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Perspectives of spatial scale in a wildland forest epidemic

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Abstract The challenge of observing interactions between plant pathogens, their hosts, and environmental heterogeneity across multiple spatial scales commonly limits our ability to understand and manage wildland forest epidemics. Using the forest pathogen *Phytopthora ramorum* as a case study, we established 20 multiscale field sites to analyze how host-pathogen-environment relationships vary across spatial scales of observation in a wildland pathosystem. We developed statistical models of disease intensity across five nested levels of spatial aggregation, from an individual host through four broader spatial extents of observation. Analyses were conducted from two spatial perspectives: a *focal view*, where disease intensity at one scale was examined as a function of broader-scale landscape conditions, and an *aggregate view*, where disease intensity and landscape conditions was observed at the same scale of spatial aggregation. For each perspective, separate models were developed to compare direct field measurements of host density versus less expensive remotely sensed estimates of host habitat as predictors of disease in landscape-scale studies. From both perspectives, models using direct measurements of host density performed better than models using remotely sensed estimates of host habitat across all four spatial extents. We found no significant difference in model performance at the individual level. From the focal view, the performance of host density models declined with increasing spatial extent, whereas the performance of host habitat models improved with spatial extent. These results illustrate how the scale of observation – both spatial extent and measurement detail – can influence conclusions drawn from epidemiological models of wildland pathosystems.

Keywords Host density · Landscape epidemiology · Multilevel · Multiscale · *Phytophthora ramorum* · Sudden oak death

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Introduction

The landscapes of wildland plant pathosystems exhibit substantial environmental heterogeneity compared to agricultural croplands or intensively managed ecosystems, such as tree plantations. The increased complexity and cross-scale interactions in these landscapes make it challenging to measure and analyze the drivers of disease dynamics. Even when measurements of environmental heterogeneity are undertaken there is rarely a single correct scale of observation that is known a priori for a given pathosystem (Meentemeyer et al. 2012; Fig. 1). Meanwhile, host-pathogen interactions are intricately embedded within communities, ecosystems, and entire landscapes, so it is essential that we understand the role that environmental heterogeneity across multiple scales plays in the spread and persistence of wildland diseases (Ostfeld et al. 2005; Burdon et al. 2006). In addition, the scale of observation and analysis has been recognized as influential to results and conclusions drawn from models (Wiens 1989; Plantegenest et al. 2007; Chave 2013). Still, our best empirical understanding of

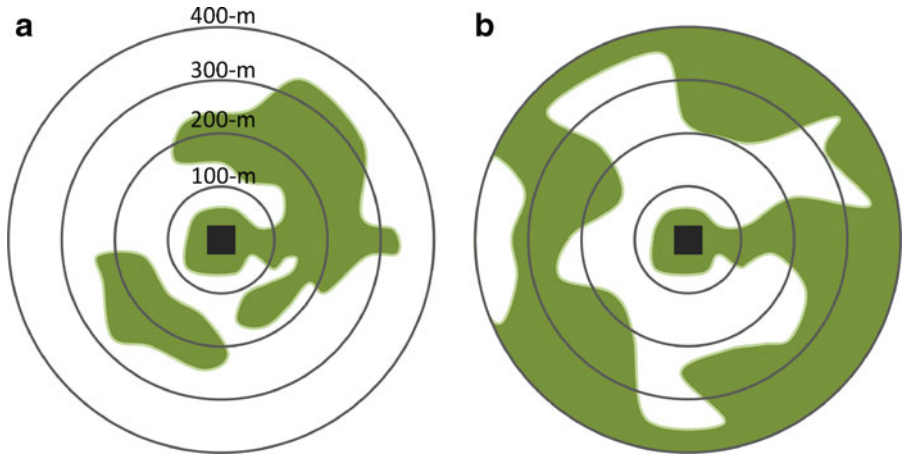

Fig. 1 The selection of spatial extent for a given study may influence the model results and conclusions. In this hypothetical example, the choice of a single spatial extent at 100-m would suggest that both plots (represented as *black squares*) are surrounded by identical amounts of contiguous host habitat (show in *green*). In contrast, a slightly larger 200-m extent would suggest that plot '**a**' was surrounded by more host habitat, whereas even larger spatial extents (i.e., 300–400-m) would reveal that plot '**b**' is actually surrounded by a substantially larger area of contiguous host habitat. Reprinted from Meentemeyer et al. (2012) with permission from Annual Reviews via Copyright Clearance Center

host-pathogen-environment interactions is predominantly derived from analyses conducted at single (and small) spatial scales.

The emerging discipline of landscape epidemiology provides concepts and tools for analyzing the role that environmental heterogeneity plays in the development of epidemic trajectories across space and time. Yet, in a recent review of 143 landscape epidemiological studies, Meentemeyer et al. (2012) found that only 13 % utilized a multiscale approach. Perhaps the slow progress toward understanding scale-dependent processes in wildland pathosystems stems from the more complex spatial heterogeneity of biotic and abiotic conditions compared to experimental and intensely managed settings. These circumstances present a special challenge for studying wildland pathogens across large geographic areas, because our ability to collect data of direct epidemiological relevance decreases as study extents increase. For example, our ability to directly measure environmental heterogeneity (e.g., solar radiation and host density) in the field across multiple scales becomes progressively less feasible as study extents expand in scope and duration. As a compromise, multiscale disease studies in natural ecosystems often resort to using indirect measures of environmental heterogeneity (e.g., elevation and land cover), which serve as surrogates for the underlying epidemiological drivers. Thus, the scale of observation may especially affect our understanding of host-pathogen-environment interactions in wildland settings.

In this study, we compare the performance of landscape epidemiological models parameterized with direct field measurements to those using indirect measurements of biotic environmental heterogeneity from two spatial perspectives across multiple extents. We focus on a heterogeneous forested landscape infested by the emerging wildland plant pathogen *Phytophthora ramorum* (Phylum *Oomycota*), the causal agent of sudden oak death in North America (Rizzo and Garbelotto 2003). *P. ramorum* was discovered in the mid-1990s in the greater San Francisco Bay Area of California (Rizzo and Garbelotto 2003; Rizzo et al. 2005), and spread rapidly via local and long-distance dispersal events, resulting in current infestations from southwestern Oregon to the Big Sur region of California (Václavík et al. 2012). This pathogen causes diseases expressed in two ways: stem canker infections, and foliar and twig infections (Rizzo et al. 2005). Canker infections have led to extensive mortality of trees throughout coastal forests of California and Oregon, particularly coast live oak (*Quercus agrifolia*), California black oak (*Quercus kellogii*), as well as tanoak (*Notholithocarpus densiflorus*) (Rizzo et al. 2005; Meentemeyer et al. 2008a). The infectious inoculum from this pathogen is produced through sporulation from the nonlethal foliar infections, predominantly on non-canker host species

(Rizzo et al. 2005). Although recognized as a generalist pathogen with an incredibly broad range of hosts (>130 confirmed host species; APHIS 2013), the abundant inoculum production responsible for driving epidemic spread across the landscape arises predominantly from two highly-competent foliar host species: California bay laurel (*Umbellaria californica*) and tanoak. Tanoak is unique in this pathosystem as the only species known to be susceptible to lethal canker infections as well as function as a competent foliar host for inoculum production.

