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

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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 native *Neonectria* fungi that 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: 1) local contagion within stands; 2) regional contagion, or among patch infection-reinfection dynamics; 3) variation in host susceptibility linked to genetic and/or environmental heterogeneity; and 4) climate effects on population growth of BBD organisms.

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

Keywords: beech bark disease; forest pestilence; *Cryptococcus fagisuga*; *Neonectria faginata*; *Neonectria ditissima*; spatial epidemiology

Abbreviations: Beech bark disease; BBD

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 (Liebhold & Tobin, 2008; Liu *et al.*, 2007). Once an area is fully colonised, 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 (Burdon *et al.* 1989; Koenig,

1999; Krivan *et al.*, 2008; Ryti & Case, 1986). Understanding the degree and spatial scale at which populations co-vary permits inference about underlying mechanisms likely to have produced observed patterns (Bjørnstad *et al.*, 1999; Liebhold *et al.*, 2004; Peltonen *et al.*, 2002; Silvertown *et al.*, 2001). 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; Liebhold & Tobin, 2008; Post, 2005). Such an approach may 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 (Aukema *et al.*, 2008; Liebhold & Tobin, 2008; Liu *et al.*, 2007; Peltonen *et al.*, 2002). Consideration of multiple spatial scales can improve mechanistic understanding of important system processes, as 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 (N. faginata* or *N. ditissima,* and possibly *Bionectria ochrolueca;* Castlebury *et al.*, 2006; Ehrlich, 1934; Houston, 2005; Houston *et al.*, 2005). *Neonectria faginata,* however, is highly specific to the BBD system and appears to predominate in the aftermath zone of the disease, where this study was conducted (Houston, 1994a; but see Kasson & Livingston, 2009). Both insects and fungi are required for disease development – insect feeding facilitates colonisation of phloem tissue by *Neonectria* (Ehrlich, 1934), though exact mechanisms remain elusive (Cale *et al*., 2011). 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).

Beech bark disease 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 *N. coccinea* var. *faginata*) are native to North America (Castlebury *et al*., 2006). *Neonectria ditissima* (formerly *N. 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 reduced abundance of large beech but increased densities of smaller beech trees (Ellison *et al.*, 2005; Garnas *et al.*, 2011a; Houston 1994**b**).

Biological mechanisms promoting spatial autocorrelation at multiple scales

In this study we evaluated spatial patterns in the abundance of BBD organisms to test hypotheses regarding the roles of contagion, variation in host susceptibility, and exogenous demographic effects. Specifically, we hypothesised that BBD dynamics are influenced to varying degrees by 1) local contagion driven by dispersal of insects and fungal spores from infected to adjacent, uninfected trees; 2) regional contagion, where stand BBD severity is linked to the frequency and intensity of colonisation from nearby infected stands; 3) variation in host susceptibility linked to genetic and/or environmental heterogeneity; and 4) 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, considered separately) where scale insects and fungi have been long established and are effectively endemic, so we did not consider invasive spread as a theoretical candidate for producing spatial patterns in our data, but we acknowledge the importance of the initial invasion process as a driver of spatial pattern at a broader geographic scale. (Morin *et al.*, 2007). Because some of the hypothetically important drivers of population dynamics differ in 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: 1) trees within stands; 2) stands within forests; and 3) 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 one per cent 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, though the contribution of the local environmental variability is unknown (Houston & Houston, 2000; Wargo, 1988). 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 due to 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 that produce 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 subcontinental 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 geographic barriers, or where directional asymmetry (anisotropy) exists (Forchhammer & Post, 2004; Peltonen *et al.*, 2002; Post, 2005; Stenseth *et al.*, 2002, Halkka *et al.*, 2006). Covariance that decays rapidly with distance or that is characterised by travelling waves indicates the importance of dispersal (Bjørnstad *et al.*, 2002; Grenfell *et al.*, 2001). 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 (Bjørnstad *et al.*, 1995; Hanski *et al.*, 1991), 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), but it seemed 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.*, 2011b).

