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Scale dependence of disease impacts on quaking aspen (*Populus tremuloides*) mortality in the southwestern United States

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Abstract. Depending on how disease impacts tree exposure to risk, both the prevalence of disease and disease effects on survival may contribute to patterns of mortality risk across a species' range. Disease may accelerate tree species' declines in response to global change factors, such as drought, biotic interactions, such as competition, or functional traits, such as allometry. To assess the role of disease in mediating mortality risk in quaking aspen (*Populus tremuloides*), we developed hierarchical Bayesian models for both disease prevalence in live aspen stems and the resulting survival rates of healthy and diseased aspen near the species' southern range limit using 5088 individual trees on 281 United States Forest Service Forest Inventory and Analysis plots in the southwestern United States.

We found that disease prevalence depended primarily on tree size, tree allometry, and spatial variation in precipitation, while mortality depended on tree size, allometry, competition, spatial variation in summer temperature, and both temporal and spatial variation in summer precipitation. Disease prevalence was highest in large trees with low slenderness found on dry sites. For healthy trees, mortality decreased with diameter, slenderness, and temporal variation in summer precipitation, but increased with competition and spatial variation in summer temperature. Mortality of diseased trees decreased with diameter and aspen relative basal area and increased with mean summer temperature and precipitation. Disease infection increased aspen mortality, especially in trees of intermediate size and trees on plots at climatic extremes (i.e., cool, wet and warm, dry climates).

By examining variation in disease prevalence, mortality of healthy trees, and mortality of diseased trees, we showed that the role of disease in aspen tree mortality depended on the scale of inference. For variation among individuals in diameter, disease tended to expose intermediate-size trees experiencing moderate risk to greater risk. For spatial variation in summer temperature, disease exposed lower risk populations to greater mortality probabilities, but the magnitude of this exposure depended on summer precipitation. Furthermore, the importance of diameter and slenderness in mediating responses to climate supports the increasing emphasis on trait variation in studies of ecological responses to global change.

Key words: allometry; aspen; climate change; competition; disease; Forest Inventory and Analysis; hierarchical Bayesian modeling; Populus tremuloides; *tree mortality*.

Introduction

Biotic agents can play a major role in tree mortality across species and regions (Allen et al. 2010). In particular, disease may increase tree vulnerability to drought-induced mortality by contributing to declines in water and carbon relations (McDowell et al. 2008, Marchetti et al. 2011, Anderegg et al. 2012). Assessing the contribution of disease to tree mortality risk, separate from drought-induced mortality, is complicated, because the prevalence of disease may vary with some of the same factors influencing mortality and may not uniformly increase mortality risk in all individuals or populations. If mortality risk is not amplified uniformly by disease prevalence, disease might act to push trees already at risk over a demographic edge or expose trees not otherwise vulnerable to greater mortality risk (Fig. 1). Both the prevalence of disease and the influence of disease on tree vulnerability likely contribute to observed patterns of mortality risk across a species' range.

Quaking aspen (*Populus tremuloides*; see Plate 1) is a widely distributed North American tree species (Appendix A: Fig. A1). However, aspen is experiencing a substantial decline in the southern portions of its range (sudden aspen decline; Kulakowski et al. 2013, Worrall et al. 2013) and disease prevalence (i.e., the observation of disease in individual aspen trees) is chronically high in aspen stands of the southwestern United States (Shepperd et al. 2006). Individual aspen tree deaths are often attributed, in part, to infection by fungal pathogens associated with butt rot and stem cankers, such as

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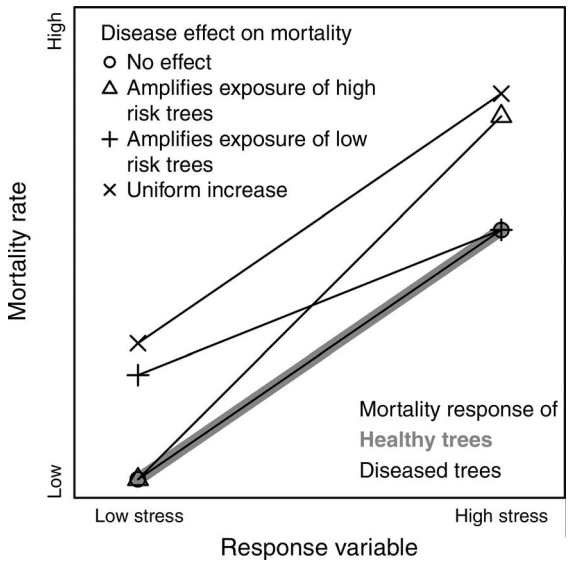

FIG. 1. Conceptual diagram illustrating alternative hypotheses about disease effects on quaking aspen (*Populus tremuloides*) mortality across stress gradients represented by climatic or stand structural conditions (e.g., increasing drought or decreasing tree size; *x*-axis). Disease effects can be characterized by comparing mortality rates of healthy and diseased trees. The four potential effects that are represented are no disease effect, disease amplifies mortality exposure for high stress trees only, disease amplifies mortality exposure for low stress trees only, and uniform increases in mortality rates with disease.

Armillaria and *Cytospora* spp. (Frey et al. 2004, Shepperd et al. 2006, Marchetti et al. 2011). Infected trees may be less capable of coping with drought, contributing to sudden aspen decline (Hogg et al. 2008, Anderegg et al. 2013*b*, Worrall et al. 2013). Although sudden aspen decline has increased in scope during recent decades (Worrall et al. 2008), the individual and combined contributions of climatic and disease effects are unresolved. Given that aspen forests account for a large portion of the region's biodiversity and carbon storage, continued declines represent a major threat to Rocky Mountain landscapes (Huang and Anderegg 2011, Kuhn et al. 2011).