Previous studies on *P. ramorum* have reported that the majority of pathogen transmission occurs locally (<10-m), though dispersal may occur regularly within a 200-m radius of infected sites (Davidson et al. 2005; Swiecki and Bernhardt 2007; Mascheretti et al. 2008). Rare long-distance dispersal events (> 1-km) are the most likely cause of the patchy distribution of this pathogen across northern California and into southern Oregon (Meentemeyer et al. 2004, 2008a; Ellis et al. 2010; Václavík et al. 2012). Although there have been a number of studies focusing on the epidemiology of the *P. ramorum* pathosystem – elucidating some general disease-environment relationships – most of these have been spatially implicit and/or used a single resolution and extent (e.g. Hansen et al. 2008; Cobb et al. 2010; Haas et al. 2011; Hüberli et al. 2011; Metz et al. 2012). We recognized two studies of this system that applied multiscale approaches by examining relationships across either local to landscape (Condeso and Meentemeyer 2007) or local to regional scales (Cushman and Meentemeyer 2008). Of these, Condeso and Meentemeyer (2007) provided the most compelling study on disease-environment relationships of *P. ramorum* across multiple scales. The authors used high-resolution forest cover data to examine the relationship between the proportion of forested area (host habitat) and disease intensity in focal plots across nested spatial scales of increasing extent. They discovered that the explanatory power of their models improved when host habitat at broader scales was included as an explanatory variable up to a 200-m radius around focal plots.

Although forest cover proved to be useful for explaining disease severity in the study by Condeso and Meentemeyer (2007), it is an indirect measure of the mechanism(s) involved in the underlying process of disease transmission. Specifically, host habitat cover may be considered a surrogate for host density, however, high amounts of host habitat cover do not necessarily confer high host density. As such, using host habitat in place of for host density may limit our understanding of disease dynamics in pathosystems regulated by density-dependence mechanisms. Furthermore, the spatial variability in disease intensity of pathosystems that are influenced by diversity-disease risk processes, such as sudden oak death (Haas et al. 2011), may also be better understood by examining the relationship between disease and host density across multiple scales.

Here, we examine host-pathogen-environment relationships across multiple spatial scales in a wildland *P. ramorum* pathosystem. Our goal is to understand how the perspective of scale used for observation and analysis, as well as choice of variable, influences the inference and conclusions drawn from landscape epidemiological models. Using a field-based sampling strategy, we collected data on the pathogen, hosts, and environmental heterogeneity across five levels of spatial aggregation: individual host stems and four broader (nested) spatial extents. We analyze these data from two spatial perspectives: a *focal view*, where disease intensity at one scale is examined as a function of broader-scale landscape conditions, and an *aggregate view*, where disease intensity and landscape conditions are observed at the same scale of spatial aggregation. For each perspective, we develop multiple models in order to compare direct field measurements of host density versus less expensive remotely sensed estimates of host habitat as predictors of disease in landscape-scale studies.

Methods

Data collection

We designed and implemented a field-based sampling strategy to capture the spatial heterogeneity of host-pathogen interactions across multiple spatial scales. During the spring of 2010, we established 20 multiscale sites on the western side of Sonoma Mountain in southeastern Sonoma County, California (Fig. 2). At each site we collected data on disease intensity and host density at nested extents of 15-m×15-m, 60-m×60-m, 140-m×140-m, and 220-m×220-m. We performed a census of all stems within the 15-m extent, recording the diameter at breast height (DBH; breast height=1.4-m) on stems of the three main *P. ramorum* host species (coast live oak, black oak, and bay laurel) occurring in the study area that measured ≥2-cm DBH. We also recorded the presence of stems of other species that measured≥5-cm DBH.

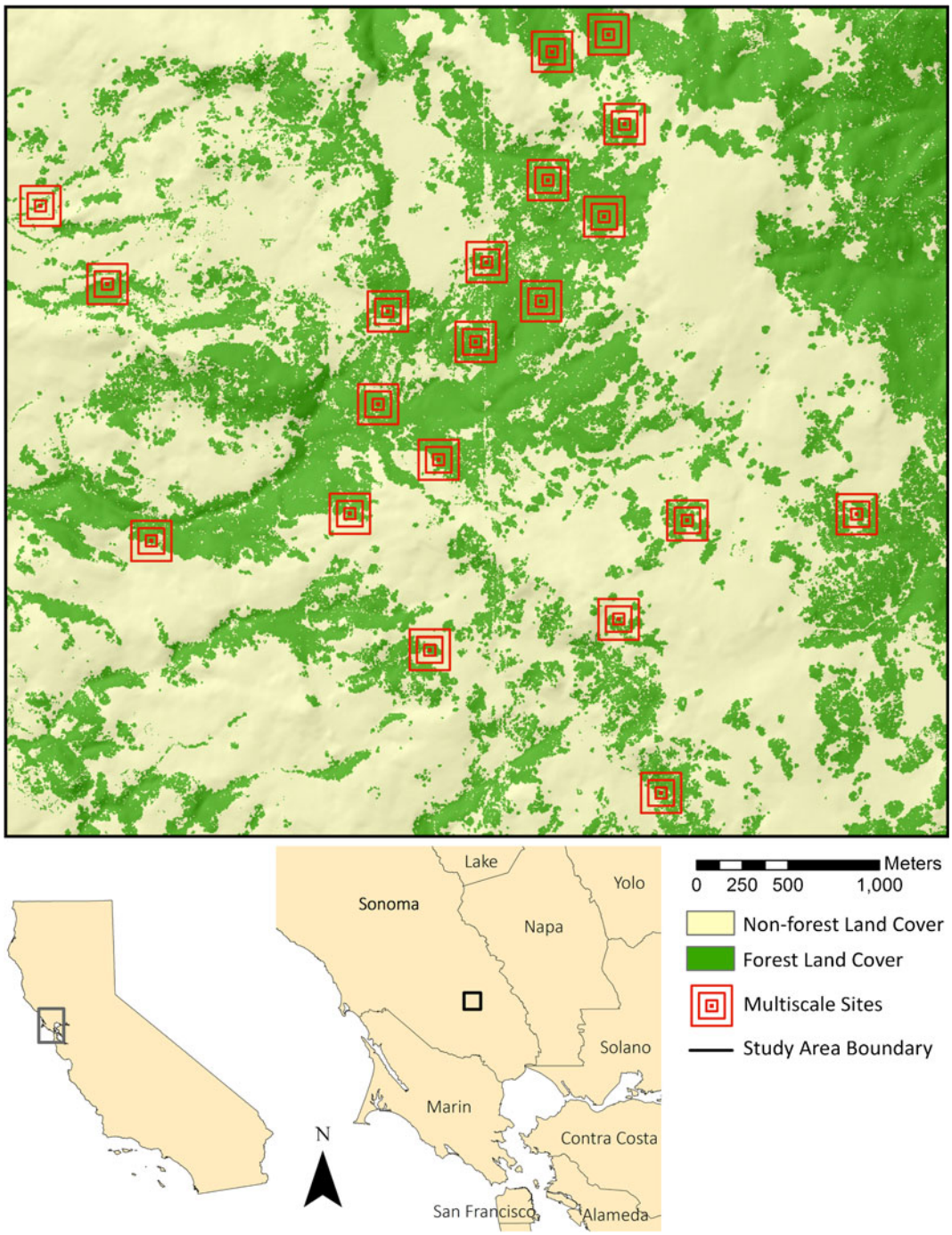

Fig. 2 Study area and multiscale study site locations in Sonoma County, California. The nested spatial extents of each site correspond to 15-m×15-m, 60-m×60-m, 140-m×140-m, and 220-m×220-m

A complete census across all extents at each site was considered cost-prohibitive, so we quantified host density and disease intensity at the 60-m, 140-m, and 220-m extents using the point-quarter transect sampling method (Cottam and Curtis 1956). We established 36 evenly spaced satellite plots around the 15-m extent at each site using line transects (Morrison 1996) (Fig. 3a). Each satellite plot was divided into quadrants, the

