmits, suffers mortality, and contributes to Heterobasidion root disease persistence within a multi-genus host system ('Yosemite'; Figure 1; Rizzo & Slaughter, 2001; Rizzo et al., 2000). The Yosemite epidemic is one of few known *H. irregulare* occurrences on the western slopes of the Sierra Nevada Range and the system is similar to the Eastside pine epidemic in that these study plots include only a minor component of non-hosts, primarily California black oak (*Quercus kelloggii*), white fir and Douglas fir (*Pseudotsuga menziesii*). This *H. irregulare* epidemic has been traced back to the mid-1940s when it emerged as an inadvertent result of tree removals conducted for a bark beetle suppression campaign (Rizzo & Slaughter, 2001; Slaughter & Rizzo, 1999). A 1950s survey mapped and isolated the pathogen in symptomatic trees following logging activity, which became the basis for a permanent plot-based monitoring effort which began in 1971 (Rizzo et al., 2000; Slaughter & Rizzo, 1999).

Lastly, we investigated the dynamics of *H. occidentale* in western slope Sierra Nevada forests dominated by *Abies* species ('Fir'; Figure 1). These forests have relatively higher tree species diversity compared to the other two epidemiological systems, but pathogenicity is restricted to red and white fir (Garbelotto et al., 1997; Garbelotto & Gonthier, 2013). Non-host species include a substantial component of incense cedar and pines including ponderosa pine, Jeffrey pine, sugar pine (*P. lambertiana*) and western white pine (*P. monticola*). The potential host species mountain hemlock (*T. mertensiana*) is not present in these plots due to their low elevation. This plot network was also established in 1972 as part of a regional post-drought tree mortality survey that identified Heterobasidion root disease centres on western-slope *Abies*-dominated forests of the Stanislaus and El Dorado National Forests (Slaughter & Parmeter, 1989). It is very likely that *H. occidentale* occurs with *H. irregulare* in the Yosemite dataset just as it probably occurs in the Eastside pine dataset, but *Abies* hosts are almost entirely absent within the Yosemite plots meaning a direct comparison of *H. occidentale* across the systems is not possible.

2.2 | Field surveys

A common survey method has been applied to each plot network since their inception and has been maintained over several decades of monitoring including transitions among lead personnel. To ensure proper understanding of the data collection procedures and limitations, the authors worked with previous lead surveyors in the field and with original data records as part of a data ownership transfer during the penultimate survey (2010–2011). The survey approach is unusual, plots do not have a fixed area, instead the surveyed area changes with the extent of disease to capture the dynamic nature of tree mortality while also capturing regeneration within plots. While an expanding plot size is unconventional, the final size of each disease centre could not be known during the initial survey. The decision rule for which trees are added to the survey as the extent of disease increases is, regrettably, described somewhat differently across the previous reports on these plot networks (Rizzo et al., 2000; Slaughter & Parmeter, 1989, 1995). We are able to leverage these separate datasets to address a set of common questions due to a unifying and epidemiologically informed philosophy, which defines the rules for expansion of individual monitoring plots (G. Slaughter, personal communication). Following initial pathogen establishment on a stump, disease progression is largely driven by root-to-root spread in each of these systems (Garbelotto et al., 1997). Therefore, at each survey up to two trees beyond the extent of the disease centre at the time of each survey were added to the monitoring dataset. These trees were constrained such that they must be located within a plausible distance of root-to-root contact of trees that define the canopy gap or the trees which neighbour those defining the canopy gap edge (respectively). In practical terms, this distance never exceeded 8 m and allows disease centre monitoring to expand up to, but not beyond, a natural or artificial border such

as a road, non-forest ecosystem, rock outcrop, or other analogous feature. In practical terms, the approach tracks tree mortality and canopy opening size associated with the disease and results in different census areas across plots (Rizzo et al., 2000).

Within the census area, all trees ≥1 cm diameter at breast height (1.37 m) were initially mapped and measured for diameter, monumented with aluminium tags, and rated for overall health, such as living/dead status, tree crown health rating, and other notable observations including mistletoe infestation level (see Rizzo et al., 2000; Slaughter & Parmeter, 1995). At each remeasurement, all previously mapped trees were surveyed for root disease symptoms and rated for health. Diameter was remeasured at least once every decade along with changes in disease centre area, individual tree mortality and tree recruitment were recorded at each survey. Changes in root disease area were determined through mapping of tree mortality and calculating the resulting area of the disease centre as a convex hull with the margins determined by the furthest distances to the face of the bole of dead trees. Disease centre calculations are based on the extent of mortality as opposed to canopy opening which allows for disease centres to include live trees, including the non-hosts and regenerating seedlings and saplings central to understanding if these canopy disturbances are persistent over several decades. Plot remeasurements were generally conducted every 2–3 years for the first 2 decades and approximately every 8 years, thereafter with the most recent surveys conducted in October 2019 for the 'Yosemite' plot network ($N = 21$, 49 years of monitoring) and between July and August 2019 for the 'true fir' plot network ($N = 23$, 48 years of monitoring). The 'Eastside pine' plot network was last measured during July and August 2020 ($N = 47$, 49 years of monitoring).

2.3 | Data analysis

We conducted a pre- versus post-disease comparison of relative changes in forest composition and structure with a focus on metrics of canopy species dominance. This approach differs from a before-after-control or the same approach with an 'intervention' (a treatment), the so-called BACI experimental approach. The extensive period of observation combined with regular inclusion measurements outside the disease centre allowed us to exclude plots where a clear and confounding canopy disturbance rendered a specific plot no longer useful to contrast these diseases across habitat types. Several plots with clear or severe impacts of wildfire, harvest, development (roads or trails), and Armillaria root disease were excluded from our analysis. Data from all surveys were consolidated into a single database and audited across field seasons and field crews for the nearly 5 decades of data collection. Plot records, which could not be validated in the two most recent surveys conducted by the authors (2013 and 2019/2020), were eliminated. Most of these discarded measurements were associated with incomplete or inaccurate data collected on trees outside the disease centres.