Intrinsic factors, such as competition (Kashian et al. 2007, Zegler et al. 2012), extrinsic forces, such as drought (Rehfeldt et al. 2009, Worrall et al. 2010, 2013), and, rarely, the interactions between the two (Bell et al. 2014) have been implicated in recent declines. While dendrochronological work has corroborated some of these patterns at the individual scale (Hogg et al. 2002, 2008), these studies have not explicitly examined how individual variation in traits alters mortality risk. In particular, individual differences in size and allometry can result in disparate levels of stress under similar growing conditions (Callaway et al. 1994, Cavender-Bares and Bazzaz 2000, Delucia et al. 2000). Aspen populations in the southwestern United States are thought to be particularly vulnerable to drought (Rehfeldt et al. 2009, Worrall et al. 2013) and to have experienced dramatic intensification of drought stress (Williams et al. 2012). We know of no study simultaneously quantifying variation in aspen mortality rates as a function of individual traits, competitive environment, and moisture limitations, let alone the role that disease plays in mediating them.

To better understand the impact of disease in mediating individual tree mortality, we examined both disease prevalence and the impact of disease on mortality rates in aspen populations of the southwestern United States. Using repeat measurements from 281 U.S. Forest Service Forest Inventory and Analysis (FIA) plots, we developed hierarchical Bayesian models to examine variation in disease prevalence in live trees and consequent survival patterns of 5088 individual trees in Colorado, New Mexico, and Arizona, USA. Our objectives were to quantify the influence of individual traits (e.g., diameter and slenderness), competitive environment (e.g., stand basal area, aspen dominance, and site quality), and moisture limitation (e.g., topography and climate) on observed patterns of disease and mortality; and to assess the role of disease in modifying aspen mortality risk (Fig. 1). By examining both individual-level disease and mortality, we show the importance of accounting for scale and biotic influences in studies of demographic responses to moisture stress.

METHODS

Study area and data collection

Though aspen is distributed across many of the mountains of the western United States, we focused our work on populations measured in the southwestern United States (Appendix A: Fig. A1). The study region included portions of southwest Colorado, northern Arizona, and northern New Mexico, where FIA plots were remeasured using the same sampling design for adult trees (diameter at breast height $\geq$12.7 cm) during two inventories; had no recent record of fire or timber harvest; and included initial measurements of live tree damage. In this region, aspen are a substantial component of post-disturbance landscapes where they can form dense, mono-specific stands (Kulakowski et al. 2013, Shinneman et al. 2013). When disturbances are absent for prolonged periods of time, shade-tolerant conifers encroach, and eventually replace, all but the largest individuals in many aspen stands.

FIA plots were distributed across the United States' forested areas (approximately one plot per 2500 ha), though only a subset of plots included measurements of disease status on live aspen trees, had no record of recent fire or timber harvest, and were remeasured during a later inventory. Each plot k includes four 168.3-m^2 subplots j on which environmental conditions, such as slope and aspect, were recorded. Every tree i at least 12.7 cm in diameter at breast height was identified to species and measured for many attributes, including diameter (d_{ijk}; cm) and height (m). While remeasurements of plots were easily identified, we used a tree matching algorithm

TABLE 1. Description of covariates and interaction terms used to explain variation in individual-level mortality across sites of quaking aspen (*Populus tremuloides*).

Effects	Explanations
Main	
Diameter, d_{ijk} (cm)	Diameter at breast height (1.5 m above ground) of tree i in subplot j on plot k (i.e., tree ijk).
Slenderness, s_{ijk} (m/cm)	The ratio of tree height (m) to tree diameter (cm) for tree ijk.
Competition index, c_{ijk} (m^2)	Summed basal area of all trees in subplot j on plot k (i.e., subplot jk) taller than individual ijk.
Relative basal area, b_{jk}	The proportion of basal area comprised by aspen in subplot jk.
Solar radiation index, r_{jk}	A metric positively related to exposure based on slope, aspect, and latitude for subplot jk.
Summer temperature, T_k (°C)	40-year mean summer temperature (June to August) for plot k.
log(summer precipitation), P_k (measured in mm)	40-year mean log summer precipitation (June to August) for plot k.
Summer temperature deviation, t_k (°C)	Mean deviation of summer temperature from T_k during the past 5 years for plot k.
log(summer precipitation) deviation, p_k (measured in mm)	Mean deviation of log summer precipitation from P_k during the past 5 years for plot k.
Quadratic	
$d_{ijk} \times d_{ijk}$, $s_{ijk} \times s_{ijk}$, $c_{ijk} \times c_{ijk}$, $b_{jk} \times b_{jk}$, $r_{jk} \times r_{jk}$, $T_k \times T_k$, $P_k \times P_k$, $t_k \times t_k$, and $p_k \times p_k$	
Interaction	
1) Slenderness influences vulnerability to drought ($s_{ijk} \times r_{jk}$, $s_{ijk} \times T_k$, $s_{ijk} \times P_k$, $s_{ijk} \times t_k$, and $s_{ijk} \times p_k$)	Tree slenderness has been related to adaptation to drought as well as responses to drought in the short-term (Delucia et al. 2000, Worrall et al. 2010).
2) Large trees have more resources, but have more to maintain ($d_{ijk} \times c_{ijk}$, $d_{ijk} \times r_{jk}$, $d_{ijk} \times T_k$, $d_{ijk} \times P_k$, $d_{ijk} \times t_k$, and $d_{ijk} \times p_k$)	Large trees may be less sensitive if they have greater access to limiting resources, but may also be more vulnerable, as they must acquire more resources to meet respiration demands (Cavender-Bares and Bazzaz 2000).
3) Competition may alter stress differentially ($c_{ijk} \times b_{jk}$, $b_{ijk} \times r_{jk}$, $c_{ijk} \times r_{jk}$, $c_{ijk} \times T_k$, $c_{ijk} \times P_k$, $c_{ijk} \times t_k$, and $c_{ijk} \times p_k$)	Competitive stress depends on the identity of the competitors and the degree to which resources are limiting (Bell et al. 2014).
4) Climate is multivariate and depends on topographic exposure ($r_{jk} \times T_k$, $r_{jk} \times P_k$, $r_{jk} \times t_k$, $r_{jk} \times p_k$, $T_k \times P_k$, $T_k \times t_k$, $P_k \times p_k$, and $t_k \times p_k$)	The exposure, as well as long-term mean and short-term deviations in summer temperature and precipitation, combine to influence water stress (Anderegg et al. 2013*a*).

