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Sporulation Potential of *Phytophthora ramorum* Differs Among Common California Plant Species in the Big Sur Region

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Abstract

Sudden oak death (SOD), caused by the generalist pathogen *Phytophthora ramorum*, has profoundly impacted California coastal ecosystems. SOD has largely been treated as a two-host system, with *Umbellularia californica* as the most transmissive host, *Notholithocarpus densiflorus* less so, and remaining species as epidemiologically unimportant. However, this understanding of transmission potential primarily stems from observational field studies rather than direct measurements on the diverse assemblage of plant species. Here, we formally quantify the sporulation potential of common plant species inhabiting SOD-endemic ecosystems on the California coast in the Big Sur region. This study allows us to better understand the pathogen's basic biology, trajectory of SOD in a changing environment, and how the entire host community contributes to disease risk. Leaves were inoculated in a controlled laboratory environment and assessed for production of sporangia and chlamydospores, the infectious and resistant propagules, respectively. *P. ramorum* was capable of infecting every species in our study and almost all species produced spores to some extent. Sporangia production was greatest in *N. densiflorus* and *U. californica* and the difference was insignificant. Even though other species produced much less, quantities were nonzero. Thus, additional species may play a previously unrecognized role in local transmission. Chlamydospore production was highest in *Acer macrophyllum* and *Ceanothus oliganthus*, raising questions about the role they play in pathogen persistence. Lesion size did not consistently correlate with the production of either sporangia or chlamydospores. Overall, we achieved an empirical foundation to better understand how community composition affects transmission of *P. ramorum*.

Keywords: competency, leaf dip assay, leaf disc assay, sudden oak death, transmission potential

Emergent diseases operate in dynamic and complex ecological communities of multiple interacting hosts and pathogens (Johnson et al. 2015b). The vast majority of human, wildlife, and plant diseases are caused by multihost pathogens (Woolhouse et al. 2001) and as a result, control strategies often require detailed knowledge about many host–pathogen interactions. Gathering information on host susceptibility and infectiousness (i.e., host competency) is important in order to inform targeted strategies for management in new or changing environments.

Sudden oak death (SOD) is a prominent forest disease caused by the generalist invasive oomycete pathogen *Phytophthora ramorum*, which has profoundly impacted California coastal ecosystems since its initial observations in the mid-1990s (Garbelotto et al. 2003; Rizzo and Garbelotto 2003). Symptoms can be expressed as lethal canker infections, primarily on tanoak (*Notholithocarpus densiflorus*) and a subset of true oak species (*Quercus* spp.), and nonlethal foliar and twig lesions (Rizzo et al. 2005). Field measurements among three dominant species showed that bay laurel (*Umbellularia californica*) produced the greatest concentration of *P. ramorum* inoculum in the form of infectious sporangia, tanoak produced significantly fewer, and no sporangia were recovered from coast live oak (*Q. agrifolia*) (Davidson et al. 2005, 2008). Naturally infected redwood (*Sequoia sempervirens*) foliage was incubated in the laboratory and found to occasionally produce sporangia, but the concentrations were not quantified (Davidson et al. 2008). Although tests for sporulation on additional California species have not been published, field observations suggested that other common plant species either contribute very little or not at all to transmission (Grünwald et al. 2019; Meentemeyer et al. 2004). Thus, SOD has largely been treated as a two-host system, with bay laurel being the most transmissive host, tanoak less so, and all remaining species as noninfectious. This conventional knowledge has formed the foundation for disease risk maps (Meentemeyer et al. 2008), management plans (Cobb et al. 2013, 2017), and many ecological studies (e.g., Cobb et al. 2010, 2012; Dillon and Meentemeyer 2019).

The goal of this study is to formally quantify the inoculum production potential of common *P. ramorum* hosts from California coastal forests. While much has been learned about the disease in the absence of such a comprehensive survey, we are motivated for several reasons. First, we are filling in the gaps of the pathogen's basic biology. Multiple studies have already assessed the competencies of host species outside of California as a result of the pathogen's potential to cause epidemics in new locations (Harris and Webber 2016; Hüberli et al. 2008; Ireland et al. 2012; Jinek et al. 2011; Linderman and Davis 2007; Tooley and Browning 2009), but the same has yet to be done for the region most severely affected by the disease.

Quantifying sporulation potentials also allows us to understand more about the trajectory of SOD in a changing environment. In addition to possible genetic changes in the pathogen, variation in either host or climatic variables will influence its epidemiology. For example, increased fuel loads created by SOD-induced mortality interact with wildfires to increase fire severity (Metz et al. 2011). This leads to increased mortality of aboveground stems (Metz et al. 2013) and belowground genets (Simler et al. 2018), but also increased opportunity for sexual regeneration for some species in a forest dominated by asexually reproducing species (Simler-Williamson et al. 2019). The fire–disease interaction presents an interesting opportunity for compositional shifts to take place in novel gaps. Moreover, climate change is expected to shift the geographic ranges of host species, as well as alter the distribution of landscapes with favorable growth conditions for the pathogen (Meentemeyer et al. 2011). Overall, uncertainty in the future's forest composition and the distribution of

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the pathogen provokes newfound reason to understand the sporulation potential of species beyond what is conventionally understood.