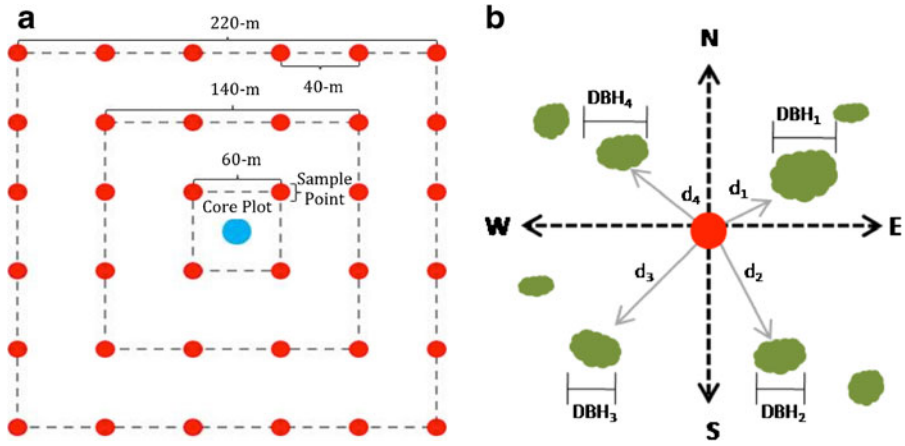

Fig. 3 The point-quarter sampling strategy employed at each of the 20 multiscale survey sites. **a** The configuration of evenly-spaced satellite plots (*red dots*) sampled around the 15-m extent (*blue dot*) to create the four nested scales; **b** illustration of the point quarter sampling method used at each of the satellite plots (720 total), where 'd_x' is the distance from the centre of each satellite plot to the nearest stem in each quadrant, and 'DBH_x' is the diameter at breast height of that stem

distance from the centre of each satellite plot to the nearest stem≥5-cm DBH in each quadrant was measured, and species of that stem was recorded (Fig. 3b). To calculate bay laurel density across the three broader extents, we first calculated an estimate of the total stem density at each extent using the point to plant distances measured at the satellite plots within that extent in the following equation (Cottam and Curtis 1956),

$$Total\,stem\,density = 1/(mean\,point\text{–}to\text{–}stem\,distance)^2 \quad (1)$$

We then calculated the relative density of bay laurel stems at each extent as the number of bay laurel stems divided by the total number of stems of all species. In order to calculate an estimate of bay laurel stem density at each extent, we applied the relative density of bay laurel stems and total stem density in the following equation,

$$Bay\,laurel\,stem\,density = relative\,density\,of\,bay\,laurel\,stems * total\,stem\,density \quad (2)$$

resulting in the number of stems per square metre at each extent. At the 15-m extent we only applied Eq. 2 because the census we conducted at that extent meant that the total stem density was empirically derived and so Eq. 1 was unnecessary. When we calculated the estimates of bay laurel density at the larger spatial scales, we included the stems recorded at the 15-m extent in the calculation of relative density. Thus, the host density estimate calculated for each extent was composed of data aggregated from smaller nested extent.

To assess disease intensity we conducted 60-s counts of leaves exhibiting *P. ramorum* symptoms on all bay laurel stems (cf. Condeso and Meentemeyer 2007) encountered at each site. The disease intensity at each extent was calculated as the total number of symptomatic leaves by summing the number of symptomatic leaves across the nested scales. For example, the disease intensity at the 15-m extent was the sum total of the leaf counts within that extent, while the disease intensity at the 60-m extent was the sum of the leaf counts at 15-m plus the sum of the leaf counts from the satellite plots at the 60-m extent.

We used a geographic information system to calculate the proportion of host habitat and the average elevation at each extent across the 20 multiscale sites. The proportion of host habitat was calculated by extracting the number of cells within each extent that were classified as host habitat from a 5-m resolution grid and dividing this value by the total number of cells in that area. This host habitat surface was originally developed from 1-m resolution ADAR imagery (see Condeso and Meentemeyer 2007). Average elevation in metres was calculated from a 10-m resolution United States Geological Survey digital elevation model by averaging the values of the cells within each extent.

Analysis

We modeled disease intensity as a function of elevation, and either bay laurel density or the proportion of host habitat, across five levels of spatial aggregation:

individual bay laurel stems, and the four spatial extents (15-m, 60-m, 140-m, and 220-m). At each of the four spatial extents, we examined disease intensity as a function of the predictor variables observed at the same extent (*aggregate view*), as well as at each larger extent (*focal* view), using separate ordinary least squares regression models (OLS). For example, disease intensity at the 60-m extent was modeled as a function of the predictors at that same extent (aggregate view models), and then in two other models using observations of the predictor variables at each of the larger extents (focal view models). We ran paired models at each extent (one using host density and the other using host habitat) in order to compare the effect on model performance of directly measured host density versus the host habitat surrogate. Elevation was included as a predictor variable in each model in order to account for some of the variation in the physical environment across this landscape. To satisfy assumptions of normality for OLS, we applied transformations to the disease intensity, host density, and host habitat variables (i.e. symptomatic leaf count, bay laurel density, and the proportion of host habitat). Disease intensity and host density were transformed using the natural logarithm, and host habitat was arcsine transformed.

Disease intensity at the individual level was measured as the symptomatic leaf count of an *individual* bay laurel stem from the census at the 15-m extent across the 20 multiscale sites. We modelled the individual's disease intensity from the focal view as a function of stem DBH, elevation, and either host density or host habitat in separate models using predictor variables observed at each of the four spatial extents. In order to model disease intensity at the level of individual stems we needed to account for the grouping of the individual stems within the same extent at each site, so we used a generalized linear mixed effects modelling approach (Gelman and Hill 2007). This is similar to generalized linear models where the family of distributions (e.g. binomial or Poisson) of the response variable is designated, but also provides the additional benefit of including random effects that account for unobserved variance resulting from hierarchical structuring of data, e.g. clustering of individuals within sites (Gelman and Hill 2007; Bolker et al. 2009). In our case, we needed to account for the unobserved variance affecting stems grouped within the 15-m extent of each site, so we identified the 'site' as a random effect. Since we were modelling count data, we specified the Poisson family of distributions (Bolker et al. 2009; O'Hara and Kotze 2010). Preliminary analysis revealed that our count data was overdispersed, so we included a data-level variance component, 'stem', as an additional random effect (Gelman and Hill 2007, pp. 325–331).

All variables were standardized prior to running models, because our goal was to compare the inference, statistical significance, and relative influence of the covariates, as well as the goodness-of-fit and/or explanatory power of the models. We assessed and compared the goodness-of-fit of the mixed effects regression models (individual-level, focal view) using the Akaike information criterion (AIC; Akaike 1974). This information theoretic approach offsets the log-likelihood of a model with a penalty for each predictor variable, enabling the identification of which model of a set is most likely capable of reproducing the observed data (Burnham and Anderson 2002). Lower AIC values indicate better model fit, and a change in AIC of four may indicate a significantly better model (Burnham and Anderson 2002, p. 446). The performance of ordinary least squares regression models were assessed based on explanatory power (the adjusted R^2), as well as their goodness-of-fit, measured as AICc. The AICc metric is an extension of AIC for small sample sizes, and as sample size increases AICc converges to AIC (Burnham et al. 2010). We used AICc to assess the models of disease intensity at the four spatial extents because there were only 20 replicates at these levels (i.e. one for each site), compared to 331 stems across all the sites for the individual level models.

Prior to modelling, we examined the collinearity among predictor variables, selecting a threshold of 0.50 for variable inclusion in the same model. All statistical analyses were conducted using the R Statistical Software, version 3.0.2 (R Core Team 2013). Generalized linear mixed effects models were implemented using the 'lmer' function in the 'lme4' package (Bates et al. 2013) and ordinary least squares regression models were implemented using the 'lm' function in the base 'stats' package. The 'AICc' function from the 'MuMIn' package (Barton 2013) was used for calculating AICc of each OLS regressions.