Notes: Variables shown are diameter (d_{ijk}), slenderness (s_{ijk}), competitive index (c_{ijk}), aspen relative basal area (b_{jk}), solar radiation index (r_{jk}), 40-year average summer temperature (T_k), 40-year average log(summer precipitation) (P_k), 5-year deviation in summer temperature (t_k), and 5-year deviation in log(summer precipitation) (p_k) on disease prevalence and mortality in aspen. Interaction effect groups 1–3 deal with how tree individual environment influences responses to stress, such as competition, exposure, and climate. Interaction group 4 deals with interactions between physical environmental variables.

(Pollard et al. 2005) and visual inspection of the data to link tree measurements across inventories from the FIA database. From these data, we calculated several individual- and plot-level measures of aspen tree traits and competitive environment (see Table 1 for details). The ratio of height to diameter, or tree slenderness (s_{ijk}; m height/cm diameter), was negatively related to aspen decline in southwest Colorado (Worrall et al. 2010). We accounted for the asymmetric nature of competition in forest ecosystems (Watt 1947) by calculating an individual-based competition index (c_{ijk}; m^2), though this variable may also represent other density-dependent effects. To account for the influences of community composition on the competitive environment, we calculated aspen relative basal area (b_{jk}; unitless). An interaction between c_{ijk} and b_{jk} allows for both inter- and intra-specific density dependent effects to be represented. Plot locations were provided with error added (±1 km) to preserve private-property owner privacy (Woudenberg et al. 2010). While subplot-level measures of site quality (e.g., site index) were available, analyses indicated that this variable was not useful in describing either disease or survival processes and was not included in our statistical analyses.

We focused on a subset of plots measured initially between 1995 and 2002 and measured again between 2001 and 2012, because measurements included visual assessment of damage to trees, including disease, and remeasurement allowed us to examine the mortality process. All plots used the same sampling design. There were 5088 individual aspen trees on 281 plots (at least two live aspen per plot) that were alive in the first time step, measured live or dead during the following measurement, and had a damage type assigned while still alive. Of these trees, 22% had some sign of disease, 99% of which were stem rots, butt rots, or cankers, to which aspen mortality is commonly attributed (Shepperd et al. 2006). Because observation of disease infection relied on imperfect visual assessment, disease prevalence estimates are likely an underprediction of

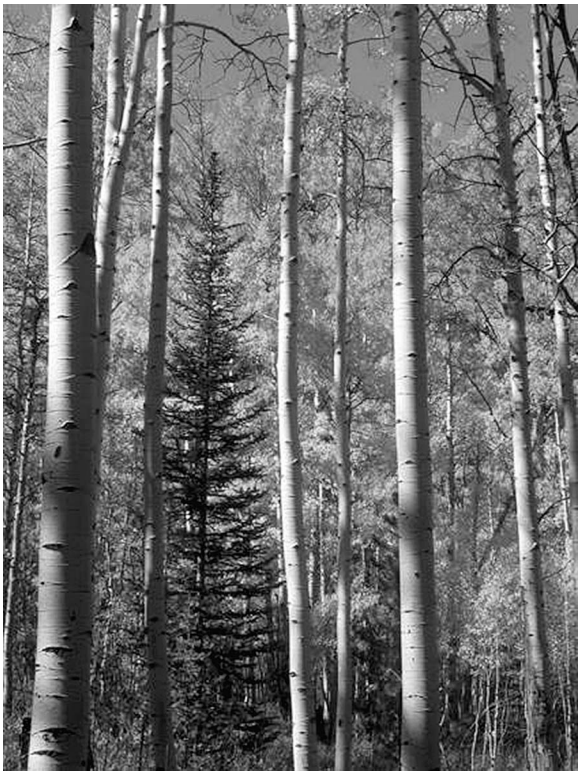

PLATE 1. A stand of quaking aspen in Summit County, Colorado, USA showing the initial phases of conifer encroachment. A color version is available in Appendix C. Photo credit: J. B. Bradford.

actual infection rates. However, fruiting bodies for cankers and butt rots, where present, are easily identified, suggesting that relatively advanced stages of these diseases were observed with high fidelity.

To represent spatial and temporal variation in climate, 40-year means and 5-year deviations (5-year mean minus 40-year mean) in summer temperature (T_k and t_k, respectively) and log(summer precipitation) (P_k and p_k, respectively) were calculated based on 4-km (2.5-arcminute) annual historical climate data (PRISM Climate Group 2012; data *available online*).[4] Previous research indicated that aspen physiological responses to climate are correlated to summer temperature and precipitation (Anderegg et al. 2013*a*). For each climate variable, the period for which means and deviations were calculated ended during the year preceding plot measurement. Given that our study area covered a broad geographic area, that fine-scale climate data and FIA plot locations can both introduce errors, and that annual data were needed to calculate climatic deviations, we contend that the 40-year means and 5-year deviations based on 4-km resolution data represent broadscale spatiotemporal climatic gradients. While the spatial resolution of these data are not likely to represent the climatic complexity of our study area (Ashcroft 2010), increasingly small spatial-scale climate data products introduce error into ecological studies (Bucklin et al. 2012, Bishop and Beier 2013), as do errors in spatial locations of plots characteristic of FIA data (Gibson et al. 2014). To capture small-scale, topographic variation in moisture, we used slope, aspect, and latitude for each subplot to calculate solar radiation index (r_{jk}; unitless), which is high for steep, south-facing slopes, especially at low latitudes (Keating et al. 2007).

