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Inferring controls on the epidemiology of beech bark disease from spatial patterning of disease organisms

Jeffrey R. Garnas*[1], David R. Houston[†], Mark J. Twery[‡], Matthew P. Ayres* and Celia Evans[§]

**Department of Biological Sciences, Dartmouth College, Hanover, NH 03755, [†]US Forest Service, Danville, VT 05828, [‡]US Forest Service Northern Research Station, South Burlington, VT 05403, and [§]Department of Science, Paul Smith's College, Paul Smith, NY 12970, U.S.A.*

Abstract

1 Spatial pattern in the distribution and abundance of organisms is an emergent property of collective rates of reproduction, survival and movement of individuals in a heterogeneous environment.

2 The form, intensity and scale of spatial patterning can be used to test hypotheses regarding the relative importance of candidate processes to population dynamics.

3 Using 84 plots across eastern North America, we studied populations of two associated plant parasites, the invasive felted beech scale *Cryptococcus fagisuga* Lind. and the native *Neonectria* fungi, which together cause beech bark disease (BBD).

4 We evaluated spatial patterns at the scales of trees within stands, stands within the forest and forests within the landscape to examine four hypothetically important factors in the ecology of the disease: (i) local contagion within stands; (ii) regional contagion, or among patch infection–reinfection dynamics; (iii) variation in host susceptibility linked to genetic and/or environmental heterogeneity; and (iv) climate effects on population growth of BBD organisms.

5 Analyses revealed an unexpected lack of spatial aggregation in BBD populations among trees, stands and forests. This implies that propagule pressure is generally sufficiently high throughout the infested region of North America such that neither trees nor stands are spared from the disease by dispersal limitations of the disease agents. Furthermore, variation in tree and stand level susceptibility has minimal impact on BBD dynamics and climate is not a conspicuous driver of abundance within the core range of BBD.

Keywords Beech bark disease, *Cryptococcus fagisuga*, forest pestilence, *Neonectria ditissima*, *Neonectria faginata*, spatial epidemiology.

Introduction

Spatial pattern in the distribution and abundance of organisms is an emergent property of collective rates of births, deaths and movement of individuals in a heterogeneous world. At the beginning of a biological invasion, the invading population is typically aggregated around the port of entry (Liu *et al.*, 2007; Liebhold & Tobin, 2008). Once an area is fully colonized, however, the full spectrum of dispersion patterns becomes possible (aggregated to random to overdispersed), depending on dispersal and spatial patterns in survival and reproduction. Habitat patchiness, movement behaviour and intra- and interspecific interactions are all frequently noted as drivers of spatial patterns in population density (Ryti & Case, 1986; Burdon *et al.*, 1989; Koenig, 1999; Krivan *et al.*, 2008). Understanding the degree and spatial scale at which populations co-vary permits inference about the underlying mechanisms likely to have produced the observed patterns (Bjørnstad *et al.*, 1999; Silvertown *et al.*, 2001; Peltonen *et al.*, 2002; Liebhold *et al.*, 2004). Growing recognition of the power of such analyses, along with improved data availability and analytical tools, have motivated studies of spatial pattern at the scale of landscapes or even continents (Kendall *et al.*, 1998; Post, 2005; Liebhold & Tobin, 2008). Such an approach may

Correspondence: Jeff Garnas. Tel.: +27 12 420-3854; fax: +27 12 420 3960; e-mail: jeff.garnas@fabi.up.ac.za

[1]Present address: Zoology and Entomology, Forestry and Agricultural Biotechnology Institute, University of Pretoria, Pretoria 0002, South Africa.

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be particularly relevant for forest pestilence because outbreaks often occur at regional scales, and evidence is accumulating that large-scale fluctuations in abundance are predominantly linked to spatially correlated exogenous variation (Peltonen *et al*., 2002; Liu *et al*., 2007; Aukema *et al*., 2008; Liebhold & Tobin, 2008). Consideration of multiple spatial scales can improve the mechanistic understanding of important system processes because patterns evident at one scale are not always important or detectable at another and may even be reversed (Tilman & Kareiva, 1997; Ylioja *et al*., 2005).

Beech bark disease in North America

Beech bark disease (BBD) in North America is a cankering disease of American beech *Fagus grandifolia* Ehrl. arising from the interaction of two primary causal organisms: an eriococcid scale insect *Cryptococcus fagisuga* Lind. and any of two or possibly three ascomycete fungi of the genus *Neonectria* (*Neonectria faginata* or *Neonectria ditissima)* and possibly *Bionectria ochrolueca* (Ehrlich, 1934; Houston, 2005; Houston *et al*., 2005; Castlebury *et al*., 2006). *Neonectria faginata*, however, is highly specific to the BBD system and appears to predominate in the aftermath zone of the disease, where the present study was conducted (Houston, 1994a; but see also Kasson & Livingston, 2009). Both insects and fungi are required for disease development. Insect feeding facilitates the colonization of phloem tissue by *Neonectria* (Ehrlich, 1934), although the exact mechanisms remain elusive (Cale *et al*., 2012). In addition, the cankering response of beech in response to fungal infection creates bark microstructure that may favour scale insect survival in harsh conditions, raising the possibility of positive feedbacks among the causal organisms (Houston, 2005). High-density fungal infections can girdle and kill trees, and individuals that survive infection are more susceptible to biotic and abiotic stress (Lovett *et al*., 2006). Insects and fungi are independently transported among trees by wind (Ehrlich, 1934).