Finally, this study will generate a more nuanced perspective of how the entire host community may contribute to disease risk. Current epidemiological models have typically only considered bay laurel and tanoak as transmissive hosts (Cobb et al. 2012; Ndeffo Mbah and Gilligan 2010). These models are highly sensitive to minor increases in inoculum load (Cobb et al. 2012), implying that undetected low-competency hosts may undermine optimal management strategies (Ndeffo Mbah and Gilligan 2010). The role of nonhost species must also not be disregarded; after accounting for the densities of bay laurel and tanoak, more diverse plant communities were associated with a lower probability of SOD infection (Haas et al. 2011, 2016). The mechanisms driving a negative relationship between diversity and disease risk are highly debated and speculated about (Halsey 2019; Johnson et al. 2015a; Rohr et al. 2020), but one suggestion is that nonhost species may lower transmission by reducing encounter rates between infected and susceptible individuals (Keesing et al. 2006). Data on sporulation potential can leverage nearly two decades of ecological data, making SOD an ideal model system to elucidate mechanisms driving the disease-diversity relationship involving a forest pathogen.

This study assesses the sporulation potential of common plant species inhabiting SOD-endemic ecosystems on the California coast. The intent of this study is to estimate the interspecific variation in sporulation potential. In order to control for phenological differences that would manifest in a statewide survey (Dodd et al. 2008), we focused our sampling efforts on the Big Sur region, where we use a network of 280 long-term monitoring plots to identify the top 10 most common plant species in both redwood and mixed evergreen forests (Metz et al. 2011, 2012). Leaf tissue from each species was inoculated in a controlled laboratory environment and assessed for production of its two spore types, sporangia and chlamydospores. Sporangia and their released zoospores are the pathogen's infectious propagules and thick-walled chlamydospores might facilitate long-term survival in the soil (Fichtner et al. 2007), but consensus on their epidemiological role is still unclear. Specifically, we address the following questions: (i) How do plant species vary in sporangia and chlamydospore production? (ii) Is lesion area positively correlated with spore production?

Materials and Methods

Field sampling. The forests in Big Sur broadly split into two types, one composed of mixed evergreen species and the other with a similar composition but dominated by redwood (Metz et al. 2012). The three most common species in mixed evergreen forests are bay laurel, coast live oak, and tanoak; in redwood forests they are redwood, tanoak, and bay laurel (Metz et al. 2012). We inoculated leaf tissue from the top 10 most ubiquitous plant species in our plot network (measured as the number of plots in which each species occurs) for both forest types (Table 1). Plant samples were collected in the Big Sur area from Landels-Hill Big Creek Reserve (36.070° N, 121.599° W) and nearby private property in May and early June 2019, the seasonal period with greatest sporulation (Davidson et al. 2005, 2008). For each species, we collected healthy looking, fully flushed leaves from 32 individuals. They were rinsed with deionized water, placed in moist plastic bags inside of a cooler, and refrigerated overnight. Inoculations were conducted the following day using *P. ramorum* isolate PR979 (GenBank accession MN783356), which was originally isolated in 2012 from a bay laurel tree located within the plot network.

Experimental design. Separate experiments were conducted for the broadleaf and conifer species in May and June, respectively. We inoculated leaf discs cut with a cork borer (size 9, 1.25 cm in diameter) from broadleaf species (Widmer 2015), but since cutting discs from conifer needles is not possible, entire shoots or needles were inoculated using a detached leaf dip method (Denman et al. 2005). For the broadleaf species, two leaf discs from each of the 32 individuals were inoculated and quantified for either sporangia or chlamydospores. An additional two leaf discs cut from 10 or 32 individuals were used as controls (32 controls were used for tanoak, oak species, and bay laurel; 10 controls were used for all other species). Controls were inoculated with sterile water and spores were quantified to assess the presence of the naturally occurring *Phytophthora* pathogen. In order to limit intraindividual variation, all leaf discs from an individual were cut from the same leaf when possible. For the conifer species, two shoots or needle fascicles from 32 individuals were inoculated with either sporangia or sterile water and the replicates were subsequently sampled for both sporangia and chlamydospore counts. Although it is more difficult to directly compare species responses from different assays, we decided to inoculate the majority of species using leaf discs because we were more able to limit sampling and methodological error. The leaf disc assay allowed us to scale up replication, which was necessary to detect statistically significant differences among many species, and inoculum was more consistently applied on the flat leaf discs. We attempted to compare results from the two inoculation methods by also performing the leaf dip assays on 10 individuals of bay laurel and tanoak. Overall, leaves from 317 and 106 individuals were used in the leaf disc and leaf dip assays, respectively.

Inoculations. To produce inoculum, the *P. ramorum* isolate was passaged through a rhododendron leaf to ensure pathogenicity and grown on 20% unclarified V8 agar for 7 days at 20°C under natural light. Cultures were flooded with sterile water and incubated under the same conditions. After 48 h, we scraped the colonies to dislodge the sporangia. Sporangia suspensions were rinsed with sterile water and filtered with four-ply cheesecloth. The concentration was estimated with a hemocytometer and adjusted to 4,000 sporangia/ml (Tooley and Browning 2009).

Following a modified protocol from Widmer (2015), leaf discs were placed into quartered Petri dishes and 15 µl of inoculum was pipetted onto the underside surface. In order to maintain high relative humidity, the Petri dishes were closed with lids lined with sterilized Whatman No. 1 filter paper dampened with 150 µl of sterile water and placed inside of moist clear plastic containers. The leaf discs were incubated under natural light at 20°C and were removed after 5 days for quantification of sporangia, chlamydospores, and lesion area. To estimate a minimum baseline of sporangia originating from the inoculum source alone, we also incubated eight drops of the sporangia solution without a host.