[4] ftp://prism.nacse.org/monthly/

Traits, competition, and drought influences on stress

Individual traits, competitive environment, and moisture limitation can all alter tree stress and influence both the prevalence of disease and mortality in aspen. Functional traits are increasingly incorporated into studies of species response to climate change (e.g., Buckley et al. 2011, Eckhart et al. 2011). In the case of trees, both size (Cavender-Bares and Bazzaz 2000) and slenderness (Callaway et al. 1994, Delucia et al. 2000) influence responses to resource limitation. In conjunction with these traits, competitive environment can mediate tree mortality responses to climatic stressors (Kashian et al. 2007, Bell et al. 2014). Finally, drought is often implicated in sudden aspen decline (Hogg et al. 2002, 2008, Rehfeldt et al. 2009, Worrall et al. 2010, 2013), either because populations in dry regions (e.g., long-term mean summer temperature is high and summer precipitation is low) are at greater risk, or because short-term increases in drought elevate risk above long-term averages. Therefore, we examined the main and quadratic effects of diameter (d_{ijk}), slenderness (s_{ijk}), competitive index (c_{ijk}), aspen relative basal area (b_{jk}), solar radiation index (r_{jk}), 40-year average summer temperature (T_k), 40-year average log(summer precipitation) (P_k), 5-year deviation in summer temperature (t_k), and 5-year deviation in log(summer precipitation) (p_k) on disease prevalence and mortality in aspen (Table 1). We examined interactions representing the potential importance of allometry, tree size, competitive environment, and climatic complexity in mediating climatic impacts on disease and mortality. We limited our analysis to the interactions falling into one of these four groups to reduce model complexity and to focus on biologically realistic interactions.

Model development

To evaluate the influences of traits, competition, and climate on disease and mortality, we developed two hierarchical Bayesian statistical models. In both cases, we included plot-level random effects to account for variation in disease prevalence and mortality not characterized by the covariates. This plot-level variation may arise from many sources, including unmeasured or unmeasurable environmental and genetic variation, such as variation in the vulnerability of different aspen stands due to their clonal nature. The processes associated with random effects can play a major role in outcomes for

individuals, populations, or species (Clark et al. 2010). The following models assume that random effects, subplot-level variables, plot-level variables, and associated interaction terms account for dependencies among individuals within the same population, meaning that we assume that observations on individuals are conditionally independent. For observations of disease prevalence, we employed a hierarchical logistic regression model. If the prevalence of disease damage in live trees y_{ijk} was either observed ($y_{ijk} = 1$) or not ($y_{ijk} = 0$) for tree i in subplot j and plot k, then we can model the prevalence of disease damage y_{ijk} as $y_{ijk} \sim$ Bernoulli(θ_{ijk}), where

$$\theta_{ijk} = (1 + \exp[-(x_{ijk}\beta + \alpha_k)])^{-1} \quad (1)$$

was the probability of disease being observed for individual ijk, x_{ijk} is the length M vector of covariates, β is the length M vector of parameter values, and α_k is a plot-level random effect distributed as $N(0, \tau^2)$, and τ^2 is the random effect variance.

For mortality, we wished to differentiate between diseased and healthy trees while still accounting for stand-level differences not otherwise characterized by the covariates in the model. Because the sample intervals dt_k (years) differed among plots, we also needed to account for differing lengths of time between sample events. Therefore, we developed a hierarchical Bayesian survival model with shared plot-level random effects. For each diseased or healthy individual, we know whether the individual tree ijk died ($z_{ijk} = 0$) or survived ($z_{ijk} = 1$) during the measurement interval dt_k. We modeled survival as $z_{ijk} \sim$ Bernoulli(ρ_{ijk}) such that

$$\rho_{ijk} = \exp\Big(-\exp[1(y_{ijk} = 1) \times x_{ijk}\lambda + 1(y_{ijk} = 0) \times x_{ijk}\mu + \gamma_k] \times dt_k\Big) \quad (2)$$

where $x_{ijk}\lambda$ accounts for the fixed effects for diseased trees, λ is a length M parameter vector for diseased trees, $x_{ijk}\mu$ accounts for the fixed effects for healthy trees, μ is a length M parameter vector for healthy trees, $1(\bullet)$ is an indicator function which equals unity if $\bullet$ is true and zero otherwise, and γ_k are the plot-level random effects for survival distributed as $N(0, \omega^2)$, and ω^2 is the random effect variance. Similar to the disease model, the plot-level random effects γ_k represents stand-level variation in survival not otherwise characterized by the covariates.

We adopted a two-stage method for model fitting where we employed a probabilistic model selection method and incorporated plot-level random effects to the selected model to account for differences among plots not represented by the covariates. To identify the role of individual traits, competitive environment, and drought on both disease and mortality, we examined whether main, quadratic, and interaction terms should be included in a model by explicitly estimating the marginal probability that any given covariate was included in the model (Kuo and Mallick 1998, O'Hara and Sillanpää 2009). We used the method of model selection described by Kuo and Mallick (1998), which simultaneously estimates regression parameters and the associated probabilities of inclusion in the model. Only fixed effects were incorporated in the model selection stage (i.e., $\alpha_k = 0$ and $\gamma_k = 0$), because we found that random effects tended to absorb all plot-level variation, resulting in very low probabilities of inclusion for climate variables, and, to a lesser extent, solar radiation index and aspen relative basal area, compared to model selection with fixed effects only. After model selection, we fit the median probability model (model including covariates with marginal probability of inclusion 0.50 or greater) with random effects (see Eqs. 1 and 2). Detailed descriptions of model selection and fitting can be found in Appendix B.