BBD was putatively introduced from Europe into Nova Scotia, Canada at the end of the 19th Century with the arrival of scale insects on imported plant material (Ehrlich, 1934). It now appears that associated *Neonectria* species (including *N. faginata*, formerly *Neonectria coccinea* var. *faginata*) are native to North America (Castlebury *et al*., 2006). *Neonectria ditissima* (formerly *Neonectria galligena*) is associated with target canker on a variety of deciduous hosts. BBD has had strong impacts on the deciduous forests in northeastern North America by dramatically reducing survivorship but increasing recruitment, such that affected forests have a reduced abundance of large beech but increased densities of smaller beech trees (Houston, 1994b; Ellison *et al*., 2005; Garnas *et al*., 2011a).

Biological mechanisms promoting spatial autocorrelation at multiple scales

In the present study, we evaluated spatial patterns in the abundance of BBD organisms aiming to test hypotheses regarding the roles of contagion, variation in host susceptibility and exogenous demographic effects. Specifically, we hypothesized that BBD dynamics are influenced to varying degrees by (i) local contagion driven by the dispersal of insects and fungal spores from infected to adjacent, uninfected trees; (ii) regional contagion, where stand BBD severity is linked to the frequency and intensity of colonization from nearby infected stands; (iii) variation in host susceptibility linked to genetic and/or environmental heterogeneity; and (iv) climate effects on the population growth of BBD organisms. All of our population estimates come from the core range of BBD (with the exception of four sites in Michigan, which are considered separately) where scale insects and fungi have been long established and are effectively endemic, and so we did not consider invasive spread as a theoretical candidate for producing spatial patterns in our data, although we acknowledge the importance of the initial invasion process as a driver of spatial pattern at a broader geographical scale. (Morin *et al*., 2007). Because some of the hypothetically important drivers of population dynamics differ with respect to the scale at which patterning would be expected and because identifying the appropriate scale *a priori* is not always possible, we studied the system at three different spatial scales: (i) trees within stands; (ii) stands within forests; and (iii) forests within the subcontinental landscape.

Local scale (trees within forest stands)

The processes of local contagion (driven by dispersal among nearby trees at a scale of a few metres; H1) and of spatially structured variation in host tree susceptibility (arising from genetic or environmental patchiness influencing the incidence and severity of BBD on individual beech trees; H3) both predict spatial aggregation at the scale of trees within stands. Approximately 1% of beech trees are resistant to scale insect attack (Houston & Houston, 2000). Resistance is correlated with low total nitrogen and amino acid content in the bark and is under some genetic control, although the contribution of the local environmental variability is unknown (Wargo, 1988; Houston & Houston, 2000). Beech genotypes tend to be spatially clustered within stands because trees reproduce both vegetatively from root suckers and via seed, many of which are planted in sibling groups as a result of the caching behaviour of jays (Johnson & Adkisson, 1985; Jones & Raynal, 1986; Kitamura & Kawano, 2001). There also exists the potential for fine-scale spatial variation in soil type, nutrient, water or light availability that could influence BBD susceptibility.

Mesoscale (stands within forests)

The hypotheses of regional contagion (H2) and coarse-scale variation in disease susceptibility (H3) both predict spatial covariance at the scale of stands within forests. For the former, if patch or stand-level extinction of one or both BBD organisms were common, re-infection from neighbouring sources would likely be clustered around sites producing migrants that disperse tens to hundreds of metres. Under the latter mechanism, BBD susceptibility would be linked to stand or site-level conditions that themselves co-vary in space (e.g. beech density, stand age, species composition, slope, elevation and soil type).

Landscape scale (forests within the landscape)

Spatial autocorrelation at the scale of forests within the sub-continental landscape would support a role for spatially correlated climatic (Moran) effects (H4). At this scale, knowledge of landscape structure, decay distance in the autocorrelation function (e.g. the minimum distance between sites at which they are no longer correlated) and directionality in covariance can suggest which mechanisms dominate the spatial epidemiology. For example, dynamics that are synchronous at spatial scales beyond those likely to be linked by dispersal would implicate climate or some other broad environmental forcing, as would correlated fluctuations that span geographical barriers, or where directional asymmetry (anisotropy) exists (Peltonen *et al*., 2002; Stenseth *et al*., 2002; Forchhammer & Post, 2004; Post, 2005; Halkka *et al*., 2006). Covariance that decays rapidly with distance or that is characterized by travelling waves indicates the importance of dispersal (Grenfell *et al*., 2001; Bjørnstad *et al*., 2002). Finally, temporal aspects of spatial covariance can also lend additional explanatory power; patterns in spatial covariance that are relatively static in time suggest regional variation in habitat quality or important community interactions (Hanski *et al*., 1991; Bjørnstad *et al*., 1995), whereas temporally variable patterns may point to stochastic influences (Liebhold *et al*., 2004). For populations with cyclical dynamics, even moderate dispersal can be surprisingly powerful in creating large scale synchrony (Bjørnstad *et al*., 1999), although it appeared unlikely *a priori* that this would be important for BBD because there is no signal of cyclical dynamics, based on time series data (Garnas *et al*., 2012).