Detached leaves were held at the petioles, submerged in inoculum to a depth of 4 cm for 30 s, and placed inside a moisture chamber with the underside down (Denman et al. 2005). Each chamber contained 6 to 12 leaves of the same treatment depending on available space. Leaves sat on a sterilized metal mesh platform contained in clear plastic containers lined with moist paper towels. The leaves were incubated under the same conditions as the leaf discs for 5 days. After 48 h, we gently rinsed the control and treatment leaves with running distilled water to ensure that recovered sporangia were produced postinoculation, rather than from residual inoculum.

Assessments of lesion area and spore production. We estimated lesion area by photographing the underside of all leaf discs and measuring the area of visible necrosis with the ImageJ software program (version 10.2; National Institutes of Health). We counted sporangia by selecting half of the samples, adding 100 µl of water to each disc, and scraping with the flat side of a flame-sterilized T-pin 20 times on the underside and 10 times on the topside (modified from Ireland et al. 2012). We pipetted the dislodged sporangia into 0.2-ml strip tubes, added 2.5 µl of lactophenol cotton blue (LPCB) solution (a killing agent and fungal preservant), and stored them at 4°C for up to 3 months. Estimating sporangia production involved adding 5 µl of well-mixed sporangia solution onto a slide, counting all of the sporangia under a compound microscope, and repeating for a total of three times. The remaining half of the leaves were used for chlamydospore quantification. Since chlamydospores are typically embedded within the leaf tissue, we counted them directly on the leaves. The leaf discs were placed into 2-ml tubes of 1 M potassium hydroxide (KOH), which makes the leaf tissue transparent, and stored at room temperature for 2 to 4 weeks before counting under the compound microscope (Fichtner et al. 2009, 2012). For leaves with large quantities of chlamydospores (i.e., >200 spores), we used the micrometer in the microscope eyepiece reticle as a transect to sample a smaller proportion.

In order to process the leaves from the leaf dip assay in a similar manner, from the inoculated regions we randomly removed six needles from redwood and Douglas fir (*Pseudotsuga menziesii*) shoots, removed two of three needles from ponderosa pine (*Pinus ponderosa*), and cut two leaf discs with the cork borer from bay laurel and tanoak. Photographs were taken for assessments of lesion area. As described above, we added 100 µl of water to half of these samples, scraped the leaves, and collected the sporangia solution. Similarly, we added the other half of the samples to 2-ml tubes filled with KOH for chlamydospore counting.

Viability of the pathogen. To test the viability of the pathogen and detect any latent infections, we attempted to reisolate viable *P. ramorum* sporangia and mycelia from up to two replicates of symptomatic and asymptomatic leaves (delineated by the presence or absence of visible lesions) for each species-treatment group. Several species in the control group had no samples with lesions; conversely, many inoculated species exclusively had samples with lesions. To test sporangia viability, we dropped 10 µl of sporangia suspension onto oomycete-selective pimaricin-ampicillin-rifampicin-pentachloronitrobenzene agar medium (Jeffers and Martin 1986) before adding LPCB. Using these same samples, we tested the viability of mycelia by plating leaf tissue that was surface sterilized for 30 s in 10% bleach and rinsed in sterile water. We considered samples with recovered cultures to be successfully infected by a viable isolate. Plates were incubated at 20°C in the dark and monitored for mycelial growth for the following 3 to 4 days.

Data analysis. To analyze how the production of sporangia differed among species, we used separate Bayesian generalized linear mixed models for the leaf disc and leaf dip assays. We assumed a Poisson likelihood with a log link function and included species identity as a fixed effect predictor, individual identifier (ID) as a varying intercept because we subsampled from each individual three times for a more accurate estimate, and an offset variable (log-leaf area) to account for differences in leaf area in the leaf dip assay. Although the offset had no effect on the data coming from the leaf disc assay because the discs were equally sized, it allowed us to easily standardize the counts to spores per 1 cm^2.

Chlamydospore counts were overdispersed and, for several species, zero inflated. Thus, for both assays, a series of regular and zero-inflated Poisson and negative binomial models were compared using leave-one-out cross-validation (Vehtari et al. 2017) and differences were assessed with expected log pointwise predictive density (ELPD) (Vehtari et al. 2017). For the leaf disc assay, the best performing model was the zero-inflated negative binomial model with species as a predictor, an offset term (log-area sampled) to correct for different sampling intensities, and species predicting the zero-inflation probability (ΔELPD = −39.3, SE = 7.3). The leaf dip assay model had the same structure, except the zero-inflation probability was predicted by a single intercept (ΔELPD = −2.7, SE = 1.8).

For each of the above models, pairwise contrasts between species intercepts were performed on the models' posterior values. We report mean spore counts by back-transforming the posterior values of the species intercepts, given in units of sampled spores per square centimeter per microliter. We standardized sporangia counts to total sporangia per square centimeter by multiplying the values by 102.5 µl, the volume of liquid added to the sample. The chlamydospore values did not need additional transformations.

To examine whether spore production was associated with lesion area, regressions were run for the two spore types, but data from both assays were included together. For the sporangia regression, predictors included lesion area with slopes and intercepts varying by species, a dummy variable for assay, and individual ID as a varying intercept. The best performing chlamydospore model was a zero-inflated negative binomial model (ΔELPD = −19.9, SE = 7.9). Predictors included lesion area with slopes and intercepts varying by species, a dummy variable for assay, an offset term (log-area sampled), and species predicting the probability of zero inflation.