Given that the survival process very likely differs between healthy and diseased individuals, the mortality risk for any given individual depends not only on the survival process but also the disease prevalence. Differences in the mortality responses of healthy vs. diseased trees to traits or environment can provide evidence regarding the role that disease plays in aspen mortality (Fig. 1). For example, if disease amplifies mortality in high stress trees, then diseased trees experience exposure to stress more acutely. Conversely, if disease amplifies mortality in low stress trees, then disease acts to expose individuals to stress they would otherwise not experience. All statistical analyses were performed in R (R Development Core Team 2010) using JAGS (Plummer 2003). Covariates were relativized to range from zero to unity to improve model convergence and interactions used centered covariates to reduce multicollinearity.

Results

Our results indicated negative effects of tree slenderness (s_{ijk}) and mean summer precipitation (P_k), a positive effect of tree diameter (d_{ijk}), and weak trends of other variables on disease prevalence (Fig. 2, Table 2, and Appendix B: Table B1). While other variables were included in the median probability model (Table 2), they had little effect (Fig. 2c–f) or exhibited large uncertainties (i.e., wide credible intervals, Fig. 2h and i). Disease prevalence in aspen depended on nearly all the proposed interactions (Table 2), but few of the parameter estimates differed from zero; only in four cases did 95% credible intervals exclude zero (Appendix B: Table B1, Fig. B1).

Ten-year mortality rates for healthy trees declined with diameter (d_{ijk}), slenderness (s_{ijk}), and deviations in summer precipitation (p_k), increased with competition index (c_{ijk}) and mean summer temperature (T_k), exhibited a unimodal response with mean summer precipitation (P_k), and exhibited no clear responses to relative aspen basal area, (b_{jk}) solar radiation index (r_{jk}), and deviations in summer temperature (t_k) (Fig. 3). In comparison to the disease prevalence model, the

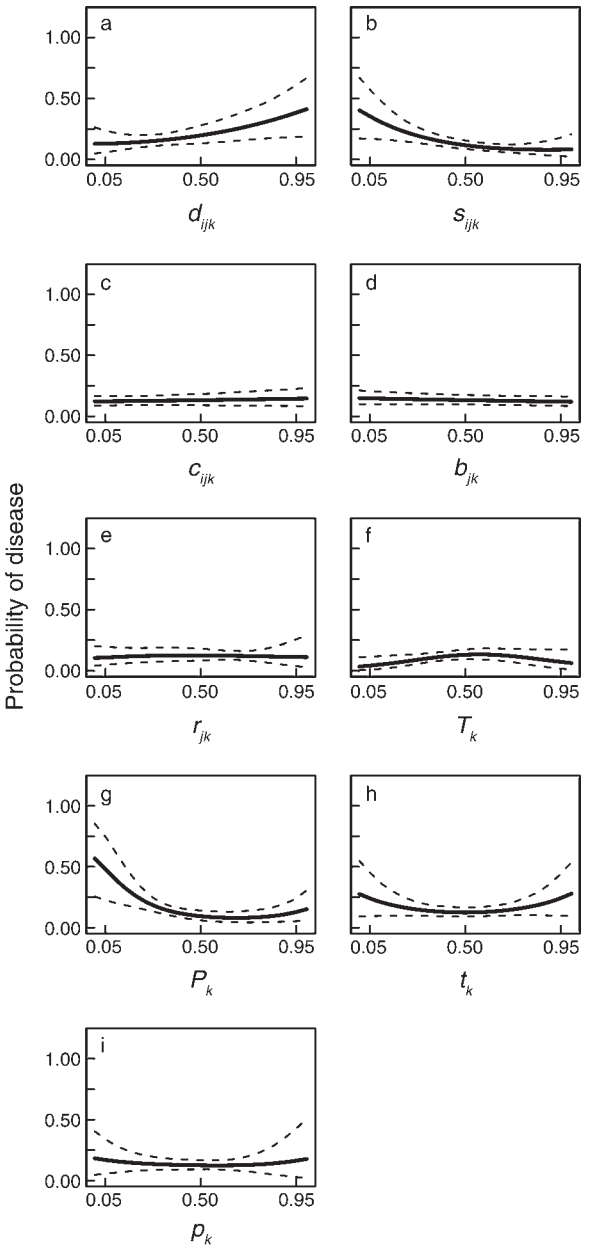

FIG. 2. Mean (solid line) and 95% credible intervals (dashed lines) predicted probabilities of disease. To make predictions, all variables except for the variable of interest were held constant at their means. Variables shown are (a) diameter (d_{ijk}), (b) slenderness (s_{ijk}), (c) competitive index (c_{ijk}), (d) aspen relative basal area (b_{jk}), (e) solar radiation index (r_{jk}), (f) 40-year average summer temperature (T_k), (g) 40-year average log(summer precipitation) (P_k), (h) 5-year deviation in summer temperature (t_k), and (i) 5-year deviation in log(summer precipitation) (p_k) on disease prevalence and mortality in aspen.

mortality response of healthy trees depended on fewer interactions (Table 2), but eight of these were different from zero (Appendix B: Table B2). Furthermore, the strength of the interactions, as measured by the degree of curvature in bivariate predictions (Figs. 4 and 5), was greater compared to interactions for disease prevalence (Appendix B: Fig. B1). Ten-year mortality rates for healthy trees were greatest for small trees experiencing intense competition from above on plots with high deviations in summer precipitation and low solar radiation index (Fig. 4a–c). Solar radiation index altered the summer temperature at which mortality started to increase (Fig. 4d). The greatest positive effects of competition on mortality were observed when temperature and precipitation deviations were low (i.e., negative) and when aspen relative density was high (Fig. 4e–g). In healthy trees, mortality increased with long-term mean summer temperature, but only long-term mean summer precipitation was moderate (Fig. 5a).