Materials and methods

General approach

We assessed spatial aggregation in disease agent populations at three spatial scales using a hierarchical design. In all cases, we estimated current disease agent populations (independently for scale insects and *Neonectria*) on many individual beech trees at two zones (0–2 m and 2–4 m high) using a 0–5 and a 0–4 point scale, respectively (Houston *et al*., 2005; Garnas *et al*., 2011b; see Supporting information, Table S1). We averaged population estimates across height zones to obtain a single measure per tree. Sampling was restricted to trees > 10 cm diameter at breast height (DBH) because smaller trees are only rarely colonized by scale (Houston, 1994b). Estimates for scale insects were based on densities of the waxy secretions produced by feeding adults. For *Neonectria*, visual estimates of abundance class were based on the density of current year fruiting structures (perithecia or conidia). Although this method may miss some recent infections that are not yet fruiting, reproductive structures are generally produced annually on infected trees and their density on the bottom 4 m of the bole should be well correlated with fungal abundance. Our estimates comprise a composite measure of abundance for *N. faginata* and *N. ditissima* because they are morphologically indistinguishable in the field and have been known to co-occur within sites and even on the same tree (Kasson & Livingston, 2009). We assume that our measurements primarily reflect the abundance of *N. faginata* because the abundance of *N. ditissima* in the aftermath zone of BBD appears to be generally low (Houston, 1994a). We also measured a suite of tree- and site-level attributes including DBH, crown class, tree health, crown thinning or chlorosis, several measures of BBD-related bole defect (e.g. raised and sunken cankers, etc.), slope, aspect, plot basal area and species composition. Latitude and longitude were recorded for all sites using a Garmin GPS (Garmin International Inc., Olathe, Kansas). To assess dominant trends in our data, and to identify possible covariates, we evaluated correlation matrices among trees and sites for all measured variables.

Plot selection and measurement

Local scale. We studied spatial patterns among trees within stands with replicated 0.28-ha plots within which we tagged and mapped all stems of all tree species and estimated the abundance of disease agent populations on each individual beech tree. The plots were shaped as seven overlapping circles (radius of 15 m, reflecting the use of an ultrasound distance-measuring tool (DME 360; Haglöf AB, Sweden); see Supporting information, Figs S1 and S2). In 2005, we spatially mapped all trees in seven sites in Maine, Massachusetts, New York and West Virginia (1, 1, 3 and 2 sites, respectively). Sites were originally selected by one of us (D.R.H.) in 1979 to capture both the advancing front and the aftermath zone of BBD (Garnas *et al*., 2012).

We used a Monte Carlo resampling approach to test for patterns of contagion among trees within stands (Milgroom & Lipari, 1995). Most trees had at least a few scale insects, although they ranged from barely detectable to highly abundant. We classified the status of each tree for scale infestation as low or high (scale index ≤ 1 versus > 1) such that approximately half of the trees fell into each category (the same tests with alternative thresholds for low versus high gave very similar results). For *Neonectria*, which was frequently absent on trees, we classified trees based on presence versus absence. For each study stand, we calculated the nearest infected neighbour distance (NIND) for each infested/infected tree with respect to both scale insects and fungi. Trees within 6 m of the plot boundary were excluded as focal trees to preclude edge effects. The mean NIND for the stand was then compared with the frequency distribution of possible NINDs assuming random dispersion. Accordingly, we randomly assigned disease agent status to each tree within the stand, keeping the total infection frequency and tree locations constant, and then recalculated the mean NIND. We repeated this with replacement 5000 times and compared the resulting frequency distributions with the empirical value for each site. Nonrandom dispersion was indicated by extreme values for the empirical NIND compared with the frequency distribution of possibilities under the null hypothesis of random dispersion.

Mesoscale (stands within forests)

In 2007, we randomly selected 22 0.1-ha permanent plots in Bartlett Experimental Forest (Bartlett, New Hampshire) from a subset of 41 plots studied by Twery and Patterson (1984).

Within each plot, we estimated as before the abundance of disease agents on each beech tree > 10 cm DBH. Median distance between plots was 1.6 km (range 68 m to 2.58 km). In 2008, we replicated this sampling design in Hubbard Brook Experimental Forest (near Lincoln, New Hampshire). Coordinates for 25 plots were randomly generated in advance of the study and locations were stratified to approximate the range of distances between plots sampled at Bartlett Forest (median distance 1.77 km; range 71 m to 5.0 km). To increase sampling efficiency, and because there were no pre-existing plots, we allowed plot size to vary at Hubbard Brook depending on the density of beech; we sampled stands outward from the plot centre to at least 30 m or until we had measured at least 15 beech > 10 cm DBH. Mean plot size was approximately the same as in the Bartlett study, although the number of beech trees per plot was less variable. This change should not have introduced any bias into estimates of plot-specific abundances for BBD disease agents, and allowed us to sample more plots.