All models were performed using brms (Bürkner 2017), a package designed to fit Bayesian multilevel models with Stan programming language (Stan Development Team 2018), and were analyzed in the R environment (R Development Core Team 2019). We used weakly informative priors, four chains with 4,000 samples per chain (including 2,000 warmup samples), and chain convergence was assessed for each estimated parameter by ensuring Rhat values were ≤1.01 (Bürkner 2017). Species with spore count values of exclusively 0 were omitted from the analyses because they created convergence issues. Model fits were visually assessed by graphically comparing observed values against posterior predictive draws. Parameters were considered significant when the 90% highest posterior density interval did not cross zero.

Results

Viability tests. We detected no viable *P. ramorum* mycelia or sporangia in the controls. Most controls had no symptomatic samples, but those that did had lesions caused by nonoomycetes. In contrast, we reisolated viable *P. ramorum* mycelia from all species except for toyon (*Heteromeles arbutifolia*) (Table 2). Toyon was found to produce sporangia when inoculated, so it is possible that reisolation methods were not optimized for this species, as was the case for highly infectious larch species in the United Kingdom (Harris and Webber 2016). Most symptomatic samples had both viable mycelia and sporangia, and when there were asymptomatic samples available, many still had viable sporangia. This confirms that *P. ramorum* is capable of producing latent, transmissible infections.

Control samples. Of 424 control samples in the leaf disc and dip assays, no chlamydospores were found and only low quantities of sporangia were recovered in two samples. For these samples, we examined additional subsamples for a total of nine 5-µl counts, and

Table 1. The top 10 plant species for mixed evergreen and redwood forests

Species name	Species code[a]	Common name	Mixed evergreen rank[b]	Redwood rank
Umbellularia californica	UMCA or UMCA-D	Bay laurel	**1 (116)**	**3 (68)**
Quercus agrifolia	QUAG	Coast live oak	**2 (101)**	**7 (12)**
Notholithocarpus densiflorus	LIDE or LIDE-D	Tanoak	**3 (83)**	**2 (86)**
Arbutus menziesii	ARME	Madrone	**4 (73)**	**5 (18)**
Q. parvula	QUPA	Shreve's oak	**5 (56)**	**4 (26)**
Q. chrysolepis	QUCH	Interior live oak	**6 (52)**	14 (2)
Toxicodendron diversilobum	TODI	Poison oak	**7 (35)**	**9 (8)**
Heteromeles arbutifolia	HEAR	Toyon	**8 (29)**	12 (4)
Ceanothus oliganthus	CEOL	Ceanothus	**9 (25)**	**8 (10)**
Pinus ponderosa	PIPO	Ponderosa pine	**10 (16)**	NA (0)
Sequoia sempervirens	SESE	Redwood	21 (5)	**1 (111)**
Acer macrophyllum	ACMA	Bigleaf maple	13 (11)	**6 (14)**
Pseudotsuga menziesii	PSME	Douglas fir	18 (6)	**10 (6)**

[a] Because *U. californica* and *N. densiflorus* were used in the leaf disc and leaf dip assay, species codes for samples from the detached leaf dip assay are UMCA-D and LIDE-D, respectively.

[b] Rank values are based on the number of plots in which each species is present (in parentheses). Bold values indicate the top 10 plant species for mixed evergreen and redwood forests.

we detected one sporangium twice in a bay laurel individual and one sporangium once in a redwood individual. We therefore considered the background levels of *P. ramorum* on our collected leaves to be negligible and all following analyses were performed on the treatment replicates only.

Table 2. Results of the viability tests for the treatment group only (no viable cultures were recovered from the control group)

	Symptomatic		Asymptomatic	
Species[a]	Mycelia	Sporangia	Mycelia	Sporangia
ACMA	+	+	NA	NA
ARME	+	+	NA	NA
CEOL	+	−	+	−
HEAR	−	−	−	−
LIDE	+	+	−	+
LIDE-D	+	+	−	−
PIPO	-	−	+	−
PSME	+	−	+	−
QUAG	+	+	NA	NA
QUCH	+	+	+	+
QUPA	+	+	NA	NA
SESE	+	−	+	−
TODI	+	+	+	+
UMCA	+	+	+	+
UMCA-D	+	+	NA	NA

[a] Species are split by lesion presence (symptomatic and asymptomatic) and viability tests (mycelia and sporangia). Mycelia (indicative of viable infections) and sporangia are denoted by plus and minus signs, which indicate presence and absence, respectively. ACMA = *Acer macrophyllum*, NA = no sample, ARME = *Arbutus menziesii*, CEOL = *Ceanothus oliganthus*, HEAR = *Heteromeles arbutifolia*, LIDE = *Notholithocarpus densiflorus*, LIDE-D = *N. densiflorus* from the detached leaf assay, PIPO = *Pinus ponderosa*, PSME = *Pseudotsuga menziesii*, QUAG = *Quercus agrifolia*, QUCH = *Q. chrysolepis*, QUPA = *Q. parvula*, SESE = *Sequoia sempervirens*, TODI = *Toxicodendron diversilobum*, UMCA = *Umbellularia californica*. and UMCA-D = *U. californica* from the detached leaf assay.