For diseased trees, the four covariates that were retained in the selected model were diameter, aspen relative basal area, and 40-year mean summer temperature and precipitation, as well as an interaction between mean summer temperature and precipitation (Table 2, Appendix B: Table B3). Ten-year mortality rates for diseased trees increased with mean summer temperature (Fig. 3a) and decreased with diameter (Fig. 3b). Weaker trends were observed for mean summer precipitation (increase; Fig. 3c) and aspen relative basal area (decrease; Fig. 3h). Mortality rates were higher for diseased trees than healthy trees for all but the warmest climates (Fig. 3a) and smallest trees (Fig. 3b). Disease increased mortality rates for trees independent of long-term mean summer precipitation (Fig. 3c) and aspen relative basal area (Fig. 3h). Disease also increased mortality for aspen with moderate slenderness (Fig. 3d), moderate short-term deviations in summer temperature (Fig. 3e), moderate solar radiation index (Fig. 3i), low competition index (Fig. 3f), and moderate to high 5-year deviations in precipitation (Fig. 3g). For diseased trees, only the interaction between long-term mean summer temperature and precipitation was retained in our model (Fig. 5b).

DISCUSSION

For aspen trees of the southwestern United States, our results indicated that disease exposed low- to moderate-stress individuals to greater risk, depending on the scale of observation. Spatial, rather than temporal, variations in summer temperature and precipitation were most strongly related to aspen mortality. Diseased trees exhibited higher 10-year mortality rates compared to healthy trees, particularly in warm, dry climates (Fig. 5c). If hydraulic failure associated with atmospheric drought is the primary cause of recent aspen mortality (Anderegg et al. 2012), disease might be amplifying this effect in areas with low precipitation and high temperatures during the growing season. This seems to corroborate predictions of major losses of aspen in low-elevation habitats where aspen experience relatively high temperatures and low precipitation (Rehfeldt et al.

TABLE 2. Posterior probability of parameter inclusion for each interaction term for models examining disease prevalence (y_{ijk}), mortality for healthy trees ($z_{ijk} \mid y_{ijk} = 0$), and mortality for unhealthy trees ($z_{ijk} \mid y_{ijk} = 1$).

Interactions	Probability of inclusion		
	Disease	Mortality (healthy)	Mortality (unhealthy)
Main effects			
d_{ijk}	**1.00**	**1.00**	**1.00**
s_{ijk}	**1.00**	**1.00**	0.48
c_{ijk}	**0.67**	**1.00**	0.34
b_{jk}	**0.60**	**0.90**	**0.52**
r_{jk}	**1.00**	**1.00**	0.22
T_k	**1.00**	**1.00**	**0.93**
P_k	**1.00**	**1.00**	**0.62**
t_k	**1.00**	**0.94**	0.24
p_k	**0.99**	**1.00**	0.08
Quadratic effects			
$d_{ijk} \times d_{ijk}$	**0.59**	**0.55**	0.28
$s_{ijk} \times s_{ijk}$	**0.53**	**1.00**	0.23
$c_{ijk} \times c_{ijk}$	**0.42**	**0.96**	0.14
$b_{jk} \times b_{jk}$	0.33	**0.89**	0.24
$r_{jk} \times r_{jk}$	**0.55**	**0.60**	0.08
$T_k \times T_k$	**0.97**	0.45	0.29
$P_k \times P_k$	**1.00**	**1.00**	0.38
$t_k \times t_k$	**0.76**	**0.93**	0.10
$p_k \times p_k$	**0.63**	0.49	0.03
(1) Slenderness influences vulnerability to drought			
$s_{ijk} \times r_{jk}$	**0.55**	**0.51**	0.05
$s_{ijk} \times T_k$	**0.63**	**0.53**	0.25
$s_{ijk} \times t_k$	**0.77**	**0.67**	0.13
$s_{ijk} \times P_k$	**0.90**	**0.55**	0.13
$s_{ijk} \times p_k$	**0.57**	**0.67**	0.02
(2) Large trees have more resources, but more to maintain			
$d_{ijk} \times s_{ijk}$	**0.62**	**0.74**	0.39
$d_{ijk} \times c_{ijk}$	0.37	**1.00**	0.14
$d_{ijk} \times r_{jk}$	**0.94**	**0.99**	0.09
$d_{ijk} \times T_k$	**0.55**	0.48	0.31
$d_{ijk} \times t_k$	**0.60**	**0.55**	0.11
$d_{ijk} \times P_k$	**0.58**	**0.52**	0.21
$d_{ijk} \times p_k$	**0.76**	**0.98**	0.03
(3) Competition may alter stress differentially			
$c_{ijk} \times b_{jk}$	0.21	**0.59**	0.07
$c_{ijk} \times r_{jk}$	**0.58**	0.42	0.04
$c_{ijk} \times T_k$	**0.67**	0.49	0.21
$c_{ijk} \times t_k$	0.38	**0.60**	0.09
$c_{ijk} \times P_k$	0.45	0.43	0.08
$c_{ijk} \times p_k$	0.39	**0.61**	0.01
(4) Climate is multivariate and depends on topographic exposure			
$r_{jk} \times T_k$	**0.98**	**0.98**	0.10
$r_{jk} \times t_k$	**1.00**	0.41	0.03
$r_{jk} \times P_k$	**0.83**	**0.50**	0.05
$r_{jk} \times p_k$	**0.83**	**0.51**	0.01
$T_k \times P_k$	**1.00**	**0.99**	**0.61**
$T_k \times t_k$	**0.99**	0.45	0.16
$P_k \times p_k$	**0.94**	0.47	0.04
$t_k \times p_k$	**0.59**	0.45	0.01

Notes: Bold values indicate parameter inclusion in the median probability model (probability of inclusion ≥ 0.5). See Table 1 for variable definitions.