We tested for spatial autocorrelation using a nonparametric spatial covariance function (Hall *et al*., 1994; Bjørnstad & Falck, 2001) using untransformed site-level means for abundance. We also tested for spatial covariance between scale insects and fungi wherever possible (i.e. where *Neonectria* was found; scale insects were present in all plots). Because the abundance of BBD disease agent can vary with tree diameter (Griffin *et al*., 2003; Houston, 2005), we also tested for spatial autocorrelation using population estimates detrended for tree size. We accommodated the overabundance of zeros (especially for tree-level estimates of *Neonectria*) using a contingency (or hurdle) model that partitions data into zero and nonzero values and estimates the slope of the presence–absence versus nonzero relationships with a continuous predictor (in this case, tree diameter) separately (Martin *et al*., 2005). In no instance did the use of size-detrended population estimates influence qualitative outcomes, and so the results from this approach are not reported. Analyses were performed in R, version 2.6.1, including package fields and ncf (Nychka, 2007; Bjørnstad, 2008; R Development Core Team, 2008).

Landscape scale (forests within the landscape)

In 2006–2007, we sampled 20 additional study plots from Maine to West Virginia originally established by D.R.H. in approximately 1979. We also added four sites in Mason County, Michigan (where BBD had recently established; O'Brien *et al*., 2001), two in central Vermont, two in Pennsylvania (Lebanon and McKean Counties) and two in Maine (Penobscot and Aroostook Counties). Site means from our mapped-tree sites (2005), as well as Bartlett and Hubbard Brook Forests, were also included in landscape level analyses, yielding a total sample size of 39 plots for the landscape analyses. Our sampling design in 2006–2007 consisted of measuring all beech and sugar maple within two 100 × 5-m random transects and two variable radius plots (minimum count of 15–20 beech). Protocols for sampling disease and tree health have been described in detail previously (Houston *et al*., 2005; Garnas *et al*., 2011b).

We used the same techniques as for plots within stands to test for spatial autocorrelations and cross-correlation across forests. Analyses of cross-correlations excluded 12 stands where there were no detectable fungal infections. We ran all landscape-level analyses both with and without Michigan sites because Michigan was an outlier spatially, was situated along the advancing front of the disease, did not contain fungi, and had comparatively high scale insect densities; qualitative results were unchanged. Analyses for landscape patterns required using data collected across three seasons, although this should not have had much effect on the patterns because there is only modest interannual variation in these forests in the abundance of scale insects and *Neonectria* (Garnas *et al*., 2012). To provide visualization of broad geographical patterning in scale insect and *Neonectria* densities, we constructed surface plots using thin plate spline regression (Nychka, 2007; R Development Core Team, 2008).

Results

Forest characteristics and patterns in BBD incidence and severity

Beech was common in all stands but varied in density, accounting for 13–85% of total live basal area among the 84 plots (mean ± SD = 38 ± 19%; Table 1). Unexpectedly, both the percent beech and total beech basal area were uncorrelated with disease agent indices. The proportion of standing beech that were dead at the time of sampling was also variable, in the range 0–42% (median 11%). There was no obvious relationship between apparent mortality and disease severity or latitude, although the highest mortality was in the Adirondacks, New York, where there was severe damage from an ice storm in 1998. The size distribution of beech stems > 10 cm also varied; the mean DBH (± SD) for beech was 19 ± 6 cm and decreased linearly with latitude. Mean tree size also declined strongly with duration of infection with BBD ($F_{1,37} = 9.05$, $P = 0.002$, $r^2 = 0.20$) in a manner consistent with patterns of disease-induced changes in forest structure described previously (Garnas *et al*., 2011b). Mean density of scale insects and *Neonectria* were relatively low in most sites (mean ± SD = 1.46 ± 0.60 and 0.31 ± 0.30,

Table 1 Summary data for 84 sites sampled for beech bark disease during 2005–2007

Site	Mean ± SD
Basal area (BA[a]) total	27.6 ± 8.8
Beech BA	9.9 ± 5.5
% beech BA	37.7 ± 19.1%
Sugar maple BA	5.1 ± 5.0
Other species BA	14.3 ± 11.3
Mean wax index	1.46 ± 0.6
% Standing dead (BA)	13 ± 11%
Mean *Neonectria* index	0.31 ± 0.30
Mean diameter at breast height for beech (cm)	18.7 ± 5.9
Count beech	44.4 ± 36.3
Slope (°)	12.4 ± 11.3
Aspect	All
Elevation (m)	459 ± 202

[a]Basal area = m^2/ha.