Results

Across the four spatial extents (15-m, 60-m, 140-m, 220-m) at all 20 multiscale sites, bay laurel density

ranged from 0.0014 to 0.1867 stems/m^2; the proportion of host habitat ranged from 0.11 to 1; elevation ranged from 273 to 691-m; and disease intensity ranged from 64 to 8936 symptomatic leaves (Appendix Fig. 7). Disease intensity on the individual bay laurel stems from the censuses at the 15-m extents ranged from 0 to 257 symptomatic leaves (mean=58) and the DBH of these stems ranged from 2 to 105.3-cm (mean=21.9). Correlation tests between predictor variables in the same models resulted in a maximum Pearson's coefficient of less than 0.40 During this analysis we found that bay laurel density had a strong positive relationship with the proportion of host habitat, especially at the broader 140-m and 220-m extents where Pearson's correlation coefficients were>0.75 (Appendix Fig. 8).

Our analyses showed that the effectiveness of host density or host habitat for predicting disease intensity varied with the model perspective and use of either a direct or indirect predictor variable. In the next two sections, we describe the results from the models of disease intensity at the individual level ("Individual-level Models") and for the suite of models at each of the four spatial extents examined from the focal and aggregate views ("Extent-level Models").

Individual-level models: focal view

After accounting for elevation, stem DBH, and the variability captured in the random effects of 'site' and 'stem', neither host density nor host habitat measured at any extent were statistically significant ($p<0.1$) in their respective models (Table 1). Based on AIC scores, the best model was with host habitat at the 15-m extent, however, the change in AIC across models was≤2 (Table 1; Fig. 4). Although the differences were small, elevation was relatively more influential at smaller extents in the host density models, whereas it was relatively more influential at broader extents in the host habitat models (Fig. 4).

Extent-level models: focal and aggregate views

From both perspectives, we found that using host density in models of disease intensity provided more explanatory power (higher adjusted R^2) and better goodness-of-fit (lower AICc) than models using host habitat at all spatial scales (Table 2; Figs. 5a-c and 6a-b). In comparing the models from the focal perspective, we found that host density models always performed better than host habitat models at each scale, while the performance of host density models at a focal extent declined as the scale at which the predictor variables were observed increased. Conversely, the performance of host habitat models at each scale tended to improve as the extent at which the predictor variables were observed increased (Table 2, Figs. 5a-c). From this perspective, the best performing host density and host habitat model (lowest AICc and highest adjusted R^2) was when disease intensity was modelled at the 140-m extent as a function of predictors observed at the 220-m extent (Table 2, Fig. 5c). Host density was statistically significant ($p<0.05$) in at least one model at each extent of disease intensity, whereas host habitat was significant only when observed at 220-m for models of disease intensity at 60-m and 140-m.

To compare the host density to host habitat models from the aggregate perspective, we plotted the adjusted R^2 and AICc (Fig. 6a), and the standardized beta coefficients of the predictor variables (Fig. 6b) from the models at each extent. Similarly to the focal perspective, models parameterized with host density provided greater explanatory power and better goodness-of-fit than models parameterized with host habitat at each extent (Table 2, Fig. 6a). The model using host density at the 15-m extent produced the best model of disease intensity from this perspective, as well as best model overall ($R^2=0.74$, AICc=39.03; Fig. 6a, Table 2), with both predictor variables statistically significant (Fig. 6b, Table 2). This contrasts with the results from the focal perspective where the best models using either host density or host habitat were at the broadest spatial scale, although the best host habitat model was still at the broadest extent (220-m) from this perspective.

The R^2 as well as AICc of the host density models decreased at the 60-m extent, although explanatory power then increased marginally at the 140-m and 220-m extents. The AICc of the host density models showed no notable difference across the 60-m to 220-m extents. The host density predictor variable was statistically significant in models at each extent, while elevation was statistically significant only in models at the 15-m and 60-m extents. In comparison, the explanatory power of host habitat models consistently increased across the four spatial extents from an R^2 of 0.11 at 15-m to an R^2 of 0.49 at 220-m (Table 2; Fig. 6a). The goodness-of-fit of the host habitat models also improved

Table 1 Standardized coefficients and model fit (AIC) from generalized linear mixed effects regression models of disease intensity on individual bay laurel stems. The predictor variables for each model, elevation and either bay laurel density or the proportion of host habitat, were calculated for the corresponding extent of that model. Model fits may be compared across all models using AIC. The standardized coefficients show the relative importance of each variable for predicting stem-level infection intensity within each model

Model extent	Stem DBH	Elevation	Bay laurel density	% host habitat	AIC
15-m	-0.2287**	0.3089**	-0.1083	–	1545
	-0.2260**	0.2709**	–	-0.1605	1543
60-m	-0.2278**	0.2813**	-0.0175	–	1545
	-0.2304**	0.3223**	–	-0.1211	1544
140-m	-0.2286**	0.3041**	-0.1037	–	1545
	-0.2312**	0.3216**	–	-0.1181	1544
220-m	-0.2286**	0.3158**	-0.1347	–	1544
	-0.2301**	0.3163**	–	-0.1064	1544

Model extent is the extent at which the predictor variables were measured or calculated; *Stem DBH* an individual bay laurel stem's diameter at breast height; *Bay laurel density* is the effect of bay laurel density in the model at each extent; *Elevation* is the effect of elevation in the model at each extent; *% host habitat* is the effect of the proportion of host habitat in the model at each extent

Statistical significance: $^{**}p<0.05$

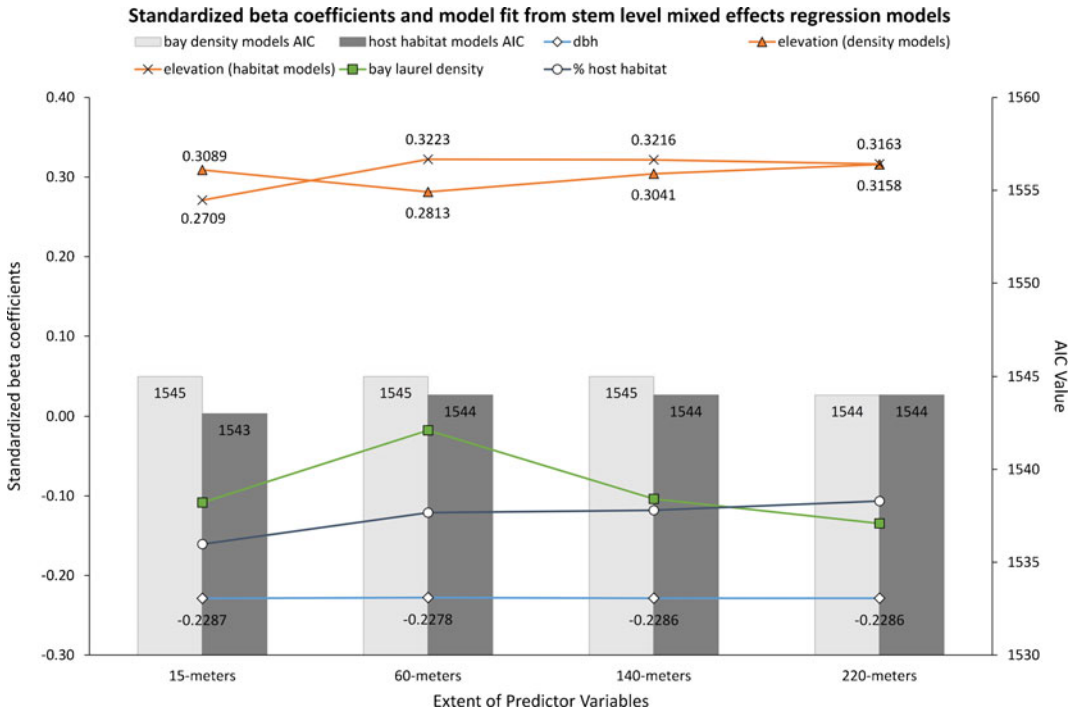

Fig. 4 Standardized beta coefficients and AIC scores from the generalized linear mixed effect models of symptomatic leaf count on individual bay laurel stems at the 15-m extent of each site. An individual's symptomatic leaf count was modelled as a function of stem DBH, elevation, and either bay laurel density or proportion of host habitat at each extent. The magnitude of the coefficients are plotted as lines using the primary y-axis on the left, with precise values reported when a variable was statistically significant at the $p<0.1$ level. Coefficient values for stem DBH varied by less than 0.01 at each scale between density and habitat models, so only the values from the density model were plotted. The AIC scores for models using either bay laurel density or proportion of host habitat are plotted as columns at each extent and use the secondary y-axis on the right. Model AIC values are reported inside the end of each column; AIC values within and across extents do not indicate any one model having notably better fit over any other model