Sporangia and chlamydospore production. Sporangia production differed among host plant species in the leaf disc assay. Some of the variation is attributed to random sampling error because we subsampled from the solutions three times; after accounting for this within-individual variation, we detected strong differences among species (Supplementary Tables S1 and S2). Bay laurel produced the greatest amount with a median ± 1 SD of 780 ± 175 sporangia/cm^2, closely followed by tanoak with 526 ± 114 sporangia/cm^2. The difference between these two species was not significant. The remaining eight broadleaf plant species produced significantly fewer sporangia (Fig. 1). All of these values except for that from madrone (*Arbutus menziesii*) were significantly higher than the estimated number of sporangia contained in the inoculum, indicating that almost all of these species were able to produce nonzero quantities of sporangia.

In the detached leaf assay, bay laurel and tanoak again produced significantly more sporangia than the other species, but they did not significantly differ between each other. On average, tanoak produced more than bay laurel with 264 ± 142 and 164 ± 114 sporangia/cm^2, respectively. These values are about two to four times lower than those from the leaf disc assay, suggesting that results from the detached leaf assay are relatively lower because of the methodology. Douglas fir and redwood produced similar quantities of sporangia, 70.7 ± 23.9 and 61.2 ± 18.6, respectively. Of the 96 subsamples from 32 individuals, we detected one sporangium from ponderosa pine, which conceivably could have been from residual inoculum that was not washed off after 48 h. Consequently, we consider the inoculum produced by ponderosa pine to be inconsequential.

Chlamydospore production also varied among plant species (Supplementary Table S1), but we were only able to confidently count spores from six broadleaf and two conifer species (Fig. 2). The leaf tissue of the true oak species, bay laurel, and redwood was too sclerotic for the KOH to adequately dissolve, making it difficult to visualize and identify the spores. For those that we could quantify, the median ± 1 SD quantities of chlamydospores produced per square

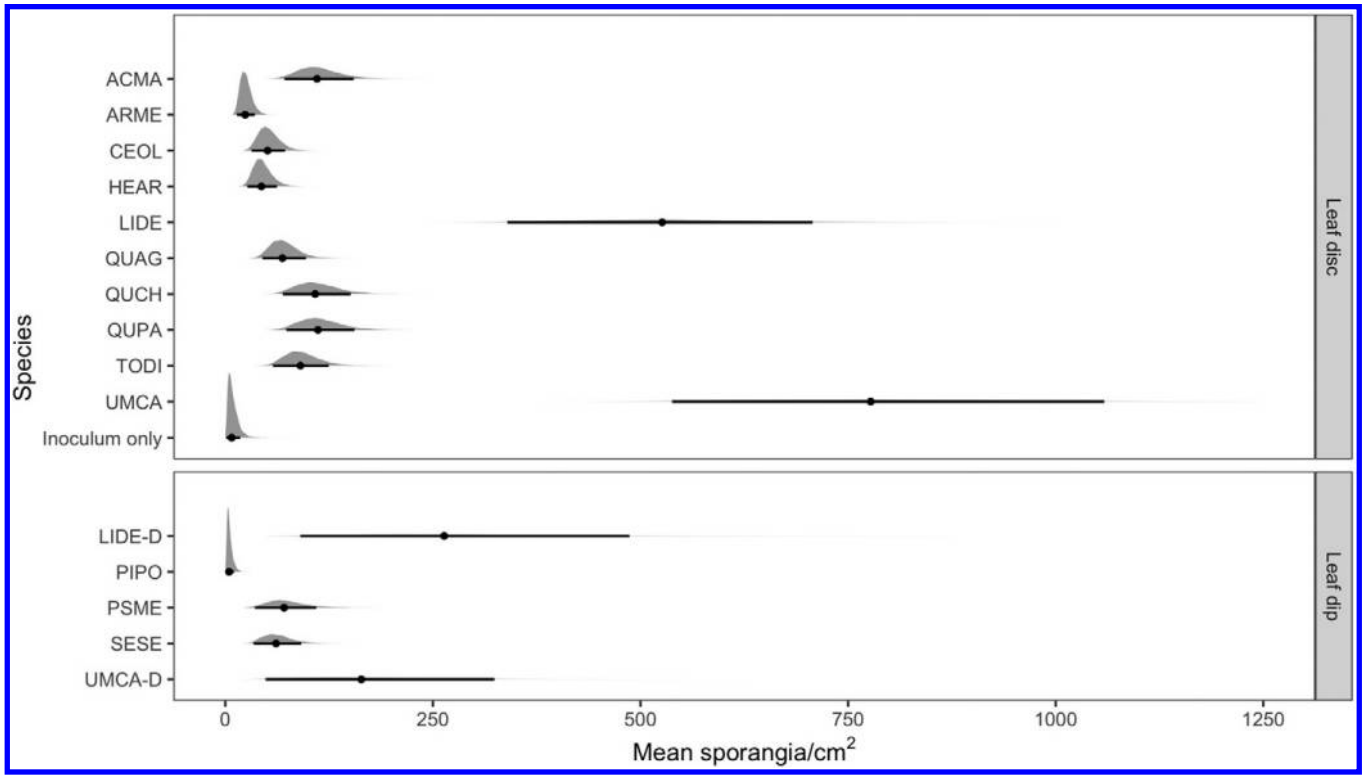

Fig. 1. Distributions (gray) depict the predicted mean sporangia per square centimeter produced on different plant species. Lines (black) represent the 90% highest posterior density interval and point estimate marks the median. The two panels are separated by assays. *Arbutus menziesii* (ARME) and the inoculum-only control were not significantly different, and neither were *Notholithocarpus densiflorus* (LIDE) and *Umbellularia californica* (UMCA) in either assay. Additionally, *Pinus ponderosa* (PIPO) was not significantly different from zero. ACMA = *Acer macrophyllum*, CEOL = *Ceanothus oliganthus*, HEAR = *Heteromeles arbutifolia*, QUAG = *Quercus agrifolia*, QUCH = *Q. chrysolepis*, QUPA = *Q. parvula*, TODI = *Toxicodendron diversilobum*, LIDE-D = *N. densiflorus* from the detached leaf assay, PSME = *Pseudotsuga menziesii*, SESE = *Sequoia sempervirens*, and UMCA-D = *U. californica* from the detached leaf assay.