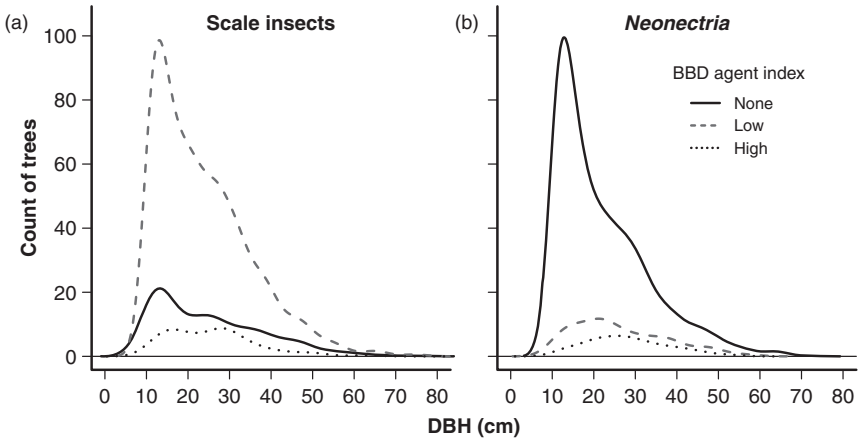

Figure 1 Frequency histograms showing tree size distributions by abundance index for scale insects (a) and *Neonectria* (b). 'None', 'Low' and 'High' correspond to '0', '> 0 to 2.5' and '> 2.5 to 5' for scale insects and '0', '> 0 to 2' and '> 2 to 4' for *Neonectria*; BBD, beech bark disease; DBH, diameter at breast height.

respectively), although most trees showed some evidence of current or prior infection. All sites contained some scale insects; however, seven sites in southern Pennsylvania plus all five sites in Michigan had no visible fungal fruiting bodies, nor obvious signs of past *Neonectria* infection, suggesting the fungus has not yet arrived in these stands. There was no relationship between mean beech DBH and site means for scale insects (Pearson's $r = -0.26$, $P = 0.13$) or *Neonectria* (Fig. 1) ($r = 0.05$, $P = 0.79$). Uninfected trees were slightly larger on average than trees harbouring scale (Fig. 1a). For *Neonectria*, the mean DBH increased slightly with infection class (Fig. 1b).

Spatial autocorrelation in site attributes

Several site attributes (Table 1) showed evidence of spatial aggregation at one or more spatial scales studied. For example, percent basal area for sugar maple was spatially correlated ($r = 0.66$ at the nearest distances, decaying to zero at approximately 275 km). Within Bartlett Forest, percent basal area for species other than beech or sugar maple was also autocorrelated ($r = 0.82$, to approximately 800 m). There were also some inter-correlations among variables. Elevation was correlated with sugar maple basal area ($r = 0.26$, $P = 0.02$) and with latitude ($r = -0.51$, $P = 0.002$), and plot slope was correlated with beech ($r = -0.24$; $P = 0.03$) and sugar maple basal area ($r = 0.27$; $P = 0.01$). Plot elevation was also strongly spatially autocorrelated (within Bartlett and Hubbard Brook forests, as well as at the landscape scale). There was little additional correlational structure in the data. Interestingly, mean scale insect and fungal densities were uncorrelated at the level of forest stands (Spearman's $r = 0.11$, d.f. $= 82$, $P = 0.31$). On individual trees, there was a weak but significantly positive correlation between insect and *Neonectria* densities ($r = 0.15$, d.f. $= 2944$, $P < 0.0001$).

Spatial dispersion in BBD agent populations

We found almost no signal of aggregation in either scale insect or fungal populations at any spatial scale. At the scale of trees within forest stands, only one of the seven plots showed spatial patterning with respect to BBD agent populations (MA 440; Table 2). In one additional stand (ME 102), there was a marginal signal of aggregation in fungal densities only ($P = 0.056$). There was no evidence for spatial autocorrelation in BBD agent populations at the scale of forest stands in either Bartlett or Hubbard Brook (Fig. 2 and Table 2) Experimental Forests (Fig. 2a, b, d, e) or within the subcontinental landscape (Fig. 2c, f).

Cross-correlation functions between scale insects and *Neonectria* showed no pattern with distance except for a modest negative association at the nearest distances at Hubbard Brook (Fig. 2g–i). At the scale of the northeastern U.S.A., surface contour plots suggested an inverse pattern in population densities for scale insects versus fungi (see Supporting information, Fig. S3). Scale insect densities were highest in

Table 2 Nearest infected neighbour distances (NIND) for scale insects and *Neonectria* on trees within seven stands in Maine, New York, Massachusetts and West Virginia

	Site	Empirical mean NIND (m)	*P*
Scale insects	NY 612	35.8	0.91
	NY 613A	10.9	0.15
	NY 613B	16.5	0.19
	MA 440	2.7	0.002**
	ME 102	2.4	0.12
	WV 820	7.3	0.14
	WV 821	5.5	0.68
Neonectria	NY 612	11.5	0.34
	NY 613B	12.6	0.58
	MA 440	2.85	0.012**
	ME 102	2.2	0.056[a]
	WV 820	8.2	0.30
	WV 821	6.1	0.20

[a]$0.10 < P < 0.05$; *$0.01 < P < 0.05$; **$0.001 < P < 0.01$.
P-values represent tests for spatial aggregation versus the null hypothesis of random dispersion.