Table 2 Results from OLS models of disease intensity at each extent from focal and aggregate views. The standardized coefficients of each predictor, AICc, and adjusted R^2 are reported for each model

Model extent	Elevation	Bay laurel density	% host habitat	AICc	Adj. R^2
Focal view					
Disease intensity at 15-m					
60-m	0.2136	0.4764*	–	59.58	0.26
	0.3936	–	0.1218	63.70	0.10
140-m	0.2555	0.3815	–	61.34	0.20
	0.3936	–	0.1218	63.70	0.10
220-m	0.2926	0.2897	–	62.56	0.15
	0.3413	–	0.2556	62.72	0.14
Disease intensity at 60-m					
140-m	0.2202	0.5005**	–	51.66	0.40
	0.3524*	–	0.2754	56.31	0.24
220-m	0.2412	0.4322*	–	53.79	0.33
	0.3262	–	0.3492*	54.94	0.29
Disease intensity at 140-m					
220-m	0.0615	0.7170**	–	47.83	0.54
	0.2103	–	0.5589**	52.97	0.40
Aggregate view					
15-m	0.2556*	0.8162**	–	39.03	0.74
	0.4358*	–	0.1550	63.43	0.11
60-m	0.1662	0.6253**	–	46.39	0.54
	0.3601*	–	0.2687	56.40	0.23
140-m	0.0685	0.7462**	–	45.23	0.60
	0.2274	–	0.5075**	54.71	0.35
220-m	-0.0056	0.8572**	–	46.47	0.62
	0.1632	–	0.6920**	52.82	0.48

Model extent refers to the extent of observation of the predictor variables from the focal perspective, and the extent of observation of the predictor and the response variables from the aggregate perspective. *% host habitat* is the proportion of host habitat

Statistical significance: '*' $p<0.1$, '**' $p<0.05$

with increasing extent from an AICc of 63.43 at 15-m to an AICc of 52.55 at 220-m, a notably better fit compared to models at the 15-m and 60-m extents ($\Delta AIC>4$; Burnham and Anderson 2002, p. 446). The relative influence of the host habitat predictor variable increased with increasing extent, but was statistically significant only at the 140-m and 220-m extents (Fig. 6b). The elevation predictor variable was statistically significant only at the 15-m and 60-m extents in the host habitat models.

Discussion

Issues of spatial scale are a key consideration in studying disease epidemics (Real and McElhany 1996; Jeger et al. 2007), and may be especially important for improving our understanding of disease dynamics in wildland pathosystems (Holdenrieder et al. 2004; Kauffman and Jules 2006). Equally important is the selection of epidemiologically relevant variables that provide useful inference and interpretable results for the efficacious management of diseases in natural ecosystems (Meentemeyer et al. 2012). In this study, we found that the scale of observation – spatial extent and measurement detail – affected model outcomes.

Our results concur, as well as provide novel insight, to findings from the multiscale study on *P. ramorum* by Condeso and Meentemeyer (2007). Their study area occurred in the same geographic region as ours, so underlying differences in habitat type and invasion history do not confound comparisons of results. By including the proportion of host habitat surrounding focal plots (a *focal view*) at increasing spatial extents as a predictor variable, Condeso and Meentemeyer (2007) found an improvement in explanatory power of infection severity with increasing scale between 50 and 200-m. We found a similar relationship in our host habitat models, where model fit improved with increasing scale of host habitat measurements around the focal extent (though not in models at the individual stem level). Our correlation analysis of the proportion of host habitat and bay laurel density suggested that the high-resolution forest cover data may be a reasonable surrogate for host density, yet our measurements of host density revealed a relationship with disease intensity that was strongest at the focal extent, suggesting that local dispersal mechanisms may be driving disease dynamics in the *P. ramorum* pathosystem. In particular, the results from models of disease intensity at the 15-m extent suggest that there may be a spatial threshold between 60 and 140-m beyond which the impacts of host density on focal epidemiological processes are substantially diminished.

The fluctuation in the explanatory power of the host density models as scale increased from the aggregate perspective may in part be a product of our multiscale sampling design. That is, the 60-m extent has proportionally the least amount of data, with only four satellite plots plus the 15-m census informing the host density and disease intensity calculations for the entire area