The final data set was employed to calculate a quantitative summary of stand changes between pre- and post-disease conditions for three forest types with variable canopy diversity for an approximately 50-year period spanning the initiation of measurements and the final survey. Pre-disease conditions were calculated as the cumulative summary of all unique trees surveyed at any of the measurements but within the final disease centre. This rendering represents stand conditions that would exist without disease-related mortality excepting, of course, unrecorded recruitment, natural mortality and changes in tree diameter, which occurred in the area initially outside of the plot boundary. We calculated multiple relative stand metrics that vary in their sensitivity to this source of uncertainty: relative density, relative dominance, and importance value, which is the average of the former two metrics rendered as a score from 0 to 200 (Nguyen et al., 2015). Post-disease condition measurements are straightforward, as relative dominance, density, and importance value are a simple calculation based on the stand composition and structure within the disease centre during the most recent survey (conducted in 2019 or 2020).

We conducted a series of statistical analyses on disease centre characteristics and changes in stand composition within each system by comparing the initial pre-disease forest structure to the observed post-disease structure (pre–post disease comparison). To address comparative disease impacts and persistence (study question 1), we compared the three disease systems in terms of cumulative disease impacts with a series of one-way analysis of variance (ANOVA) models examining disease centre area, percent stem density reduction, and percent basal area reduction. A Tukey's HSD (honestly significant difference) test was employed to examine differences between the three epidemiological systems when the overall effect term was significant ($p \leq .05$).

Relative changes in species dominance (study question 2) were investigated with a series of analysis of covariance (ANCOVA) models, which assess and quantify changes in relative dominance in each epidemiological system. All trees were classified into host or non-host categories for the respective Heterobasidion root disease system. ANCOVA models were constructed to assess differences between initial and post-observation conditions, a categorical variable, while controlling for initial host density, a continuous variable. Each model followed the general form of dominance measure = Disease + Stand + ε where Stand is a statistical control variable parameterized as pre-disease host or non-host density ha^{-1}, pre-disease host or non-host basal area m^2 ha^{-1}, or last recorded gap area m^2 for models where relative stem density, relative basal area and importance value models are the dependent variable (respectively). These parameters help address problems with relativistic response variables, namely that absolute changes in dominance will not necessarily mirror changes to total density or basal area; these variables are expected to be positively associated with the dominance of specific species or host classes represented by the dependent variable in the models. Lastly, ε is a normally distributed error term. Each model was assessed for normal distribution of error and constant variance. The log and logit transformations were necessary to meet these assumptions for the one-way ANOVAs comparing disease centre area and % basal area loss after conversion to proportions, respectively.

The logit transformation was also applied to meet these assumptions for the suite of response variables for the ANCOVA models of the single-host *H. irregulare* system (Eastside pine) after converting the importance value to a proportion. Statistical significance was considered where $p \leq .05$.

3 | RESULTS

After 48–49 years of disease monitoring, both *H. occidentale* and *H. irregulare* have exerted substantial and long-lasting changes to stand structure within disease centres with *H. occidentale* further causing changes in relative species composition. Differences in disease extent and intensity are clearly apparent when comparing Heterobasidion root disease among three forest types and two *Heterobasidion* species (study question 1; Figure 2). Total disease centre area was significantly greater in the multi-host *H. irregulare* system (Yosemite) compared with the single-host genus *H. irregulare* (Eastside pine) and *H. occidentale* multi-host genus (true fir) host–pathogen systems, while disease centre area in the latter two was not significantly different from one another (Figure 2; $p < .0001$ and $p = .99$, respectively). These differences in disease centre area were the most striking difference among the epidemiological systems. The multi-host *H. irregulare* system (Yosemite) averaged ~1400 m^2 larger, or 3.3 and 4.0 times greater in area compared with the Eastside pine and fir systems, respectively (Figure 2).

Each host–pathogen system resulted in reductions in total tree density and basal area irrespective of differences in total disease area. Stem density reduction across all species was greater in the single-host *H. irregulare* system (Eastside pine) compared with the *H. occidentale* (fir) and multi-host *H. irregulare* plot monitoring networks (Yosemite, $p < .01$, each contrast). However, the *H. occidentale* (true fir) and multi-host *H. irregulare* (Yosemite) systems were not different from each other in terms of relative density reduction ($p = .58$; Figure 2). Total basal area loss was significantly greater in *H. occidentale* host–pathogen system (true fir) compared with the *H. irregulare* single-host system (Eastside pine, $p < .0001$). The multi-host *H. irregulare* system (Yosemite) showed a trend of lower basal area loss compared with the Eastside pine and Fir systems, but these differences were not statistically significant ($p = .058$ and .074, respectively; Figure 2). As a whole, we found evidence of variable disease severity across the three study systems although it is also clear that each epidemiological system resulted in severe loss of trees as quantified by the proportion of basal area and/or density change between pre and post disease conditions.