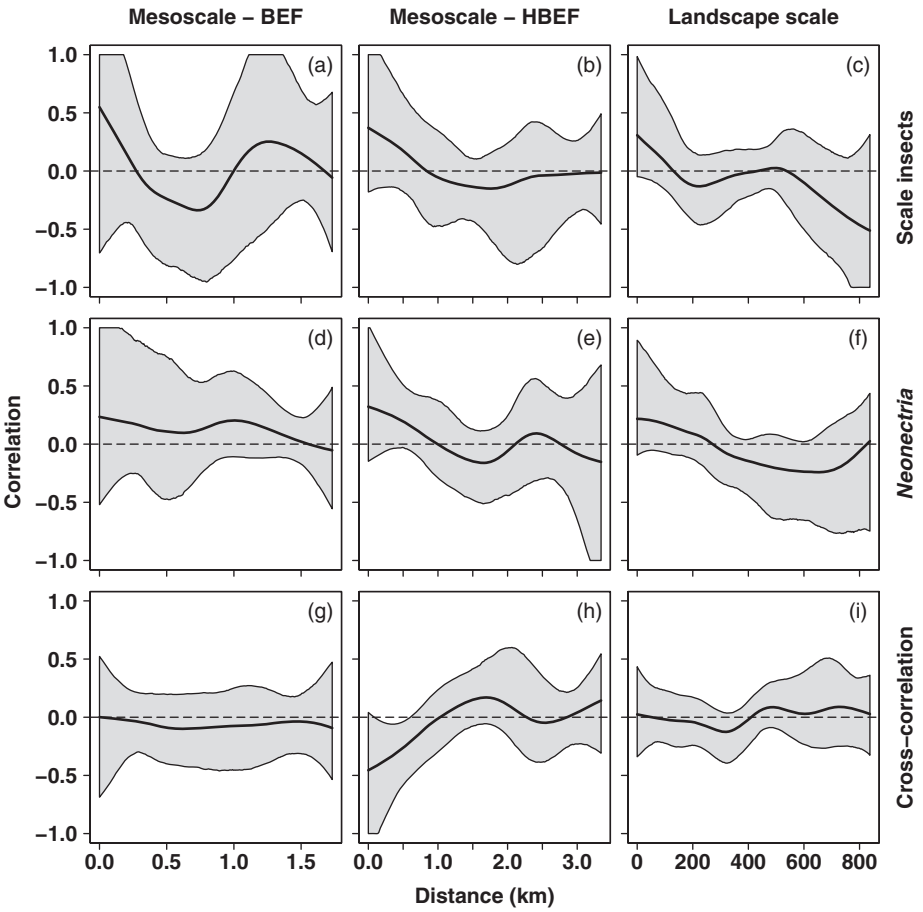

Figure 2 Spline correlograms depicting correlations (± 95% confidence intervals) as a function of distance for scale insect (a–c), *Neonectria* (d–f) and their cross-correlations (g–i). The left and centre columns correspond to the intermediate spatial scale under consideration for Bartlett Experimental Forests (BEF) and Hubbard Brook Experimental Forest (HBEF), respectively.

West Virginia and northern New England, whereas *Neonectria* densities were highest in southern New England (approximately Connecticut and Massachusetts), the approximate centre of the current range of BBD.

Discussion

Spatial dispersion in populations of scale insects and fungi associated with BBD was surprisingly unstructured at all three spatial scales that we considered (local, mesoscale and landscape). Spatial autocorrelations in both scale insect and fungal populations were only moderately positive even at near-zero distances and decayed quickly. These patterns argue against several otherwise plausible hypotheses for key factors in BBD dynamics, and have implications for the management of BBD in long-infected forests.

Inference concerning the role of dispersal in the BBD system

Diseases where colonization from highly infective individuals strongly drives the frequency or severity of infection would be expected to produce a pattern of moderate to strong spatial clustering around individual hosts harbouring high disease agent populations (Peltonen *et al*., 2002; Gilligan & van den Bosch, 2008). The lack of a strong pattern of aggregation in BBD populations at any of the scales considered demonstrates that dispersal by insects and fungi does not strongly drive BBD dynamics within the currently colonized range. Within stands, contagion from point sources (e.g. large or highly infected individuals) had no detectable effect on the occurrence of BBD within the sampled stands. Given the high frequency of infected trees across all sampled stands, it is likely that propagules (first instar insect 'crawlers' and fungal spores) occur at sufficiently high densities to effectively saturate habitats with broadly overlapping dispersal curves. This is consistent with work for other similar insects and for fungi. Mites and wingless insects regularly disperse via wind currents to distances up to 100 m or more (Washburn & Washburn, 1984), although studies in a closed forest canopy are rare. A study of felted beech scale in England captured a small but measurable proportion (0.7%) of scale insect crawlers above the canopy (18 m above the forest floor), suggesting that long distance dispersal is possible (Wainhouse, 1980). Both fungi and insects are capable of long distance, even trans-oceanic, dispersal on

wind currents, although the relative importance of such events on population dynamics is largely unknown (Isard & Gage, 2001). In addition, although BBD infection is not systemic, populations of insects and/or fungi may be sufficiently long-lived on trees so as to minimize the importance of local dispersal or metapopulation processes. Particularly for scale insects, tree level extinction appears to be a rare event (Garnas *et al*., 2012), and overwintering colonies provide a local source of new individuals that probably swamps the demographic impact of immigration from other trees. Given the comparative rarity of active *Neonectria* fruiting on trees in most stands, dynamics driven by long-term persistence on trees appears less likely for fungal populations, although the longevity of local infections within phloem tissue (before sporulation) is not well known. For *N. ditissima*, inoculum can be present on non-beech hosts within a stand independent of BBD, although the importance of transmission among host species is not understood (Houston, 1994b).