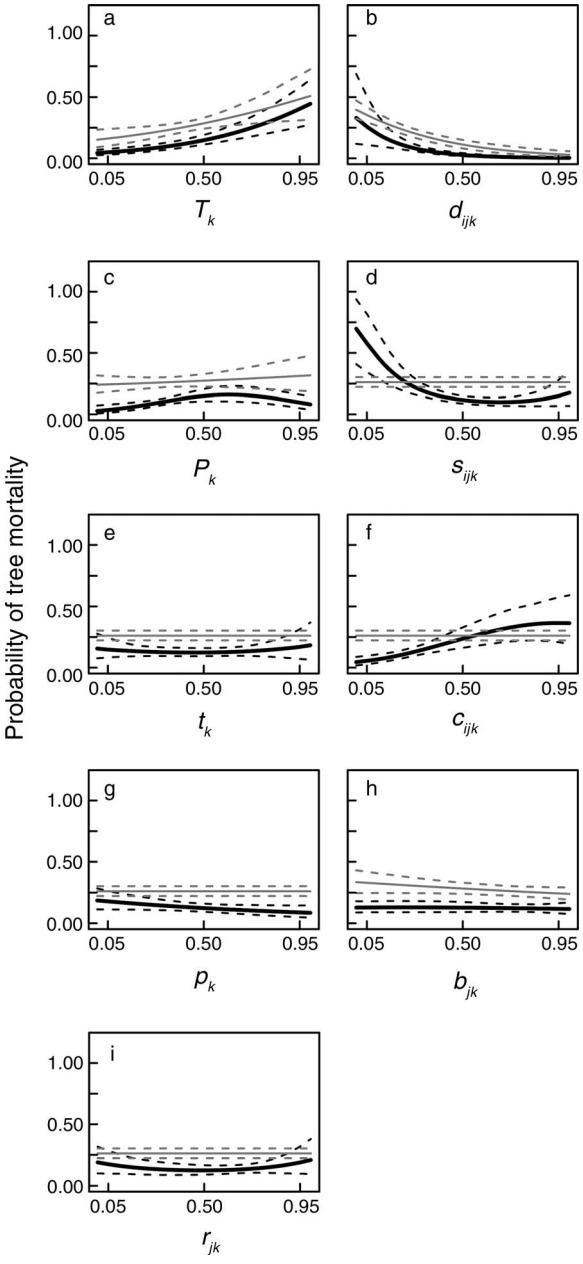

FIG. 3. Mean (solid line) and 95% credible intervals (dashed lines) for 10-year mortality rates for healthy (black) and diseased trees (gray) in response to variation in covariates. For each panel, all variables except for the variable of interest were held constant at the mean observed values. See Fig. 2 for explanations of variables.

2009, Worrall et al. 2013). However, in regions with higher summer precipitation, representing much of the aspen forest in the southwestern United States, disease effects on mortality decreased with temperature (Figs. 3a and 5c), potentially indicating that disease acts to expose populations to mortality in regions not generally associated with moisture limitation. In both cases, the largest increases in mortality associated with disease were in climates where healthy trees experience relatively low mortality risk.

As opposed to the cross-site patterns related to geographic variation in climate (Figs. 3a and 5), disease effects on 10-year mortality rates with respect to tree

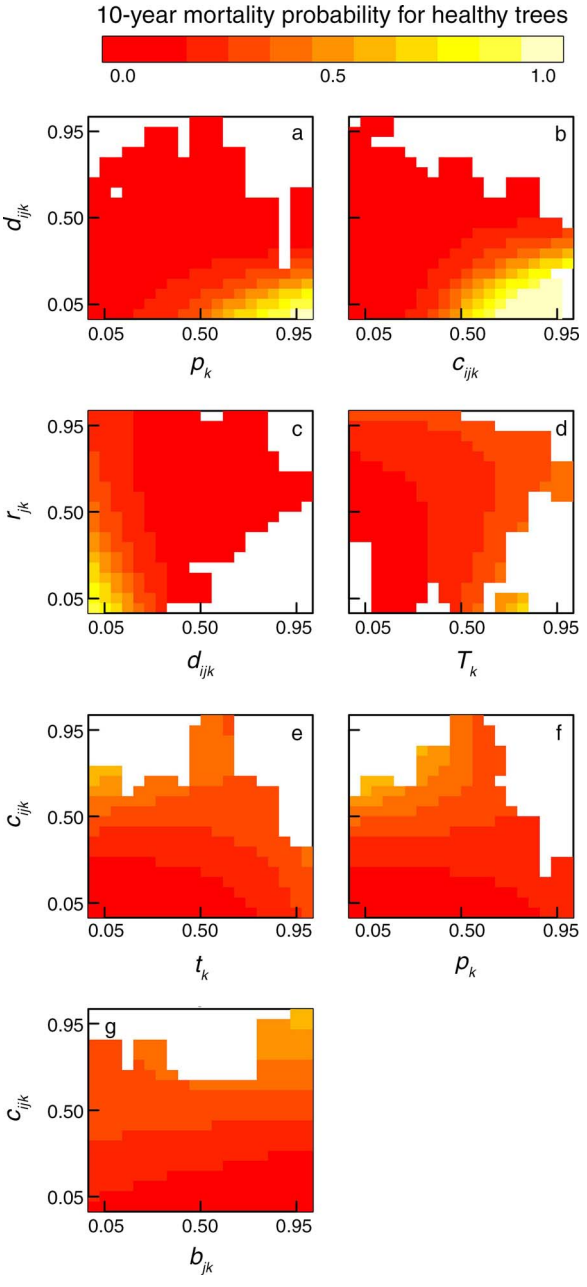

FIG. 4. Predicted 10-year mortality probabilities for healthy trees highlight interacting effects of individual condition (i.e., diameter and slenderness), stand characteristics (i.e., competition and topography), and precipitation. Yellow indicates high probability and red indicates low probability. White regions represent covariate space for which there were no plots. For each panel, all variables except for the two variables of interest were held constant at the observed means. See Fig. 2 for explanations of variables.