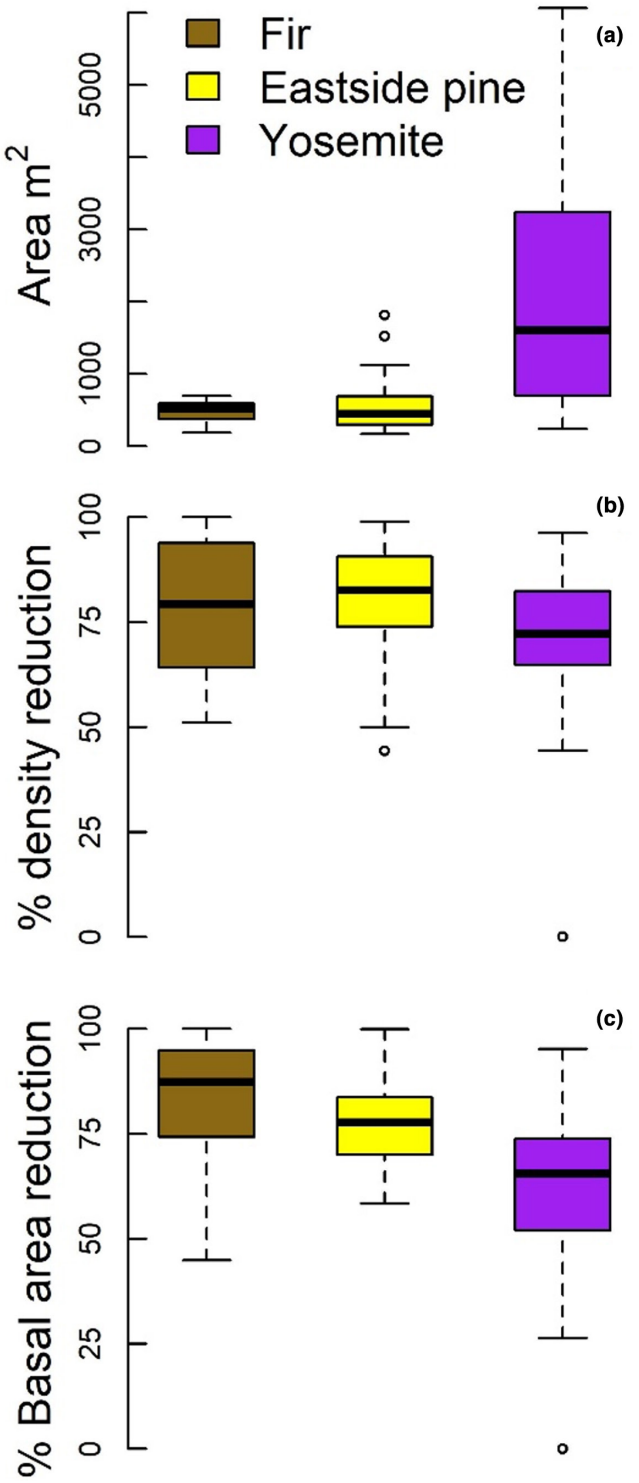

FIGURE 2 Standard boxplots identifying the median (black line) and points 1.5 times outside the interquartile range (outliners) for Heterobasidion root disease host impacts 48–49 years after disease emergence in three Heterobasidion root disease systems in California with (a) total disease centre area, (b) host % stem density reduction and (c) host % basal area reduction, in three contrasting epidemiological systems: single host *Heterobasidion occidentale* ('Fir'), single host *Heterobasidion irregulare* ('Eastside pine') and multi-host *H. irregulare* ('Yosemite'). Proportional stand change was calculated from a reconstruction of stand structure within a few years of disease emergence and stand structure measured 48–49 years later.

We also found broadly consistent effects of the statistical control variables (stem density, basal area and disease extent in units of area). The effects were largely positively associated with relative dominance of the focal species or host class in all cases except for

TABLE 1 Analysis of covariance model parameter estimates of changes in relative host and non-host dominance across three Heterobasidion root disease systems after 48–49 years.

System	Stand[a]	Disease[b]
Stand component		
Metric		
H. occidentale (fir)		
Hosts		
Relative density	4.56×10^{-4}	−0.215
Relative basal area	4.31×10^{-3}	−0.198
Importance value	0.186	−41.33
Non hosts		
Relative density	8.08×10^{-4}	0.215
Relative basal area	4.89×10^{-3}	0.198
Importance value	−0.186	41.33
H. irregulare (multi-host, Yosemite)		
Hosts		
Relative density	-[d]	-[d]
Relative basal area	2.82×10^{-3}	-[d]
Importance value	-[d]	-[d]
Non hosts		
Relative density	2.61×10^{-3}	-[d]
Relative basal area	9.39×10^{-3}	-[d]
Importance value	-[d]	-[d]
H. irregulare (single host, Eastside pine)		
Hosts		
Relative density[c]	-[d]	-[d]
Relative basal area[c]	-[d]	-[d]
Importance value[c]	-[d]	-[d]
Non hosts		
Relative density	7.0×10^{-4}	-[d]
Relative basal area	0.017	-[d]
Importance value	-[d]	-[d]

[a]Statistical control variable parameterized as host or non-host density (trees ha^{-1}), host or non-host basal area ($m^2\,ha^{-1}$), or gap area (m^2) for relative density, relative basal area and importance value models, respectively.

[b]Estimated changes in the respective parameter between pre- and post-disease emergence measurements.

[c]Logit transformation applied to the response variable.

[d]Parameters which do not meet the criteria for statistical significance ($p > 0.05$).

TABLE 2 Analysis of covariance model estimates of changes in relative dominance of incense cedar (*Calocedrus decurrens*) and pines (several species, see text) in two Heterobasidion root disease systems after 48–49.

System	Stand[a]	Disease[b]
Stand component		
Metric		
H. occidentale (non-hosts, fir)		
Incense cedar		
Relative density	9.22×10^{-4}	0.183
Relative basal area	6.08×10^{-3}	0.164
Importance value	−0.145	34.68
Pines		
Relative density	1.04×10^{-3}	-[c]
Relative basal area	5.71×10^{-3}	-[c]
Importance value	-[c]	-[c]
H. irregulare (Hosts, Yosemite)		
Incense cedar		
Relative density	9.49×10^{-4}	-[c]
Relative basal area	8.09×10^{-3}	-[c]
Importance value	-[c]	-[c]
Pines		
Relative density	2.02×10^{-3}	-[c]
Relative basal area	6.63×10^{-3}	-[c]
Importance value	-[c]	-[c]

[a]Statistical control variable parameterized as host or non-host density (trees ha^{-1}), host or non-host basal area ($m^2\,ha^{-1}$), or gap area (m^2) for relative density, relative basal area and importance value models, respectively.

[b]Estimated changes in the respective parameter between pre- and post-disease emergence measurements.

[c]Parameters which do not meet the criteria for statistical significance ($p > .05$).

non-host and incense cedar importance values in the *H. occidentale* system (Tables 1 and 2). These patterns broadly support the expectation that absolute measures of host abundance, biomass, or survey area would track relative dominance, even when disease shifts canopy structure or composition. However, the discrepancy for incense cedar dominance—in terms of importance value—occurred where it is a non-host. This also implies that initial incense cedar dominance could be inversely associated with the factors that drive disease centre area in the *H. occidentale* system.