centimeter after accounting for the zero inflation were as follows: bigleaf maple (*Acer macrophyllum*, 1,510 ± 330), ceanothus (*Ceanothus oliganthus*, 1,290 ± 287), madrone (51.4 ± 12.5), poison oak (*Toxicodendron diversilobum*, 98.7 ± 40.4), tanoak (4.20 ± 3.00), and toyon with 0 (not included in the model). Pairwise differences between these six species were all significant except for between bigleaf maple and ceanothus. In the leaf dip assay, Douglas fir produced 31.0 ± 13.1, tanoak 1.07 ± 1.49, and ponderosa pine 0 (not included in the model).

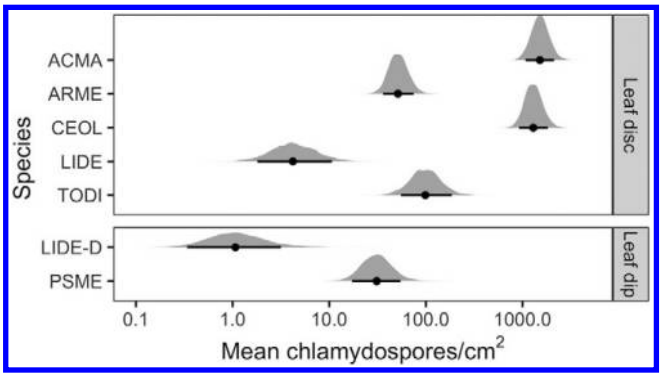

Fig. 2. Distributions (gray) depict the predicted mean chlamydospores per square centimeter produced on different plant species. Lines (black) represent the 90% highest posterior density interval and point estimate marks the median. The two different panels are separated by assays. Pairwise differences between all species were significant except for between *Acer macrophyllum* (ACMA) and *Ceanothus oliganthus* (CEOL). ARME = *Arbutus menziesii*, LIDE = *Notholithocarpus densiflorus*, TODI = *Toxicodendron diversilobum*, LIDE-D = *N. densiflorus* from the detached leaf assay, and PSME = *Pseudotsuga menziesii*.

Spores and lesion size. Except for ceanothus, madrone, and bigleaf maple, mean necrosis encompassed <50% of the exposed leaf tissue. The oaks in particular produced the lowest lesion coverages, with a mean ± 1 SD of 7.42 ± 4.48, 18.5 ± 14.9, 21.8 ± 20.0, and 6.82 ± 9.20 for tanoak, coast live oak, Shreve oak (*Q. parvula*), and interior live oak (*Q. chrysolepis*), respectively. Grouped across all species we detected a significantly positive relationship between sporangia production and lesion area (Supplementary Table S3). However, when we examined species-specific relationships, slopes were significantly positive only for bigleaf maple, interior live oak, poison oak, tanoak (from leaf dip assay), and bay laurel (from leaf dip assay) (Fig. 3). These findings indicate that for most species, lesion size does not predict sporangia quantity. Likewise, there was a positive relationship between chlamydospores and lesion area across all species (Supplementary Table S3), but the species-specific slopes were only significantly positive for bigleaf maple, ceanothus, and poison oak.

Discussion

We have shown that all host species included in this study support *P. ramorum* infections when challenged, but sporulation potential varies significantly across species. Bay laurel and tanoak were prolific sporangia producers, whereas the majority of species produced relatively lower amounts of sporangia. Large quantities of chlamydospores were also found in some species. Since our collections were exclusively from the Big Sur region, and natural variability in susceptibility to *P. ramorum* has been documented for bay laurel, tanoak, and coast live oak (Dodd et al. 2005; Hayden et al. 2011; Hüberli et al. 2012), it is possible that sporulation values and