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 Table S1 for details). We averaged population estimates across height

zones to obtain a single measure per tree. Sampling was restricted to trees > 10 cm DBH because smaller trees are only rarely colonised 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). While 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 seems 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. 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 (15 m radius, reflecting the use of an ultrasound distance-measuring tool [Haglof DME 360]; Fig. 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 (DRH) in 1979 to

capture both the advancing front and the aftermath zone of BBD (see Garnas *et al.*, 2011b for details).

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, but they ranged from barely detectable to highly abundant. We classified each tree's status for scale infestation as low or high (scale index ≤1 vs. >1) such that about half of the trees fell into each category (the same tests with alternative thresholds for low vs. high gave very similar results). For *Neonectria*, which was frequently absent on trees, we classified trees based on presence vs. 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 six metres of the plot boundary were excluded as focal trees to preclude edge effects. The average NIND for the stand was then compared to the frequency distribution of possible NINDs assuming random dispersion. For this, 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 5,000 times and compared the resulting frequency distributions to the empirical value for each site. Nonrandom dispersion was indicated by extreme values for the empirical NIND compared to the frequency distribution of possibilities under the null hypothesis of random dispersion.

Mesoscale (stands within forests)

In 2007, we randomly selected 22 0.1-hectare permanent plots in Bartlett Experimental Forest (Bartlett, NH) from a subset of 41 plots studied by Twery & 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, NH). 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 since 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. Average plot size was about the same as in the Bartlett study but 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 (Bjørnstad & Falck, 2001; Hall *et al.*, 1994) 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 non-zero values and estimates the slope of the presence-absence versus non-zero 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, so the results from this approach are not reported. Analyses were performed in R 2.6.1, including package fields and ncf (Bjørnstad 2008; Nychka 2007; R Development Core Team 2008).

Landscape scale (forests within the landscape)

In 2006-07, we sampled 20 additional study plots from Maine to West Virginia originally established by DRH and colleagues ca. 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), 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 2006-07 consisted of measuring all beech and sugar maple within two 100×5 metre random transects and two variable radius plots

(minimum count: 15-20 beech). Protocols for sampling disease and tree health were described in detail elsewhere (Garnas *et al.*, 2011b; Houston *et al.*, 2005).

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 twelve stands where there were no detectable fungal infections. We ran all landscape-level analyses both with and without Michigan sites, as 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, but 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.*, 2011b). To provide visualisation of broad geographic 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 to 85% of total live basal area among the 84 plots (mean ± SD = 38 ± 19%; Table 1). Surprisingly, both the per cent 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 ranging from 0 to 42% (median = 11%). There was no obvious relationship between apparent mortality and disease severity or latitude, though the highest mortality was in the Adirondacks, NY, where there was severe damage from an ice storm in 1998. The size distribution of beech stems > 10 cm likewise 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 elsewhere (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, respectively) though 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* (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, per cent basal area for sugar maple was spatially correlated (r = 0.66 at the nearest distances, decaying to zero at ~275 km). Within Bartlett Forest, per cent basal area for species other than beech or sugar maple was also autocorrelated (r = 0.82, to ~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). Of course, 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, df = 82, P = 0.31). On individual trees, there was a weak but significantly positive correlation between insect and *Neonectria* densities (r = 0.15, df = 2944, P < 0.0001).