We found evidence of persistent (~50 years) dominance shifts within the single host genus *H. occidentale* (fir) system, but not the multi- or single-genus host *H. irregulare* systems (Table 1; Figure 3). After 48–49 years, *Abies* host dominance in the *H. occidentale* system, collectively white and red fir, was significantly reduced in terms of each dominance measure ($p < .001$, each contrast), and the proportion of non-host species was increased in post-disease ($p < .001$, each contrast; Figure 3). In contrast, in both the multi- and single-host (pine) *H. irregulare* pathosystems, the relative dominance of hosts and non-hosts was essentially unchanged between pre- and post-disease conditions (Figure 3). In the Eastside pine system, this is better described as preordained given that non-hosts were virtually absent in both the pre- and post-disease stand conditions (Figure 3). For both *H. irregulare* systems, we found no statistically significant differences for any of the three dominance measures in pre- versus post-disease conditions, indicating that dominance was essentially unchanged by disease even though host mortality was substantial. In contrast, host community appeared to shift in the *H. occidentale*

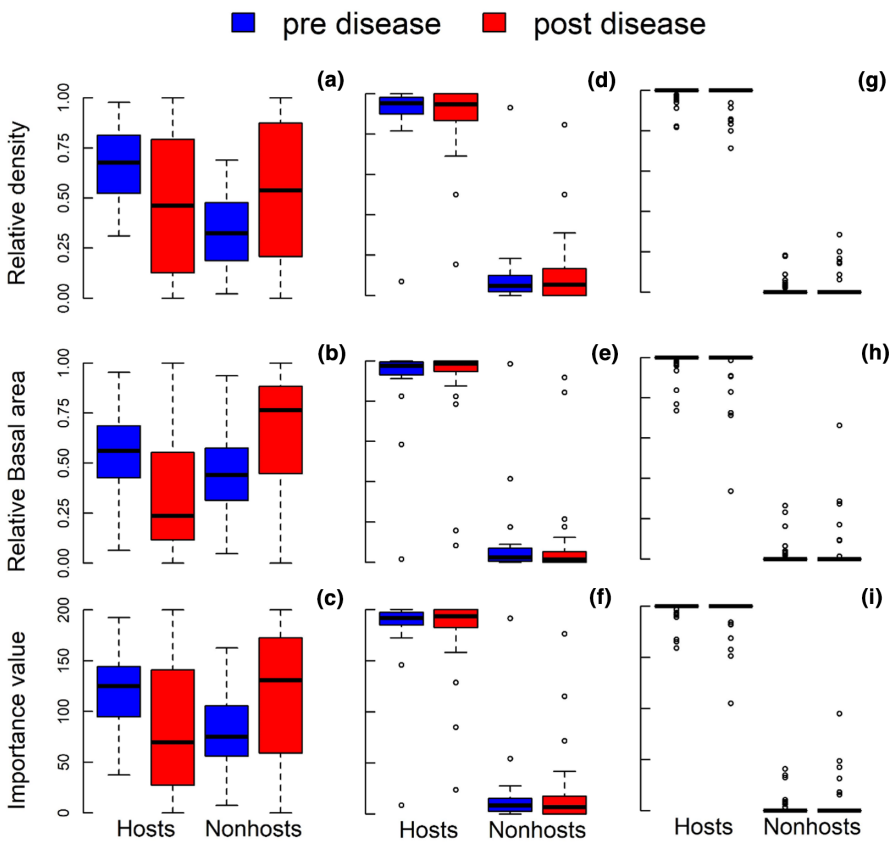

FIGURE 3 Standard boxplots identifying the median (black line) and points 1.5 times outside the interquartile range (outliners) showing relative changes in stand composition after 48–49 years contrasted between hosts and non-hosts in three epidemiological systems: single host *Heterobasidion occidentale* (a–c), single host *Heterobasidion irregulare* (d–f) and multi-host *H. irregulare* (g–i). The three systems correspond to forest tree communities dominated by: (1) fir (*A. concolor* and *A. magnifica*) in (a)–(c), (2) Jeffrey pine (*Pinus jeffreyi*) in (d)–(f) and (3) incense cedar—ponderosa pine (*Calocedrus decurrens* and *P. ponderosa*, respectively) in (g)–(i), which are pathogen hosts in each respective community. Pre-disease data are a reconstruction of stand structure within a few years of disease emergence.

system. For these three forest types, amounts or occurrences of species shifts track the degree of pre-disease canopy diversity. Here, significant change occurring between pre- and post-disease conditions in the fir system where non-host canopy species are substantial and no, or virtually no, change occurred in the Eastside pine and Yosemite disease centres where non-hosts simply do not occur in substantial amounts (Figure 3). For the Eastside pine and Yosemite disease systems, the impact of disease was a simple function of the extent of the disease centre and the severity of mortality (Figure 2).

We were able to test for changes in host and non-host dominance in greater detail for the incense cedar and pine. Both species were important components of the multi-host *H. irregulare* system (Yosemite) where both are hosts and the *H. occidentale* (fir) where both are non-hosts but support pathogen persistence as a saprotroph. Unsurprisingly, each species or group of species responded substantially differently between the two systems (Figure 4; Table 2). In the *H. occidentale* pathosystem, incense cedar dominance was substantially increased by disease on *Abies* species in terms of relative density, relative basal area and importance value ($p < .0001$, $p < .001$ and $p = .025$, respectively). In the *H. occidentale* plot network (fir), 'pine' represents four *Pinus* species (sugar, western white, ponderosa and Jeffrey pine), and, as a class, dominance of these non-hosts was not significantly changed by any of the three measures ($p > .4$ each contrast). We were unable to make a meaningful comparison of the same dominance changes in the single-host *H. irregulare* system because incense cedar is nearly absent in the plot network (5 of 4750 trees), and 'pine' as a class is equal to the class 'hosts' (Figure 3). However, in the multi-host *H. irregulare* system (Yosemite) both incense cedar and pine encompass relatively similar proportions of pre-disease host density. Here, dominance of both incense cedar and pine was unchanged (Yosemite, $p > .1$, each contrast) although we found a consistent, but not statistically significant, trend of increased incense cedar dominance for each dominance measure: relative density, relative dominance, and importance value ($p = .14$, .12 and .19, respectively). In the Yosemite host–pathogen system, pine relative density, relative basal area and importance value changes suggest decreases in pine dominance, although in each case, the change in dominance was also not statistically significant ($p = .12$, .07 and .17, respectively). Thus, only the *H. occidentale* system appeared to alter relative species dominance within root disease centres, which was principally realized as an increase in the relative dominance of incense cedar.