Inference at the mesoscale

The lack of strong spatial structure at the scale of trees and stands within forests also suggests a limited role for spatially variable susceptibility linked to tree genotypes or to local site conditions. Beech genotypes are clustered in space as a result of clonal root suckering and seed caching by jays (Jones & Raynal, 1986). That patterns of BBD infection or severity do not reflect local patches of resistance suggests that such resistance may be rare and of minor importance to the current epidemiology of the disease. This supports earlier findings that only approximately 1% of trees were resistant to experimental challenge by scale insects (Houston & Houston, 2000) and further suggests that variability in quantitative resistance or susceptibility are unlikely to show strong genetic underpinnings. The role of environmental variation that may drive patterns of susceptibility (e.g. nutrient or water availability, sunlight, or factors contributing to tree stress; Manion, 1981) is perhaps more difficult to assess, although clear relationships with spatially co-varying factors were not in evidence. We do not reject the existence of environmental correlates with disease. For example, Griffin *et al*. (2003) found the strongest effects of disease in mid-elevation sites in the Adirondack region in New York, whereas, in North Carolina, BBD is primarily confined to mountaintops despite abundant host material at lower elevations (Morris *et al*., 2002). However, our results indicate that spatially structured variation in host susceptibility is not a conspicuous driver of spatial patterns in BBD.

Inference from patterns at the landscape scale

At the broadest scale of our analyses, we also found no significant spatial autocorrelations in the abundance of either scale insects or fungi. Because of logistical constraints, this was the least robust of our analyses and we do not reject the existence of spatial pattern at the landscape scale. Nonetheless, it was based on reasonably precise population estimates for 84 plots distributed across > 500 km, and so the lack of readily detectible spatial autocorrelation argues that the abundance of BBD agents within the core range of the disease is not strongly influenced by broad drivers such as climate, and this conclusion is consistent with other findings (Garnas *et al*., 2012). This contrasts with some other studies of forest insects and mammals that have demonstrated positive spatial autocorrelation and synchronous population fluctuations at the scale of tens to hundreds of kilometres (Koenig, 1999; Williams & Liebhold, 2000; Peltonen *et al*., 2002; Stenseth *et al*., 2002; Liebhold *et al*., 2004; Johnson *et al*., 2005; Post, 2005). Most of these studies have involved species that display conspicuous population fluctuations, which is unlike BBD within the core of its established distribution in North America (Houston *et al*., 2005; Garnas *et al*., 2012).

Complex interacting drivers and the detection of spatial pattern

Spatial pattern is an emergent property of numerous biological and ecological processes acting at varying strengths, directions and scales. It is possible for forces that promote aggregation to be counteracted by forces that promote overdispersion, creating a false impression of random dispersion. The most likely general mechanism for this involves enemies of the focal organisms. For example, common mobile natural enemies that forage optimally tend to disproportionately impact high density populations, which has the effect of homogenizing abundance in the landscape (Schneider, 1992; Dolman & Sutherland, 1997). The most notable predator of *C. fagisuga* is the twice-stabbed coccinelid ladybeetle (*Chilochorus stigma* Say), which can be common in high density populations of scale insects but is never observed to exert measurable control on their prey population (Baylac, 1980; Houston, 2005). Another possibility is an entomophagous fungus (*Verticillium lecanii* Viegas) that attacks high density populations of scale insects on European beech (Lonsdale & Wainhouse, 1983). V*erticillium lecanii* has been isolated from numerous insect hosts in North America, as well as having been recovered from soil samples in beech-dominated forests, although there have been no reports of a direct association with BBD, or of conspicuous demographic impacts (Hajek *et al*., 1997; Keller & Bidochka, 1998). The mycoparasite *Nematogonum ferrugineum* (*Gonatorrhodiella highlei*) can limit *Neonectria* growth rate in culture and suppress pathogenicity *in situ*, although it appears to be of similarly limited importance to disease dynamics in nature (Houston, 1983a). We cannot envision any likely scenarios by which apparently random dispersion in BBD would be the result of aggregation counteracted by thinning from enemies.

Implications for forest management

The present study has implications for forest management in the presence (and in anticipation) of BBD. First, our results strongly refute the notion that BBD is driven by infection-reinfection dynamics or that the effects of local contagion are important to understanding or controlling disease spread (Gilligan & van den Bosch, 2008). Therefore, the removal of highly infected individuals as a strategy to mitigate BBD is

unlikely to reduce infection severity in remaining trees. For beech trees (even across variable densities within different forest types), there appears to be little possibility of escaping in space from the agents of BBD in the long term. We found no relationship between beech density and BBD incidence and severity (Morin *et al*., 2007) and thus host density appears unlikely to be a strong driver of BBD dynamics. Thus, thinning or salvage cutting to reduce host densities is unlikely to be useful in curbing BBD (Perrin, 1983). This is not to say that salvage or sanitation cutting should not play a role in managing forests with BBD, particularly where damage from falling trees and branches is of concern or where economics dictate. However, selective removal of infected beech is unlikely to significantly influence the health of residual trees. Favouring or propagating resistant genotypes may have positive consequences, although the effects are likely to be very slow in developing. We note that our studies were carried out primarily in the aftermath zone of BBD, and it should not be assumed that circumstances are the same for stands or trees along the advancing front.