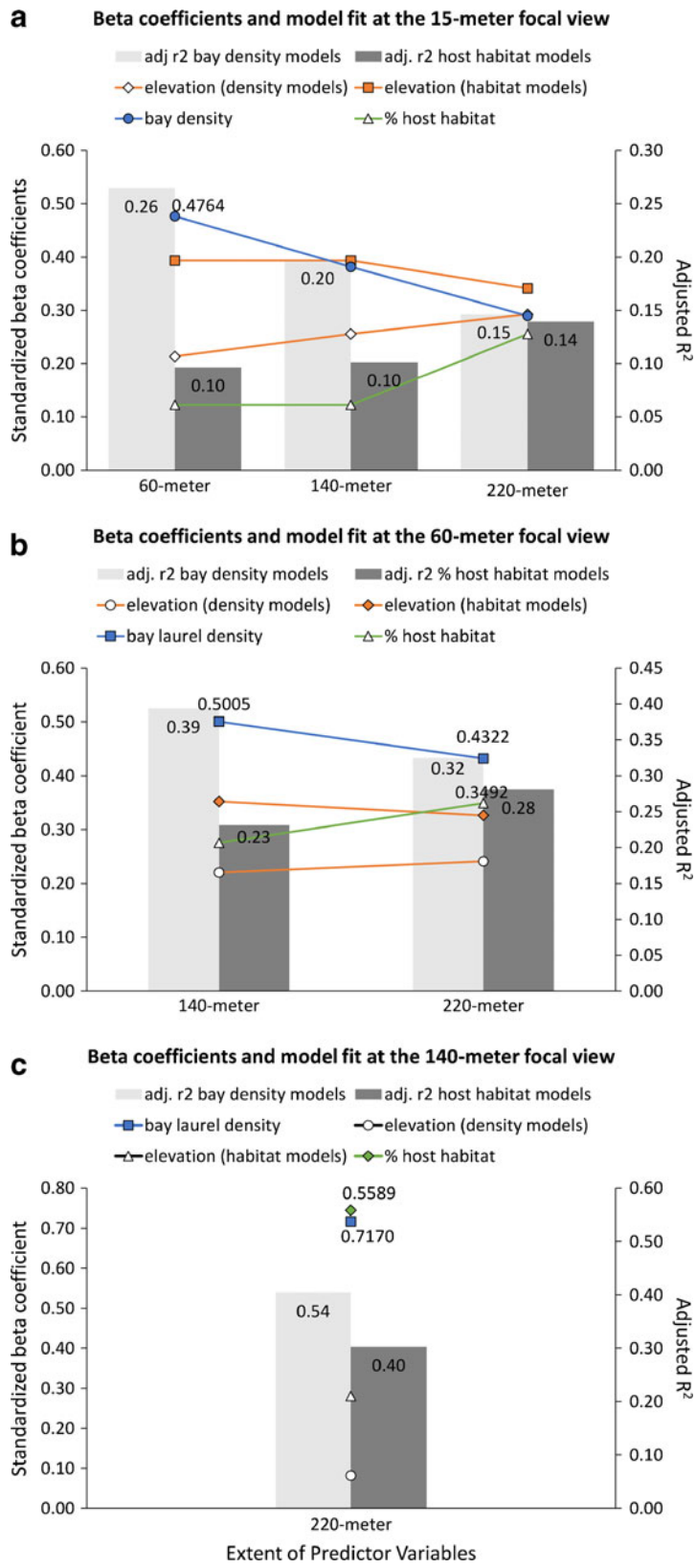

Fig. 5 Standardized coefficients and explanatory power for focal view models of disease intensity at 15-m, 60-m, and 140-m extent. The disease intensity at each extent is modelled as a function of elevation, and either bay laurel density or proportion of host habitat at each broader extent: **a** models of disease intensity at the 15-m extent; **b** models of disease intensity at the 60-m extent; **c** models of disease intensity at the 140-m extent. The magnitude of the coefficients are plotted as connected points on the primary y-axis on the left with precise values reported when $p<0.1$. Adjusted R^2 is plotted for models at each scale as columns using the secondary y-axis on the right, with the values for each model reported inside the end of each column

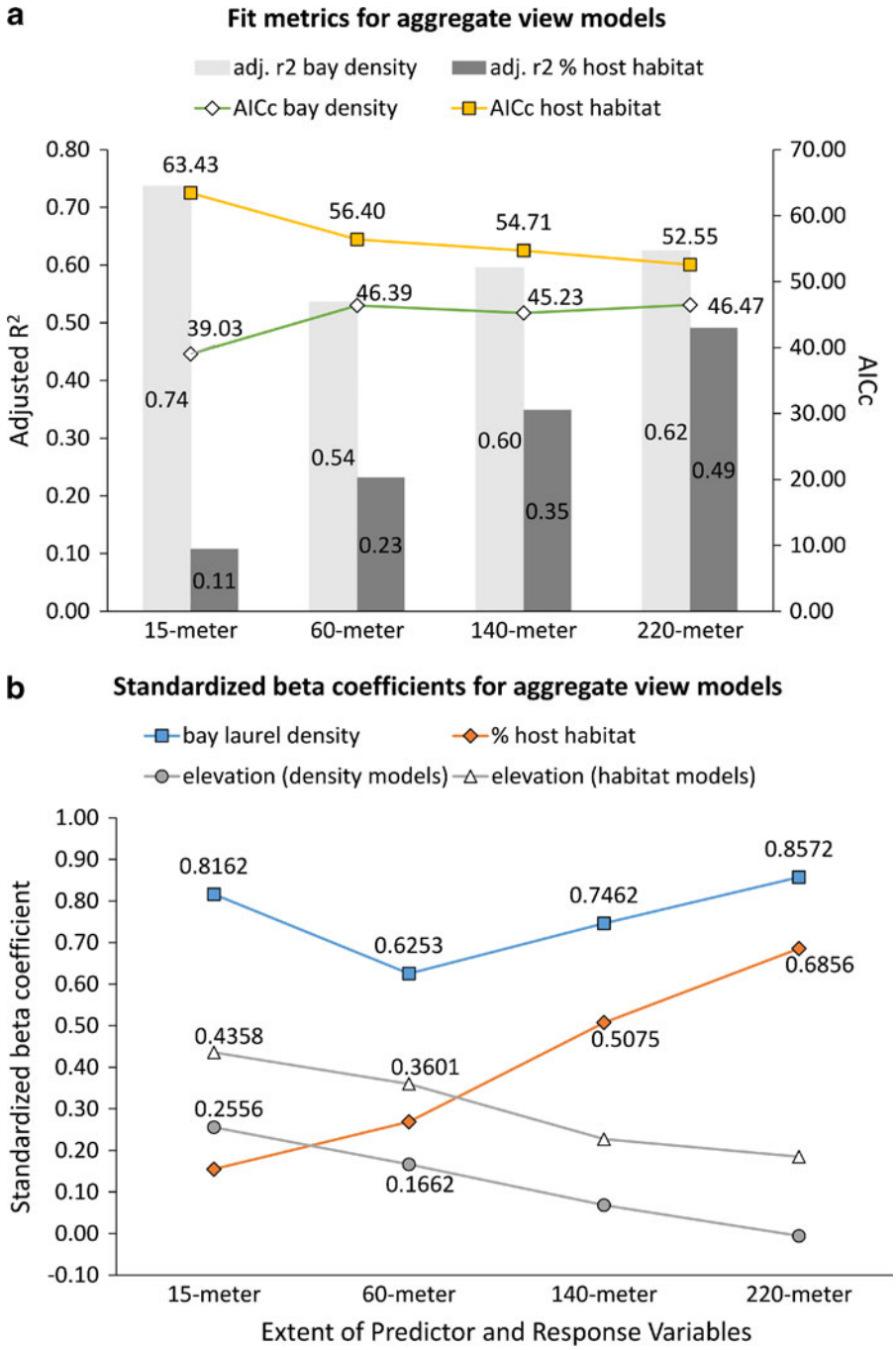

Fig. 6 Comparison of **a** model fit, and **b** relative variable influence of aggregate view models where the response variable and predictor variables (elevation and either bay laurel density or proportion of host habitat) were calculated at the same extent. While explanatory power (adjusted R^2) of models using bay laurel density increases with increasing extent from 60 to 220-m (though highest at 15-m), the model's goodness-of-fit (AICc) does not show notable improvement across the broader extents and is significantly better at the 15-m extent. Bay laurel density produced better model fit and greater explanatory power at each extent than host habitat, although goodness-of-fit and explanatory power of host habitat improved with increasing extent

(3600-m^2). With less data there is perhaps less variability in disease intensity captured at this extent, resulting in the least explanatory power of the models (lowest adjusted R^2), however, the fit of the model to the data is

in fact no worse than at the 140-m and 220-m extents (ΔAICc<2). Along these lines, the model at the 15-m extent is informed by the stem census at that scale (the most complete data for any extent), likely contributing to this being the model with the best performance. The results from this aggregate view indicate that the relationship between disease intensity and host density is best described by observation of these variables at the same extent.

The mechanism underlying the relationship between host density and disease intensity is in the processes of dispersal and transmission of inoculum. The presence of more competent hosts in a given area is likely to result in more inoculum production and therefore greater disease risk, but it may also be that host density alters physical microclimate conditions in the forest understory. This affects the physiology and vigour of pathogen reproduction and survival, as *P. ramorum* has been shown to be sensitive to moisture, humidity, and light levels (Englander et al. 2006; Tooley et al. 2008). The modest improvement of host habitat models across increasing extents may be due to this same mechanism, where greater forest cover leads to more favourable microclimate conditions for the pathogen.

At the individual level, host-pathogen interactions are driven by interactions between multiple environmental, ecological, and genetic factors (Morrison 1996; Anacker et al. 2008; Cobb et al. 2012). After accounting for individual stem characteristics (DBH) and physical properties (elevation) of the sites we found no effect of either host density or host habitat measured at any extent on the infection intensity of individual bay laurel stems. This would indicate that while the aggregate disease intensity at a site is influenced by the biotic conditions, the intensity of disease on individuals might be more strongly shaped by the individual's characteristics. The negative relationship observed between stem DBH and disease intensity could be in part due to the nearly complete fire suppression across this region over the past 65 years, which has resulted in the expansion of woodlands into areas previously dominated by grassland and chaparral (Meentemeyer et al. 2008b). As a result, the abundance and density of *P. ramorum* host species, including bay laurel, has increased over time because stems with smaller DBH are surviving longer (Meentemeyer et al. 2008b). Moreover, these smaller stems are generally located in the forest understory where they experience less exposure to solar irradiation, and overall conditions that are more favourable for pathogen survival. In turn, the positive relationship with elevation would indicate that individuals in locations that are generally cooler and wetter exhibit higher disease intensity.