4 | DISCUSSION

By comparing relative impacts and changes in canopy dominance in three California forest types impacted by *Heterobasidion* root disease, we demonstrate persistent changes in forest structure and composition on a period extending at least 49 years. The extent of mortality, measured as change in density or basal area, as well as the multi-decadal duration of these impacts help to reveal the persistence and intensity of Heterobasidion root disease impacts to forest health in California. In conjunction with previous studies demonstrating the regional scale of disease distribution, these data help contextualize Heterobasidion root disease as a

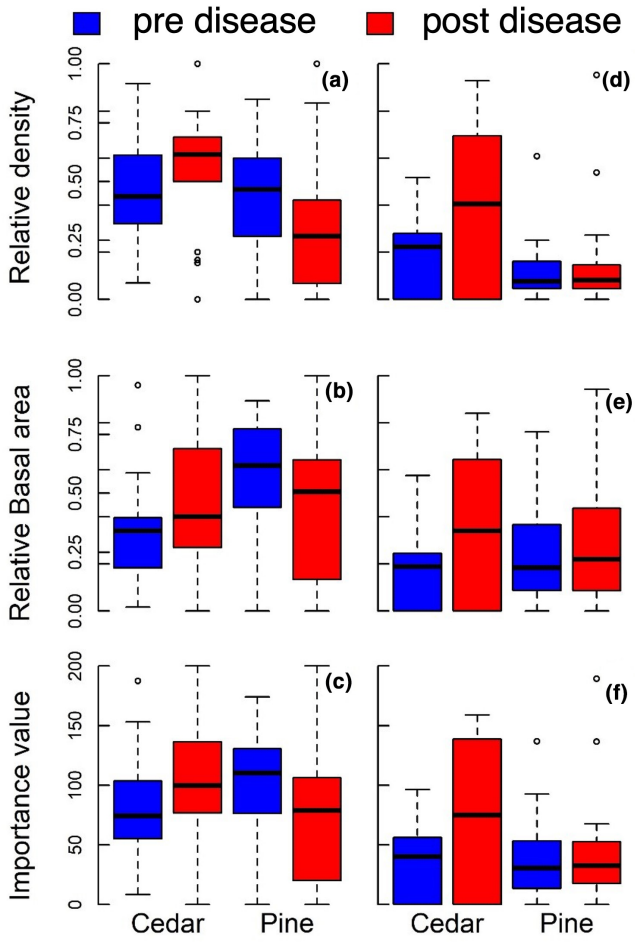

FIGURE 4 Standard boxplots identifying the median (black line) and points 1.5 times outside the interquartile range (outliners) for contrasting responses of Incense cedar (*Calocedrus decurrens*, 'Cedar') and *Pinus* ('Pine') species nearly 50 years after Heterobasidion root disease emergence in California. In the multi-species *Heterobasidion irregulare* system of Yosemite both are hosts (a–c) in contrast to the *H. occidentale* system where both are non-hosts (d–f). Pre-disease data are a reconstruction of stand structure within a few years of disease emergence.

forest health challenge: impacts persist for at least half a century, often result in locally severe host mortality, and appear to be accompanied by shifts in species composition in the most canopy diverse *H. occidentale* forest type (Garbelotto et al., 1997; James & Cobb, 1984; Rizzo et al., 2000).

In each of our study systems, disease impacts increased over the span of decades and persist on the ~50-year duration of our measurements (Rizzo et al., 2000; Slaughter & Parmeter, 1995; Figure 2). Like other root disease pathogens which attack large woody root systems, *Heterobasidion* pathogens create a 'disease of the site' causing long-term changes in stand composition, structure, and diversity (Baumgartner & Rizzo, 2001; Gómez-Aparicio et al., 2012). These patterns suggest the full timeframe of Heterobasidion root disease impacts in California forests could for more than 50 years in the absence of other transformative disturbance, such as wildfire. Even with a cessation of pathogen activity in the near term, changes in forest structure may be detectable decades in the future for these forests (Dolanc et al., 2014). Shifts in species dominance, which occurred in the *H. occidentale* system where non-hosts were relatively abundant, are expected to persist even longer as incense cedar are long-lived trees. Thus, our results show how native biological agents can contribute to long-term vegetation changes across the major forest types of the region, a pattern previously documented in response to changing wildfire dynamics and emergence of invasive pathogens (Cobb et al., 2020; Dudney et al., 2020; McIntyre et al., 2015). Shifts in species dominance, which occurred in the *H. occidentale* system where non-hosts were relatively abundant, are expected to persist even longer as incense cedar are long-lived trees. Given that *H. irregulare* establishment is often facilitated by logging, this disease can be emergent with changes in management (Garbelotto & Gonthier, 2013; Poloni et al., 2021; Slaughter & Rizzo, 1999). In contrast, *H. occidentale* may be more directly dependent on direct infection of tree boles, suggesting that diseases caused by this pathogen could become emergent with fire suppression policies, which favours the relatively fire intolerant *A. concolor* and *A. magnifica* hosts, selective logging of pine in these systems, silviculture treatments, such as thinning, aimed at increasing *Abies* dominance, or a combination of one or more of these factors (Dolanc et al., 2014; Garbelotto & Gonthier, 2013; McIntyre et al., 2015; Otrosina & Garbelotto, 2010).

Secondary spread of *Heterobasidion* pathogens occurs via tree-to-tree root contacts, and hence, infection in larger trees can be an important driver of disease centre extent (Rizzo et al., 2000). Furthermore, a study quantifying genets of *Heterobasidion* in true fir showed genet size increases with the size of the tree, which initiated the disease centre, suggesting that stand-level inoculum may be influenced by the size of the tree which initiated the disease centre, size class distribution, or basal area directly (Garbelotto et al., 1999). The relationship between infected tree size and disease extent is consistent with other root diseases where the degree of contact with inoculum reservoirs (such as buried roots) determines disease risk (Cunniffe & Gilligan, 2011; Kauffman & Jules, 2006; Morrison et al., 2014). We did not investigate within-stand spatial structure and its potential influence on disease severity, although the substantial variation in disease centre area across the host–pathogen systems (Figure 2) suggests that this line of investigation could help elucidate disease patterns at broader spatial scales (Garbelotto et al., 1997; Preisler & Slaughter, 1997; Rizzo et al., 2000).