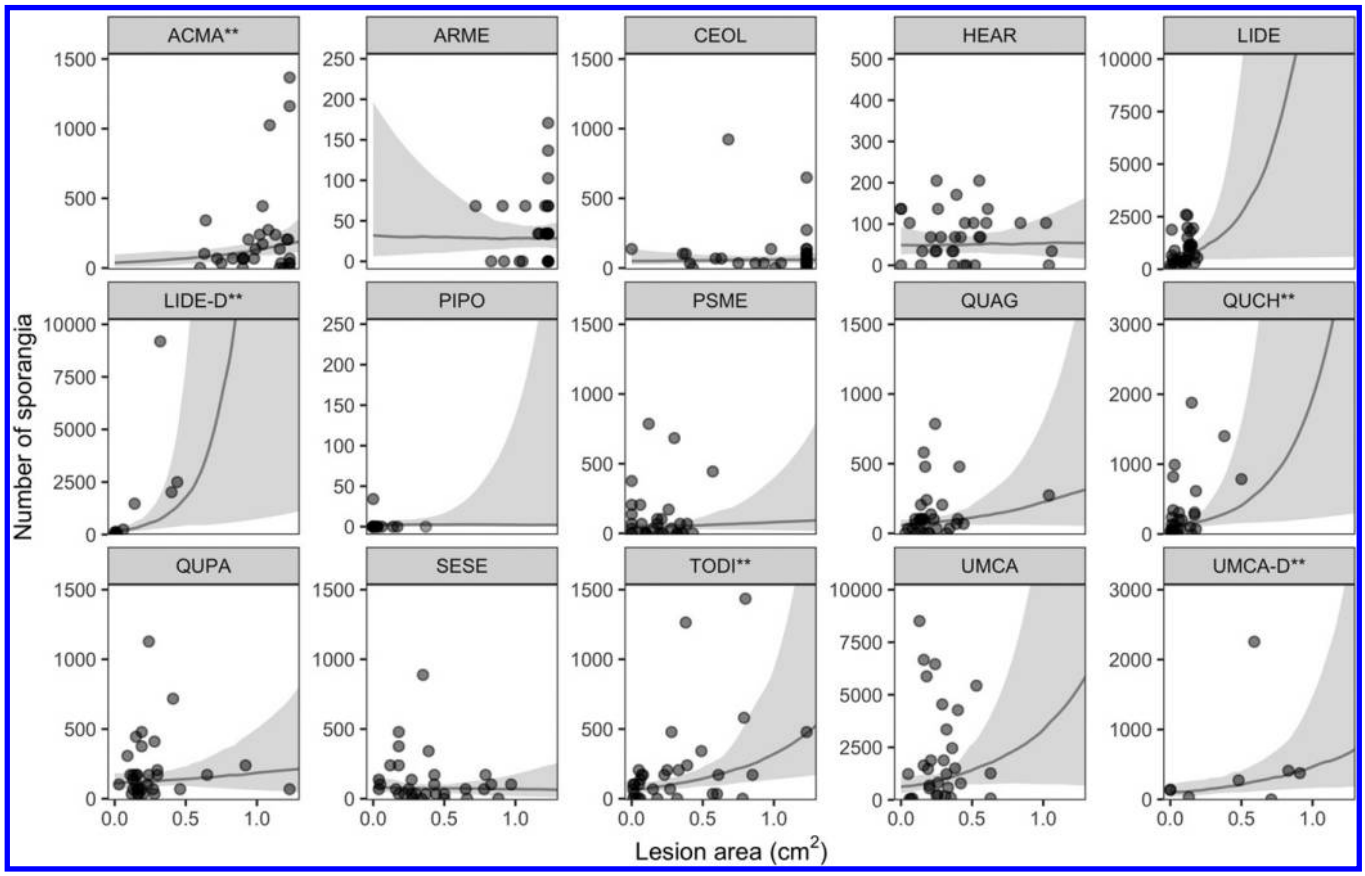

Fig. 3. The relationship between lesion area (in square centimeters) and number of sporangia produced across species. Points are mean values grouped by individual and the gray shaded region represents the posterior predictions of the model. Double asterisks denote a significant relationship between lesion area and sporangia for that species (the 90% highest posterior density interval of slope coefficient does not cross zero). ACMA = *Acer macrophyllum*, ARME = *Arbutus menziesii*, CEOL = *Ceanothus oliganthus*, HEAR = *Heteromeles arbutifolia*, LIDE = *Notholithocarpus densiflorus*, LIDE-D = *N. densiflorus* from the detached leaf assay, PIPO = *Pinus ponderosa*, PSME = *Pseudotsuga menziesii*, QUAG = *Quercus agrifolia*, QUCH = *Q. chrysolepis*, QUPA = *Q. parvula*, SESE = *Sequoia sempervirens*, TODI = *Toxicodendron diversilobum*, UMCA = *Umbellularia californica*, and UMCA-D = *U. californica* from the detached leaf assay.

relative ranks among species may differ should the study be expanded to more populations. In general, our results align closely, but not perfectly, with previous understanding of SOD-infested California coastal ecosystems.

Results from our study suggest that the sporulation potentials of tanoak and bay laurel are equivalent, but most studies across California regard bay laurel as the primary driver of SOD epidemics (e.g., Cobb et al. 2010; Dillon and Meentemeyer 2019; Garbelotto et al. 2017). Field measurements indicated that bay laurel was capable of producing orders of magnitude more sporangia than tanoak (Davidson et al. 2008), and early observational studies also found that the presence of bay laurel, but not tanoak, was the only significant host predictor in SOD infestations (Meentemeyer et al. 2008; Swiecki and Bernhardt 2002). However, the disease had not yet progressed to the northern parts of California, where tanoak is most abundant. The negative correlation between disease and tanoak distribution may have led to the insignificance of tanoak (Meentemeyer et al. 2008). Meanwhile, subsequent models from field-collected data in the Big Sur area and more northern coastal regions suggested that the two species both contribute significantly to the likelihood of SOD infections (Cobb et al. 2012; Haas et al. 2011).