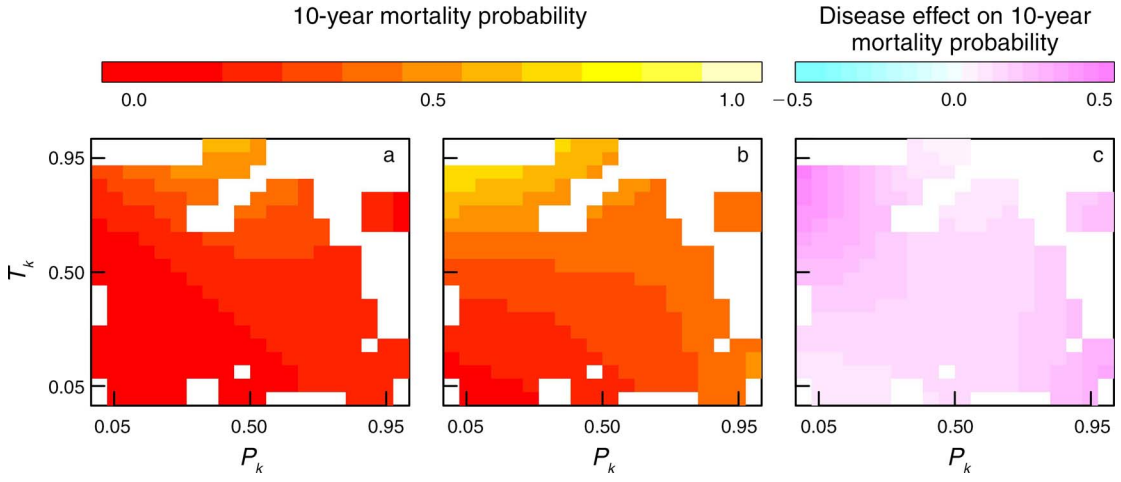

FIG. 5. Predicted 10-year mortality probabilities for (a) healthy trees, (b) diseased trees, and (c) the effect of disease on 10-year mortality probabilities (from −0.5 to 0.5) across geographic variation in summer temperature and precipitation.

diameter (Fig. 3b) seem to support the idea that disease amplifies risk experienced by individuals already at some degree of risk (i.e., moderate diameter trees). Thus, our results highlight an important scale dependence in the apparent influences of disease on mortality; individual-scale responses suggest disease contributes to self-thinning of young, dense stands while regional-scale climate responses indicate that disease may contribute to population declines in both moisture limited regions (i.e., high temperature and low precipitation) and cool, moist areas not clearly experiencing stressors, like drought. Furthermore, at the intermediate, subplot-scale, there was a slightly increasing effect of disease on aspen mortality as aspen relative basal area declines (Fig. 3h), presumably resulting from conifer encroachment. This provides support for predictions of acceleration in aspen forest succession (Bell et al. 2014).

Ecologists are increasingly interested in trait variation and its influence on species responses to environmental change as might be represented by interaction groups we examined in this study (Table 1). Our results support the hypotheses that slenderness, diameter, and competition alter vulnerability to disease and mortality and that climatic effects are complex and mediated by local topography (Table 2). Fewer covariates and interactions were present in the model for mortality in diseased trees, potentially indicating that disease homogenizes the mortality process, with tree mortality becoming only a matter of time. For example, while healthy trees experiencing low competition exhibited low mortality risk, diseased trees experienced constant, relatively high mortality regardless of competition (Fig. 3f). Alternatively, the smaller sample size for diseased trees could contribute to the identification of fewer variables, but with over 1000 diseased trees in our data set, we find this unlikely to explain all of the observed differences in model complexity. Our results support our focus on both plot-level climatic stressors and individual traits in mediating mortality responses.

Compared to mortality, characterizing patterns of disease prevalence required greater model complexity though few of the interactions differed from zero, highlighting the large uncertainties in the disease process. The large number of interactions in the disease prevalence model (Table 2) made interpretation of response surfaces difficult and contributed to greater uncertainties in predictions (Fig. 2). Furthermore, 95% credible intervals for interaction effects often included zero (Appendix B: Table B1), indicating weak and/or uncertain impacts. Despite these uncertainties, disease prevalence in small diameter, low slenderness trees appeared to be particularly sensitive to geographic variation in summer precipitation (Appendix B: Fig. B1c and d). Given that aspen slenderness was negatively correlated with diameter (Pearson correlation = −0.43), this result indicates elevated disease prevalence in both young and old trees, especially in drier climates.

Uncertainties in any study of broadly distributed forest inventory data arise from a variety of sources, constraining inference on specific responses or processes. Some uncertainties we explicitly accounted for, such as differences among plots that might increase or decrease disease prevalence or mortality rates through the use of random effects (Appendix B: Fig. B2). Other uncertainties could not easily be incorporated into any similar analysis. For example, visual inspection of disease is likely to only identify infection after the pathogen has become well established and developed fruiting bodies, meaning that our data very likely lag behind the actual process. Visual inspection represents a compromise between no data on disease and impractical, if not impossible, microbial analyses on every individual in each stand. However, individual

aspen exhibiting the advanced stage of infection represented by visual assessment are more likely to experience mortality in the short-term (Fig. 3), making relationships between disease and mortality easier to identify.

The impact of disease at the individual- and plot-scales imply differing roles of disease in size-mediated vs. climate-mediated aspen mortality. Disease may reinforce stand thinning as competition increases in young stands dominated by small to moderate size trees. Conversely, disease may both accelerate declines in the warm, dry regions and expose populations in cooler, moister climates to elevated risk. Sudden aspen decline has the potential to dramatically alter biodiversity (Kuhn et al. 2011) and carbon storage (Huang and Anderegg 2011) in the southwestern United States. While several factors are contributing to aspen mortality, including forest succession, climate change, and disease (Worrall et al. 2010, Marchetti et al. 2011, Zegler et al. 2012), uncertainty in their relative contributions and the importance of interactions persist. Furthermore, explicitly modeling interacting ecological processes, such as disease and mortality, may inform our understanding about the potential future of aspen and provide broader insight.

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Supplemental Material

Ecological Archives

Appendices A–C are available online: http://dx.doi.org/10.1890/14-1184.1.sm