In both *H. irregulare* systems, non-hosts are a relatively trivial component of stand composition (see also Dolanc et al., 2014) and host dominance remains similar between pre- and post-disease conditions even though total basal area and density are substantially reduced (Figures 2 and 3). For the Eastside pine plot network, the abundance of non-hosts is so minimal that this system is akin to a negative control as changes in tree species composition with disease are extremely unlikely. We expected that species shifts in the Yosemite plot network could occur given that host mortality rates were significantly faster in pine versus incense cedar hosts (Rizzo et al., 2000). While we found a weak trend of shifts in composition to

incense cedar, these shifts were not significant, suggesting that any compositional shifts are not apparent after ~50 years. For both *H. irregulare* systems, the size of the disease centre and proportional loss of density or basal area are defining characteristics by which disease impacts are quantified (Figure 2). In contrast, non-host dominance increased in the *H. occidentale* disease system (Table 2; Figure 4). Specifically, we found statistically significant increases in incense cedar occurred for each measure of dominance, including importance (Table 2). Thus, in contrast to our two examples of *H. irregulare*, to fully capture the impacts of disease caused by *H. occidentale* in these white and red fir host communities, one should also consider the initial prevalence of non-hosts and the potential for increases in these species in addition to disease centre size and proportional loss of basal area and density (Figures 2 and 3).

In our fir plot network, incense cedar importance value was negatively associated with disease centre area (Table 2). Given that incense cedar is not a host in the *H. occidentale* system, it is plausible that this tree would act to limit root-to-root transmission among neighbouring susceptible hosts and thus create a negative feedback on disease expansion (Demchik et al., 2020; cf. Haas et al., 2011; Hantsch et al., 2014). While our data hint at the potential for a dilution dynamic, we stress this is speculation, which requires careful follow-up before it could be trusted or used in a management context. Of note, incense cedar dominance also increased in *H. occidentale* disease centres (Figure 4). This finding is worth emphasizing as incense cedar has increased at the regional scale during the last century (Dolanc et al., 2014; McIntyre et al., 2015).

Increased incense cedar dominance in *H. occidentale* disease centres is noteworthy because this species has increased regionally, including substantial increases across the Sierra Nevada and southern Cascade ranges during the last century (Dolanc et al., 2014; McIntyre et al., 2015). The measured changes in incense cedar dominance at the regional scale are almost certainly also influenced by other region-scale issues including fire suppression, insect outbreaks, and increasingly intense drought (Dolanc et al., 2014; Healey et al., 2016; Williams et al., 2022). However, pathogen-mediated competition has been demonstrated in other emergent forest diseases and increased incense cedar dominance where it is a non-host—in the *H. occidentale* system—is consistent with the expected effects of this ecological dynamic (Cobb et al., 2010), suggesting further attention to this is justified. If *H. occidentale* leads to increased dominance of incense cedar over the decades-long window of impact for the disease caused by *H. occidentale*, it is a virtual certainty that this could enhance or conflict with management goals at the stand level. A larger-scale survey of Heterobasidion root disease centre frequency would help determine if this regional shift is enhanced by disease, especially considering the potential for negative feedback between incense cedar and *H. occidentale* disease centre area. Regional increases in incense cedar are almost certainly not solely disease driven at these larger spatial extents. So long as this is the case, the patterns we report here would be restricted to, but likely still be relevant for, local or stand-level objectives.

Landscape-level analysis of root disease outside of the Sierra Nevada suggests that reductions in carbon storage at the stand-scale can propagate to the landscape-to-regional spatial extents (Fei et al., 2019; Healey et al., 2016; Meentemeyer et al., 2012); thus, these diseases may also be consequential in terms of regional carbon sequestration and atmospheric carbon storage in California. Given the widespread occurrence of both *Heterobasidion* species we focus on here and their long-lasting stand impacts (Figure 2), *Heterobasidion*-driven changes to landscape-level carbon sequestration are plausible and could influence a range of management decisions including those for other tree mortality agents and wildfire. Regardless of changes in dominance, declines in host density and basal area were a clear pathogen impact for all three host–pathogen systems we studied (Figure 2). Although Heterobasidion root disease expansion appears to slow over time (see Rizzo et al., 2000), the long duration of the disease increases the likelihood that pathogen-weakened trees will also experience other disturbances and may be more likely to succumb to drought, insect attack, and/or wildfire (Cobb, 2022; Fettig et al., 2007; Maloney et al., 2008). Drought in particular is a persistent and increasingly acute driver of tree mortality in California and much of the western United States (Clark et al., 2016; Hartmann et al., 2015; Millar & Stephenson, 2015).

Heterobasidion root disease is distinguished from drought, and many biological agents of mortality in that stump-level establishment can be largely prevented for both *H. occidentale* and *H. irregulare* by applying one of several inexpensive and environmentally safe chemical or biological control treatments to recently cut stumps in areas where Heterobasidion root disease is not already present (Blomquist et al., 2020; Oliva et al., 2008; Poloni et al., 2021). *Heterobasidion* preventive measures applied following fuel reduction, commercial timber harvest, or other stand improvement treatments could be valuable interventions when combined with thinning aimed at addressing drought stress or bark beetle attack (Fettig et al., 2007; Poloni et al., 2021). Given the long duration, locally severe mortality, and—in at least one host–pathogen system—shifts in species composition associated with these pathogens, it is clear that Heterobasidion root diseases co-occur and can potentially interact with the other pressing forest health concerns facing managers in these true fir and pine forests of California (Cobb, 2022; Millar & Stephenson, 2015; Williams et al., 2022). An approach to mitigate the effects of Heterobasidion root disease in the context of persistent forest health concerns may be to manage species composition through selective thinning and replanting to maintain forest diversity. A strong argument can be made for integrating preventative Heterobasidion root disease treatments into forest health policy and management where recent logging operations have occurred. Maintaining the diversity of both structure and composition will ensure that there will be minimal loss of economic and ecological forest benefits. Limiting the spread of Heterobasidion root disease will likely also reduce defect in production forests leading to higher return on investment in plantations, especially in true fir systems where the pathogen can cause rot and wetwood in the lower bole (Shaw et al., 1995). While management of Heterobasidion root disease gaps could prevent tree mortality which emerges over decades, it is important to

acknowledge that the spatial heterogeneity created by canopy openings and other features such as snag habitat critical to some small mammals are often consistent with management objectives (Ostry & Laflamme, 2008). As Heterobasidion root disease continues to affect California forests in the context of climate change, forests will benefit from management methods that balance the negative pathogenic impact of Heterobasidion root disease while acknowledging the ecological services it provides in the context of these forests.

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DATA AVAILABILITY STATEMENT

Research data are not shared.

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REFERENCES

Barrett, T. M., & Robertson, G. C. (2021). *Disturbance and sustainability in forests of the Western United States*. Gen. Tech. Rep. PNW-GTR-992 (p. 992). U.S. Department of Agriculture, Forest Service, Pacific Northwest Research Station.