Spatial dispersion in BBD agent populations

We found virtually 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 Experimental Forests (Fig. 2a, b, d, and e), or within the subcontinental landscape (Fig. 2c and 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 United States, surface contour plots suggested an inverse pattern in population densities for scale insects versus fungi (Fig. S3). Scale insect densities were highest in West Virginia and northern New England, whereas *Neonectria* densities were highest in southern New England (ca. CT and MA), 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 colonisation 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 (Gilligan & van den Bosch, 2008; Peltonen 2002). 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 colonised 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 (1st instar insect "crawlers" and fungal spores) occur at high enough 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), though 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, though 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 minimise the importance of local dispersal or metapopulation processes. Particularly for scale insects, tree level extinction appears to be a rare event (Garnas *et al.*, 2011b), 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, though the longevity of local infections within phloem tissue (prior to sporulation) is not well known. For *N. ditissima*, inoculum can be present on non-beech hosts within a stand independent of BBD, though 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 due to 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 ~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, but 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, while 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. Due to 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, 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.*, 2011b). 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 (Williams & Liebhold, 2000; Koenig, 1999; Liebhold *et al.*, 2004; Peltonen *et al.*, 2002; Johnson *et al.*, 2005, Post, 2005; Stenseth *et al*., 2002). 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.*, 2011b).

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 homogenising abundance in the landscape (Dolman & Sutherland, 1997; Schneider, 1992). 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 have never been 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, 1983). V*erticillium lecanii* has been isolated from numerous insect hosts in North America as well as recovered from soil samples in beech-dominated forests, but there have been no reports of 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*, but seems 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

This 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, 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 seems 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 (see also Morin *et al.*, 2007) and thus host density seems 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, but 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 now seems probable that virtually every beech tree is exposed to BBD agents throughout the currently colonised region of North America. This is not unlike other invasive pathogens such as chestnut blight and Dutch elm disease, among others, which have spared very few trees throughout North America (Burdon *et al.*, 2006; Gibbs & Wainhouse, 1986). Within the aftermath zone (*sensu* Shigo, 1964), stands devoid of scale insects or fungi are rare or nonexistent. 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 geographic spread of BBD been relatively slow (~15 km per year; Morin *et al.*, 2007) such that only around 50% of the range of the host is currently colonised? Annual tree-level extinction is low for both scale insects and *Neonectria* where BBD is endemic (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 nearly devoid of microstructure, in contrast with the cankered and gnarled state of many trees that have survived repeated BBD infection (pers. obs.; 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* (Houston, 1983b; Perrin, 1980; Shigo, 1964), 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 geographic 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 nonexistent, and may even be negative at the largest spatiotemporal scales (Garnas *et al.*, 2011a). If scale insect success within the advancing front depends on *Neonectria*, this could seriously slow the invasion, as the fungus typically lags scale by ~10 years (Houston, 2005). However, there now seems reason to question the conventional wisdom that scale insects benefit from the presence of the fungus (Dukes *et al.*, 2009; Houston *et al.*, 1979; Shigo, 1964). Our Michigan sites had by far the highest scale insect densities observed in our multi-state surveys and also had high mortality among large beech, but *Neonectria* was either absent or extremely rare (though the fungus has been found elsewhere in MI; Castlebury *et al.*, 2006). Furthermore, the region of highest *Neonectria* abundance (western Massachusetts and southeastern New York) had relatively low abundances of scale insects (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 following the initial invasion.

Conclusion

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 abundance and distribution of organisms. By considering multiple spatial scales, our 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 seem to operate at landscape scales.

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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 *Neo*. index	0.31 ± 0.30
Mean DBH beech (cm)	18.7 ± 5.9
Count beech	44.4 ± 36.3
Slope (deg.)	12.4 ± 11.3
Aspect	all
Elevation (m)	459 ± 202

[a] Basal area = m^2/ha

Table 2. Nearest infected neighbour distances (NIND) for scale insects and *Neonectria* on trees within 7 stands in Maine, New York, Massachusetts and West Virginia. P-values represent tests for spatial aggregation vs. the null hypothesis of random dispersion.

	Site	Empirical mean NIND (m)	P-value
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†
	WV 820	8.2	0.30
	WV 821	6.1	0.20

†0.10 < p < 0.05; *0.01 < p < 0.05; **0.001 < p < 0.01

Figure captions

Figure 1. Frequency histograms showing tree size distributions by abundance index for scale insects (left) and *Neonectria* (right). "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*.

Figure 2. Spline correlograms depicting correlations (± 95% confidence intervals) as a function of distance for scale insect (top row), *Neonectria* (middle row) and their cross-correlations (bottom row). 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.