We also detected sporangia production in species implicated as epidemiologically unimportant. Generally, true oak species are considered dead-end hosts that are incapable of producing sporangia (Davidson et al. 2005), whereas all other species are considered low-competency hosts at most (Meentemeyer et al. 2004). Aside from ponderosa pine and madrone, our study showed that all of the other tested species, including true oak species, were able to produce nonzero quantities of sporangia. Moreover, several hosts abundantly produced chlamydospores, which may or may not be important for pathogen survival. These spores are known to survive at high rates in the soil for the duration of California's dry summer months (Fichtner et al. 2007) and although germination rates are low (Tooley et al. 2008), large numbers ensure the presence of viable inoculum in the soil and litter. Specifically, bigleaf maple and ceanothus produced quantities of chlamydospores that rival the maximum known concentrations on highly susceptible species, rhododendron and bay laurel (Fichtner et al. 2007, 2009).

While we believe species differences within the leaf disc and leaf dip assays may be compared, we caution against direct contrasts between assays. Cutting leaf discs involves additional wounding around the leaf edges, which might contribute to differences in sporulation potential by inducing or suppressing defenses against pathogens. However, we are unaware of studies that have examined how wounding affects local defenses on sporulation; it remains an open and worthy area of study. We estimate that species in the leaf dip assay sporulated two to four times less than species in the leaf disc assay. As a result, the sporulation potentials and lesion areas of Douglas fir and redwood may have been underestimated relative to the broadleaf species and direct comparisons with broadleaved species besides tanoak and bay laurel remain imprecise.

Given that laboratory inoculations are not perfect representations of in situ infections, we offer explanations to account for discrepancies between our findings and field-based studies. First, our inoculum concentration (4,000 sporangia/ml) corresponds to the seasonal peak of sporangia production from bay laurel leaves during a wet year (Davidson et al. 2008), conditions that exceed average. Increased inoculum loads could lead to artificially higher rates of necrosis, infection, and sporulation (Hansen et al. 2005; Tooley et al. 2004, 2013), including for species that typically do not show foliar symptoms in nature (Vettraino et al. 2008). Furthermore, the inoculated detached leaves and cut leaf discs were without whole plant systemic defenses. While lowered defenses could elevate susceptibility (Orłowska et al. 2012) and potentially sporulation, direct comparisons from both leaf discs (Brown et al. 1999; Cohen 1993; Kortekamp 2006), and detached leaves (Brooks 2008; Harrison and Lowe 1989) have shown sporulation and susceptibility rates to strongly correlate with those from intact plants.

It is also conceivable that our sporangia production estimates are not reflective of realized plant-to-plant transmission owing components of host physiology and phenology. For example, since overstory trees are suspected to have a greater influence on *P. ramorum* establishment and transmission (Metz et al. 2012; Peterson et al. 2014; Simler-Williamson et al. 2020), species in the understory may comparatively transmit the pathogen much less regardless of their sporulation potential. Additionally, deciduous species have a narrower transmission window because their leaves flush out nearly 4 to 5 months after evergreen species begin producing spores (Davidson et al. 2008). Finally, biases inherent in observational plot studies should not be overlooked. Symptoms are most obvious on common, susceptible, large trees; for this reason, field protocols often rely on bay laurel and tanoak as indicator species to represent the community's infection and mortality rates. Even if all hosts are examined for symptoms, our results and others' indicate that detection can be low since *P. ramorum* can cause latent or small lesions (Denman et al. 2009; Harris and Webber 2016).

These caveats evoke the need to assess the ecological relevance of our sporulation results by corroborating them with field-collected data. One simple first step would be to update risk maps that rely on sporulation potentials primarily grounded in expert opinion (Cunniffe et al. 2016; Meentemeyer et al. 2004, 2011) with our empirically derived sporangia values and assess whether model performance improves. Another approach is to explore the explanatory power of community competency (Johnson et al. 2013, 2015a), the cumulative product of each tree species' density and measured sporulation potential, for predicting infection risk in a stand. Johnson et al. (2013) found that community competency and its associated interactions among amphibian hosts explained 89% of the variance in total load of a trematode parasite. Through many years of research focus, researchers have amassed several rich SOD datasets complete with community and disease data, allowing a similar analysis to be completed in the near future.

Until our results are validated and calibrated with field data, we cautiously speculate on the consequences they might have on our understanding and management of SOD. We reiterate existing calls to thin both bay laurels and tanoaks in stands that are at risk or recently infested (Cobb et al. 2013; Valachovic et al. 2008), since they both appear to be significant drivers of pathogen spread. Regarding the implications of low-competency hosts, we hypothesize that they play a role in transmission at the local but not landscape scale. Perhaps the strong performance of the California statewide risk model from Meentemeyer et al. (2008), which incorporates the presence of bay laurel as the only host predictor, speaks to the notion that limited knowledge of one of the key hosts is sufficient to garner a coarse outlook on disease risk. We still suspect that pathogen spread involving more minor hosts is occurring, albeit sometimes at low intensities, given that most species produce inoculum in the form of either sporangia or chlamydospores. They may act as reservoirs, facilitating transmission to nearby high-competency hosts, which can lead to more secondary transmission and mortality at the broader scale.

Overall, this study is largely confirmatory, echoing much of what has been suspected regarding host competency but never formally and exhaustively tested. The greatest value of this study lies in what is now possible. By generating an empirical foundation for host competency, we can better explore how the composition of newly invaded or disturbance-altered communities may interact with the epidemiology of *P. ramorum*. Additionally, we now have an exciting opportunity to test the importance of host community composition on local transmission in an invasive forest pathogen system. This study augments decades of ecological monitoring, positioning SOD as an even more valuable system to explore the community's role in disease dynamics.

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