Baumgartner, K., & Rizzo, D. M. (2001). Ecology of Armillaria spp. in mixed-hardwood forests of California. *Plant Disease*, *85*, 947–951.

Blomquist, M., Larsson Herrera, S., Hofmann, J., Beram, R. C., Cleary, M., & Rönnberg, J. (2020). Size matters but is big always better? Effectiveness of urea and *Phlebiopsis gigantea* as treatment against Heterobasidion on *Picea abies* stumps of variable size. *Forest Ecology and Management*, *462*, 117998.

Clark, J. S., Iverson, L., Woodall, C. W., Allen, C. D., Bell, D. M., Bragg, D. C., D'Amato, A. W., Davis, F. W., Hersh, M. H., Ibanez, I., Jackson, S. T., Matthews, S., Pederson, N., Peters, M., Schwartz, M. W., Waring, K. M., & Zimmermann, N. E. (2016). The impacts of increasing drought on forest dynamics, structure, and biodiversity in the United States. *Global Change Biology*, *22*, 2329–2352.

Cobb, R. C. (2022). *The intertwined problems of wildfire, forest disease, and climate change interactions*. Current Forestry Reports.

Cobb, R. C., Haas, S. E., Kruskamp, N., Dillon, W. W., Swiecki, T. J., Rizzo, D. M., Frankel, S. J., & Meentemeyer, R. K. (2020). The magnitude of regional-scale tree mortality caused by the invasive pathogen *Phytophthora ramorum*. *Earth's Future*, *8*, e2020EF001500.

Cobb, R. C., Meentemeyer, R. K., & Rizzo, D. M. (2010). Apparent competition in canopy trees determined by pathogen transmission rather than susceptibility. *Ecology*, *91*, 327–333.

Cobb, R. C., Ruthrof, K. X., Breshears, D. D., Lloret, F., Aakala, T., Adams, H. D., Anderegg, W. R. L., Ewers, B. E., Galiano, L., Grünzweig, J. M., Hartmann, H., Huang, C., Klein, T., Kunert, N., Kitzberger, T., Landhäusser, S. M., Levick, S., Preisler, Y., Suarez, M. L., … Zeppel, M. J. B. (2017). Ecosystem dynamics and management after forest die-off: A global synthesis with conceptual state-and-transition models. *Ecosphere*, *8*, e02034.

Cunniffe, N. J., & Gilligan, C. A. (2011). A theoretical framework for biological control of soil-borne plant pathogens: Identifying effective strategies. *Journal of Theoretical Biology*, *278*, 32–43.

Demchik, M. C., Sakalidis, M. L., Hillstrom, M., Scanlon, K., Adams, T. A., & Minnix, K. M. (2020). Evaluating regeneration in Heterobasidion root disease infested stands in the lake states. *Forest Science*, *66*, 141–144.

Dolanc, C. R., Safford, H. D., Dobrowski, S. Z., & Thorne, J. H. (2014). Twentieth century shifts in abundance and composition of vegetation types of the Sierra Nevada, CA, US. *Applied Vegetation Science*, *17*, 442–455.

Dudney, J. C., Nesmith, J. C. B., Cahill, M. C., Cribbs, J. E., Duriscoe, D. M., Das, A. J., Stephenson, N. L., & Battles, J. J. (2020). Compounding effects of white pine blister rust, mountain pine beetle, and fire threaten four white pine species. *Ecosphere*, *11*, e03263.

Fei, S., Morin, R. S., Oswalt, C. M., & Liebhold, A. M. (2019). Biomass losses resulting from insect and disease invasions in US forests. *Proceedings of the National Academy of Sciences*, *116*, 17371–17376.

Fettig, C. J., Klepzig, K. D., Billings, R. F., Munson, A. S., Nebeker, T. E., Negrón, J. F., & Nowak, J. T. (2007). The effectiveness of vegetation management practices for prevention and control of bark beetle infestations in coniferous forests of the western and southern United States. *Forest Ecology and Management*, *238*, 24–53.

Fettig, C. J., Mortenson, L. A., Bulaon, B. M., & Foulk, P. B. (2019). Tree mortality following drought in the central and southern Sierra Nevada, California, U.S. *Forest Ecology and Management*, *432*, 164–178.

Garbelotto, M., Cobb, F. W., Bruns, T. D., Otrosina, W. J., Popenuck, T., & Slaughter, G. (1999). Genetic structure of *Heterobasidion annosum* in white fir mortality centers in California. *Phytopathology*, *89*, 546–554.

Garbelotto, M., & Gonthier, P. (2013). Biology, epidemiology, and control of Heterobasidion species worldwide. *Annual Review of Phytopathology*, *51*, 39–59.

Garbelotto, M., Slaughter, G., Popenuck, T., Cobb, F. W., & Bruns, T. D. (1997). Secondary spread of *Heterobasidion annosum* in white fir root-disease centers. *Canadian Journal of Forest Research*, *27*, 766–773.

Gómez-Aparicio, L., Ibáñez, B., Serrano, M. S., De Vita, P., Ávila, J. M., Pérez-Ramos, I. M., García, L. V., Esperanza Sánchez, M., & Marañón, T. (2012). Spatial patterns of soil pathogens in declining Mediterranean forests: Implications for tree species regeneration. *New Phytologist*, *194*, 1014–1024.

Haas, S. E., Hooten, M. B., Rizzo, D. M., & Meentemeyer, R. K. (2011). Forest species diversity reduces disease risk in a generalist plant pathogen invasion. *Ecology Letters*, *14*, 1108–1116.

Hantsch, L., Bien, S., Radatz, S., Braun, U., Auge, H., & Bruelheide, H. (2014). Tree diversity and the role of non-host neighbour tree species in reducing fungal pathogen infestation. *Journal of Ecology*, *102*, 1673–1687.

Hartmann, H., Adams, H. D., Anderegg, W. R. L., Jansen, S., & Zeppel, M. J. B. (2015). Research frontiers in drought-induced tree mortality: Crossing scales and disciplines. *New Phytologist*, *205*, 965–969.

Hawkins, A. E., & Henkel, T. W. (2011). Native forest pathogens facilitate persistence of Douglas-fir in old-growth forests of northwestern California. *Canadian Journal of Forest Research*, *41*, 1256–1266.

Healey, S. P., Raymond, C. L., Lockman, I. B., Hernandez, A. J., Garrard, C., & Huang, C. (2016). Root disease can rival fire and harvest in reducing forest carbon storage. *Ecosphere*, *7*, e01569.