Our findings add to the growing body of literature indicating that spread of *P. ramorum* is a predominantly local process (Davidson et al. 2005; Swiecki and Bernhardt 2007; Davidson et al. 2008; Mascheretti et al. 2008). Because plants are sessile and *P. ramorum* exhibits limited long-distance dispersal, the environmental conditions in small habitat patches may be sufficient to cause variation in infection on a local scale. The diminishing model fit we found between host density and disease intensity across increasing spatial scales from the focal perspective is similar to models of other passively-dispersed organisms (Mundt and Sackett 2012). Because of the opposite inference (increasing model performance with extent) from the host habitat models, we feel it is important to note that host habitat and host density may each be considered surrogate measures of infection pressure, but each provides distinct insights into disease dynamics (Burdon and Chilvers 1982). High resolution surfaces of host habitat may be an appropriate variable for examining disease-environment relationships in some instances, especially at broad spatial scales, but the measurement of factors such as host density that are more proximal to epidemiological processes can provide more meaningful insight in to the disease dynamics of wildland pathosystems.

Emerging infectious pathogens are recognized as a powerful force in shaping natural plant communities around the world, with long-term and largely unknown consequences to biodiversity and ecosystems services (Dinoor and Eshed 1984; Gilbert 2002; Burdon et al. 2006; Meentemeyer et al. 2012; Burdon et al. 2013). In a world with increased human mobility and international plant trade, it is imperative that we gain a better understanding of the diverse factors governing the dynamics of wildland pathosystems from local to global scales,

including interactions with other disturbances (Metz et al. 2011; Dillon et al. 2013). Here we have illustrated how the scale of observation – the spatial extent and the measurement detail – can influence conclusions drawn from epidemiological models of wildland pathosystems. Improved ecological forecasts of disease dynamics will require well-parameterized models that consider a range of causal factors with direct epidemiological relevance across multiple spatial scales.

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Appendix

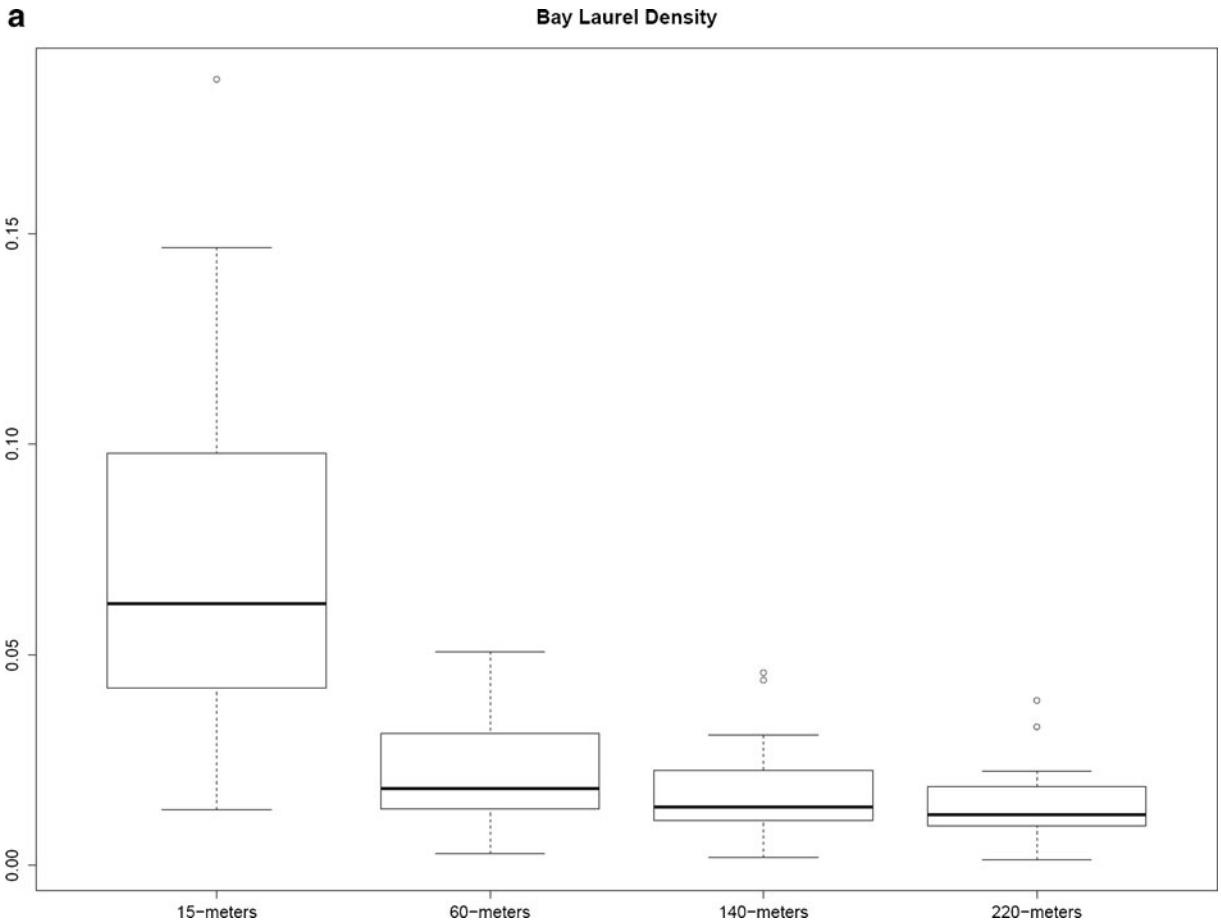
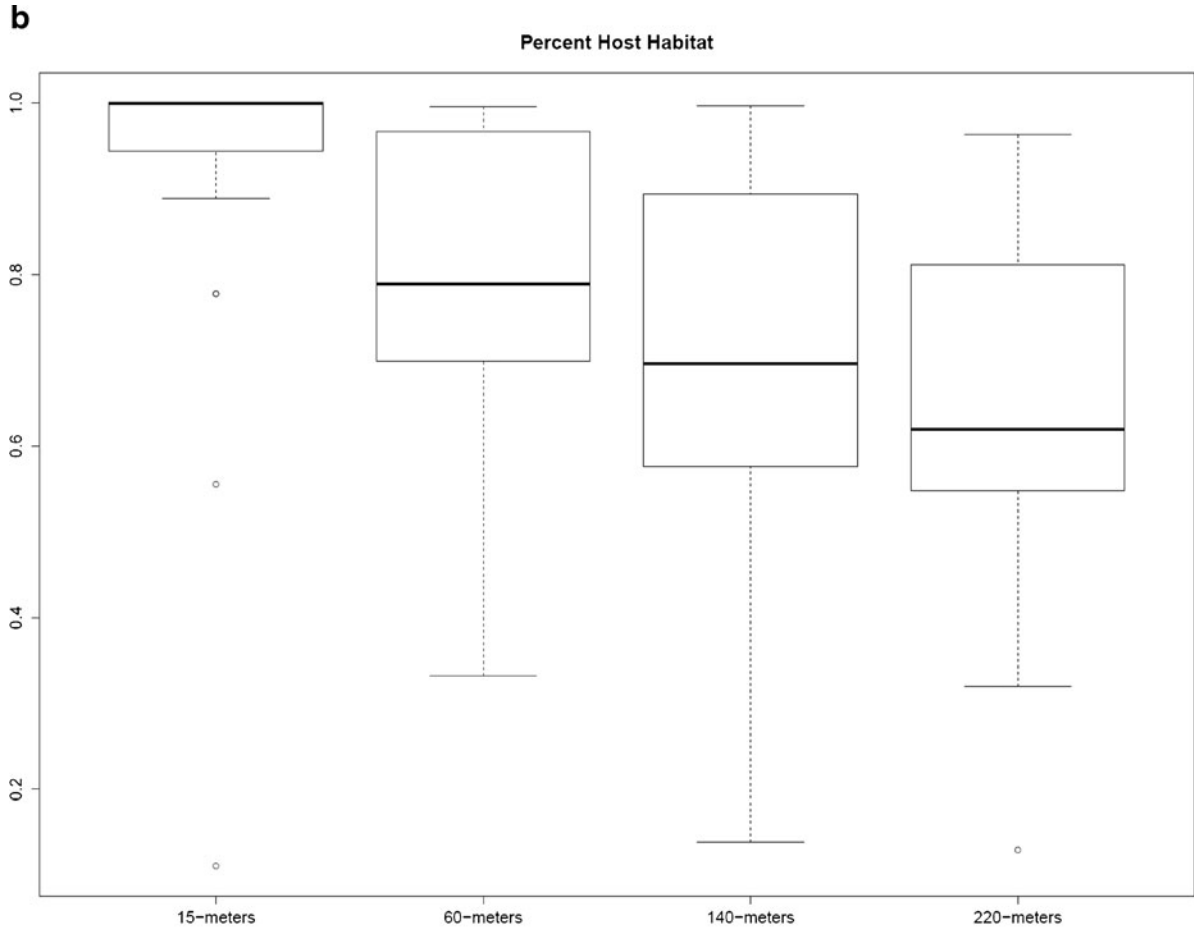