Fig. 1

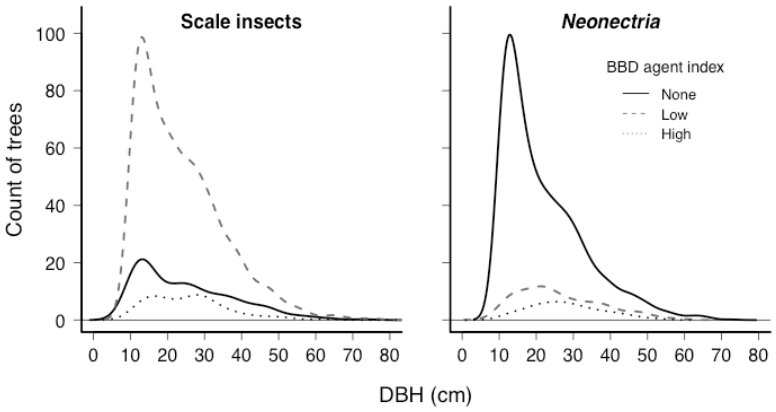
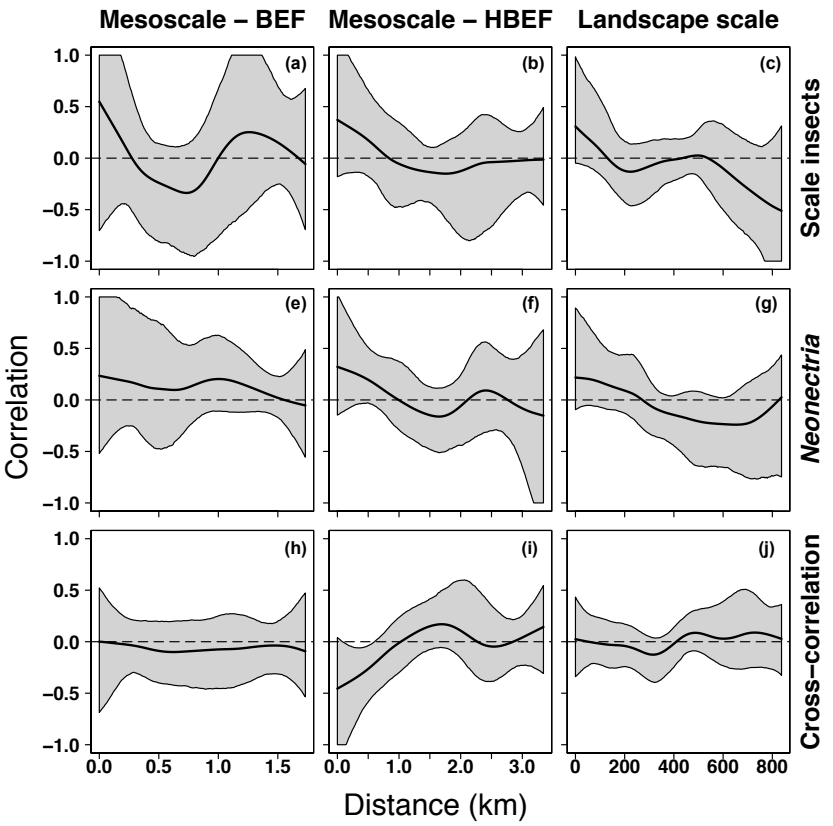

Scale insects
Neonectria
BBD agent index
None
Low
High
Count of trees
0
20
40
60
80
100
0 10 20 30 40 50 60 70 80
DBH (cm)

Fig. 2


Mesoscale – BEF
Mesoscale – HBEF
Landscape scale
Scale insects
Neonectria
Cross–correlation
Correlation
Distance (km)
(a)
(b)
(c)
(e)
(f)
(g)
(h)
(i)
(j)
1.0
0.5
0.0
−0.5
−1.0
0.0
0.5
1.0
1.5
2.0
3.0
0
200
400
600
800