Holt, R. D., & Lawton, J. H. (1994). The ecological consequences of shared natural enemies. *Annual Review of Ecology and Systematics*, *25*, 495–520.

James, R. L., & Cobb, F. W. J. (1984). Spore deposition by *Heterobasidion annosum* in forests of California [mixed-conifer forests, Stanislaus National Forest, Jackson State Forest, pollution effect on spore deposition]. *Plant Disease*, *68*, 246–248.

Johnstone, J. F., Allen, C. D., Franklin, J. F., Frelich, L. E., Harvey, B. J., Higuera, P. E., Mack, M. C., Meentemeyer, R. K., Metz, M. R., Perry, G. L., Schoennagel, T., & Turner, M. G. (2016). Changing disturbance regimes, ecological memory, and forest resilience. *Frontiers in Ecology and the Environment*, *14*, 369–378.

Kauffman, M. J., & Jules, E. S. (2006). Heterogeneity shapes invasion: Host size and environment influence susceptibility to a nonnative pathogen. *Ecological Applications*, *16*, 166–175.

Lalande, B. M., Hughes, K., Jacobi, W. R., Tinkham, W. T., Reich, R., & Stewart, J. E. (2020). Subalpine fir mortality in Colorado is associated with stand density, warming climates and interactions among fungal diseases and the western balsam bark beetle. *Forest Ecology and Management*, *466*, 118133.

Maloney, P. E., Smith, T. F., Jensen, C. E., Innes, J., Rizzo, D. M., & North, M. P. (2008). Initial tree mortality and insect and pathogen response to fire and thinning restoration treatments in an old-growth mixed-conifer forest of the Sierra Nevada, California. *Canadian Journal of Forest Research*, *38*, 3011–3020.

McIntyre, P. J., Thorne, J. H., Dolanc, C. R., Flint, A. L., Flint, L. E., Kelly, M., & Ackerly, D. D. (2015). Twentieth-century shifts in forest structure in California: Denser forests, smaller trees, and increased dominance of oaks. *Proceedings of the National Academy of Sciences*, *112*, 1458–1463.

Meentemeyer, R. K., Haas, S. E., & Václavík, T. (2012). Landscape epidemiology of emerging infectious diseases in natural and human-altered ecosystems. *Annual Review of Phytopathology*, *50*, 379–402.

Millar, C. I., & Stephenson, N. L. (2015). Temperate forest health in an era of emerging megadisturbance. *Science*, *349*, 823–826.

Morrison, D. J., Cruickshank, M. G., & Lalumière, A. (2014). Control of laminated and Armillaria root diseases by stump removal and tree species mixtures: Amount and cause of mortality and impact on yield after 40 years. *Forest Ecology and Management*, *319*, 75–98.

Nguyen, H., Lamb, D., Herbohn, J., & Firn, J. (2015). Equations to calculate important value index (IVI) of species. *PLoS ONE*, *9*(4).

Oliva, J., Samils, N., Johansson, U., Bendz-Hellgren, M., & Stenlid, J. (2008). Urea treatment reduced *Heterobasidion annosum* s.l. root rot in *Picea abies* after 15 years. *Forest Ecology and Management*, *255*, 2876–2882.

Ostry, M. E., & Laflamme, G. (2008). Fungi and diseases—Natural components of healthy forests. *Botany*, *87*, 22–25.

Otrosina, W. J., & Garbelotto, M. (2010). *Heterobasidion occidentale* sp. nov. and *Heterobasidion irregulare* nom. nov.: A disposition of North American Heterobasidion biological species. *Fungal Biology*, *114*, 16–25.

Poloni, A. L., Garbelotto, M., Lee, C. A., & Cobb, R. C. (2021). Efficacy of chemical and biological stump treatments for the control of *Heterobasidion occidentale* infection of California *Abies concolor*. *Pathogens*, *10*, 1390.

Preisler, H. K., Grulke, N. E., Heath, Z., & Smith, S. L. (2017). Analysis and out-year forecast of beetle, borer, and drought-induced tree mortality in California. *Forest Ecology and Management*, *399*, 166–178.

Preisler, H. K., & Slaughter, G. W. (1997). A stochastic model for tree survival in stands affected by Annosum root disease. *Forest Science*, *43*, 78–86.

Reinhart, K. O., Royo, A. A., Kageyama, S. A., & Clay, K. (2010). Canopy gaps decrease microbial densities and disease risk for a shade-intolerant tree species. *Acta Oecologica*, *36*, 530–536.

Rizzo, D. M., & Slaughter, G. W. (2001). Root disease and canopy gaps in developed areas of Yosemite Valley, California. *Forest Ecology and Management*, *146*, 159–167.

Rizzo, D. M., Slaughter, G. W., & Parmeter, J. R., Jr. (2000). Enlargement of canopy gaps associated with a fungal pathogen in Yosemite Valley, California. *Canadian Journal of Forest Research*, *30*, 1501–1510.

Shaw, D. C., Edmonds, R. L., Littke, W. R., Browning, J. E., & Russel, K. W. (1995). Incidence of wetwood and decay in precommercially thinned western hemlock stands. *Canadian Journal of Forest Research*, *25*, 1269–1277.

Slaughter, G., & Rizzo, D. (1999). Past forest management promoted root disease in Yosemite Valley. *California Agriculture*, *53*, 17–24.

Slaughter, G. W., & JR, P., Jr. (1989). Annosus root disease in true firs in northern and central California national forests. *Proceedings of the symposium on research and management of annosus root disease* (pp. 70–77).

Slaughter, G. W., & Parmeter, J. R. (1995). Enlargement of tree-mortality centers surrounding pine stumps infected by *Hetetobasidionannosum* in northeastern California. *Canadian Journal of Forest Research*, *25*, 244–252.

Williams, A. P., Abatzoglou, J. T., Gershunov, A., Guzman-Morales, J., Bishop, D. A., Balch, J. K., & Lettenmaier, D. P. (2019). Observed impacts of anthropogenic climate change on wildfire in California. *Earth's Future*, *7*, 892–910.

Williams, A. P., Cook, B. I., & Smerdon, J. E. (2022). Rapid intensification of the emerging southwestern North American megadrought in 2020–2021. *Nature Climate Change*, *12*, 242–234.

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