Fig. 7 Box-plots of **a** bay laurel density, **b** proportion of host habitat, and **c** symptomatic leaf counts at the four site-level extents across the 20 multiscale study sites



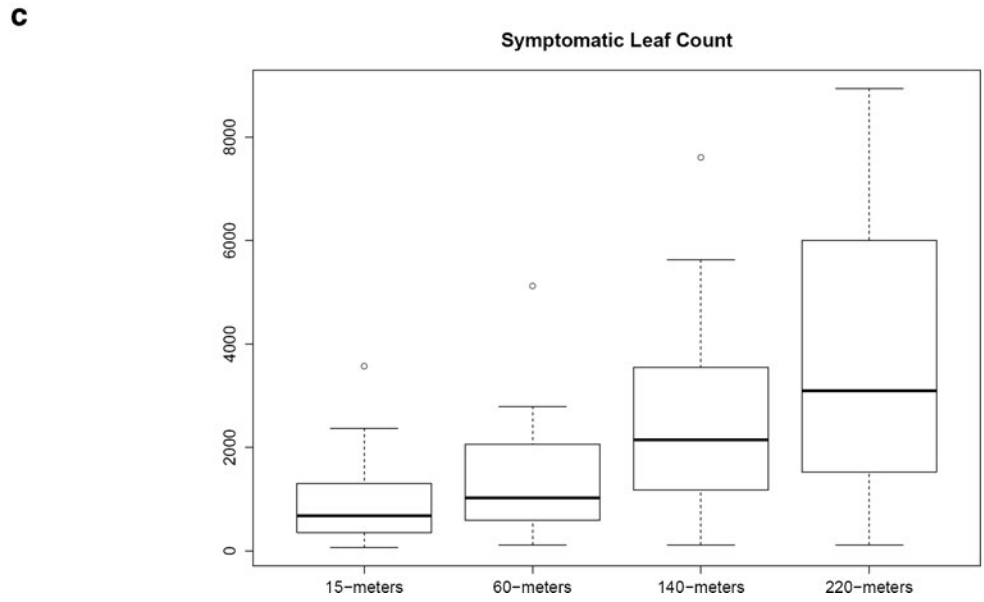


Fig. 7 (continued)

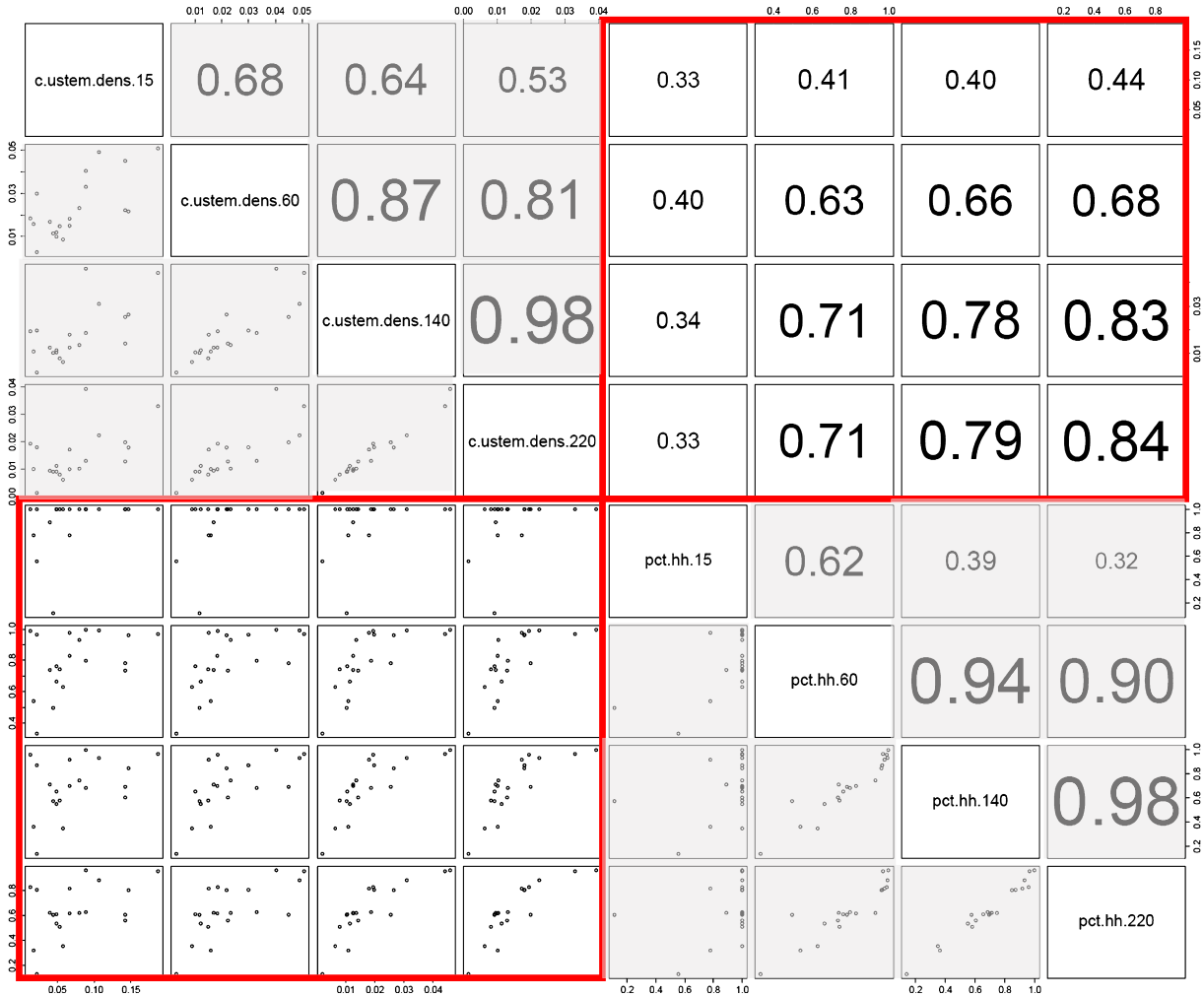


Fig. 8 Correlation plots and coefficients between bay laurel density (c.ustem.dens.X) and proportion of host habitat (pct.hh.X) across the four spatial extents of the multiscale study sites. The number at the end of each variable indicates the extent at which it was measured/calculated, e.g. 'pct.hh.60' is the proportion of host habitat calculated for the 60-m extent

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