Based on our studies, it is probable that virtually every beech tree is exposed to BBD agents throughout the currently colonized region of North America. This is not unlike other invasive pathogens, such as chestnut blight and Dutch elm disease (amongst others), which have spared very few trees throughout North America (Gibbs & Wainhouse, 1986; Burdon *et al*., 2006). Within the aftermath zone (Shigo, 1964), stands devoid of scale insects or fungi are rare or non-existent. This implies that BBD has effectively saturated the habitat and may now be in approximate equilibrium with its host population.

Future directions

There are several questions that remain unanswered with respect to BBD dynamics, development and spread. For example, if it is true that both insects and fungi are effectively not dispersal limited, why has the geographical spread of BBD been relatively slow (approximately 15 km per year; Morin *et al*., 2007) such that only approximately 50% of the range of the host is currently colonized? Annual tree-level extinction is low for both scale insects and *Neonectria* where BBD is endemic (D. R. Houston, unpublished data). A lack of dispersal limitation within and among stands, coupled with a low extinction rate even at low densities, should result in rapid rate of invasive spread. One possibility is that extinction probability is elevated on a naïve resource, and that successful establishment requires some degree of 'priming' of the host tree. Beech trees beyond the range of BBD on the whole are visibly distinct in that their bark is smooth and almost devoid of microstructure, in contrast to the cankered and gnarled state of many trees that have survived repeated BBD infection (J. R. Garnas, personal observation; Houston, 1994b). The successful establishment and survival of scale insects is clearly enhanced by microstructure on outer bark, largely a result of infection by *Neonectria* (Shigo, 1964; Perrin, 1980; Houston, 1983b), which is itself dependent on scale insect populations (Ehrlich, 1934). This positive feedback between BBD agents may be important to population dynamics and to rates of geographical spread along the invasion front. Within the endemic range, however, it appears that the dynamics of these two organisms have become largely uncoupled. Scale insect and fungal densities are only weakly correlated at the scale of individual trees and entirely uncorrelated at the scale of stands. Direct influences of the densities of each disease agent on the population growth rates of the other appear to be trivial or non-existent, and may even be negative at the largest spatiotemporal scales (Garnas *et al*., 2011). If scale insect success within the advancing front depends on *Neonectria*, this could seriously slow the invasion, as the fungus typically lags scale by approximately 10 years (Houston, 2005). However, there now appears to be a reason to question the conventional wisdom that scale insects benefit from the presence of the fungus (Shigo, 1964; Houston *et al*., 1979; Dukes *et al*., 2009). Our Michigan sites had by far the highest scale insect densities observed in our multistate surveys and also had high mortality among large beech, whereas *Neonectria* was either absent or extremely rare (although the fungus has been found elsewhere in Michigan; Castlebury *et al*., 2006). Furthermore, the region of highest *Neonectria* abundance (western Massachusetts and southeastern New York) had relatively low abundances of scale insects (see Supporting information, Fig. S3). Further research will be required to determine whether these patterns are coincidental or correctly suggest that *Neonectria* can actually suppress scale insect densities after the initial invasion.

Conclusions

Native and nonnative pests and pathogens shape forests and ecosystems worldwide. Understanding how such organisms vary and co-vary in a spatial context provides insight into many of the processes driving the abundance and distribution of organisms. By considering multiple spatial scales, the present study permitted inferences regarding the relative contribution of different demographic drivers and the spatial extent at which they operate. This general approach may have broader utility, particularly for forest pests and pathogens where relevant dynamics often appear to operate at landscape scales.

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Supporting information

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Fig. S1. Plot design for spatially mapped sites (n = 7). A Haglöf DME 360 ultrasound distance transponder was placed

consecutively at the centre of each circular subplot (marked with a blue 'x'), and distance readings were taken with a handheld receiver placed alongside each tree (i.e. diameter at breast height), aiming toward subplot centre. A second fieldworker, standing at subplot centre, recorded the azimuth for each tree using a compass. Together, distance and azimuth values were combined to accurately define each tree's location within the total plot area (0.28 ha.). Edge trees (outside the dotted red line, 6 m from the plot border) were excluded as focal trees in our analyses of spatial aggregation.

Fig. S2. Sample spatially mapped plot (New York site 613B). Blue dots represent living beech trees; circle size is proportional to diameter at breast height. Each tree was scored for a host of disease characters, as well as crown class, crown colour and fullness, and apparent overall health (for details, see Materials and Methods).

Fig. S3. Surface contour plots drawn from 35 sites (black circles) showing spatially interpolated densities of scale insects (a) and *Neonectria* (b) across northeastern U.S.A., 2005–2007. Population estimates for Bartlett and Hubbard Brook Experimental Forests (New Hampshire) represent the mean value derived from intensive sampling at these sites (n = 22 and 25 plots, respectively).

Table S1. Description of numerical codes for quantifying scale insect and *Neonectria* level on living beech trees. All codes are derived from Houston *et al*. (1979) adopted in part to facilitate comparison between current and past infestation levels in historically monitored plots (Garnas *et al*., 2012). Field training was provided by David R. Houston to ensure consistent classification.

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