Beech bark disease in northern hardwood forests: the importance of nitrogen dynamics and forest history for disease severity

Erika F. Latty, Charles D. Canham, and Peter L. Marks

Abstract: Beech bark disease has been a major cause of mortality of American beech (*Fagus grandifolia* Ehrh.) in North America during the past century. Previous studies have suggested a positive relationship between disease severity and both bark nitrogen content and tree size, presumably due to higher rates of infestation by beech scale insects, which allow more extensive infection of the tree by *Nectria* fungi. Recent concerns about nitrogen saturation in northeastern forests, particularly in old-growth forests, led us to examine patterns of disease severity as a function of bark tissue nitrogen content in old-growth and second-growth forests in the Adirondack region of New York and northern Maine. Trees growing in old-growth stands possessed significantly higher levels of bark nitrogen than similarly sized trees in second-growth forests. The severity of disease symptoms was more acute in the old-growth forests and was positively correlated with the percent nitrogen of the bark in both forest types. Comparisons of the coefficients of variation between beech bark sampled from disease-free forests in the upper peninsula of Michigan and that sampled from diseased forests indicated that elevated bark nitrogen concentrations in diseased trees were a cause and not an effect of disease presence. While there was no difference in disease severity between control and nitrogen-fertilized forests in Maine, these forests had both been exposed to the disease for longer time periods than the other studied forests and they are likely approaching nitrogen saturation.

Résumé : En Amérique du Nord, la maladie corticale du hêtre a été une cause importante de mortalité du hêtre d'Amérique (*Fagus grandifolia* Ehrh.) au cours du dernier siècle. Des études antérieures ont laissé entrevoir qu'il y avait une relation positive entre d'une part la sévérité de la maladie et d'autre part le contenu en azote de l'écorce et la dimension des arbres, probablement à cause du taux élevé d'infestation par la cochenille du hêtre qui favorise une plus forte infection des arbres par les champignons du genre *Nectria*. Des préoccupations récentes au sujet de la saturation en azote dans les forêts du nord-est, particulièrement dans les vieilles forêts, nous ont amené à examiner le comportement de la sévérité de la maladie en fonction du contenu en azote de l'écorce dans les vieilles forêts et les forêts de seconde venue de la région des Adirondacks, dans l'État de New York et le Nord du Maine. Les arbres qui croissent dans les vieux peuplements présentent des niveaux d'azote dans l'écorce significativement plus élevés que les arbres de même dimension dans les forêts de seconde venue. La sévérité des symptômes de la maladie est plus aiguë dans les vieilles forêts et est positivement corrélée avec le pourcentage d'azote dans l'écorce dans les deux types de forêts. La comparaison des coefficients de variation entre l'écorce de hêtre échantillonnée dans des forêts exemptes de maladie, dans la péninsule nord du Michigan, et l'écorce provenant de forêts infectées montre que la concentration élevée de l'azote dans l'écorce des arbres malades est une cause et non un effet de la présence de la maladie. Alors qu'il n'y a pas de différence dans la sévérité de la maladie entre des forêts fertilisées à l'azote et des forêts témoins dans le Maine, ces deux types de forêts ont été exposées à la maladie pour une période de temps plus longue que les autres forêts étudiées et sont selon toute vraisemblance sur le point d'être saturées en azote.

[Traduit par la Rédaction]

Introduction

American beech (*Fagus grandifolia* Ehrh.) has experienced a substantial decline due to beech bark disease since the predisposing agent, the woolly beech scale *Cryptococcus fagisuga* Lind. (= *Cryptococcus fagi* Baer.) (order Homoptera: family Eriococcidae), was first introduced into Halifax, Nova Scotia, on ornamental European beech trees (*Fagus sylvatica* L.) prior to the 1890s (Ehrlich 1934; Houston 1994). In the Adirondack region of New York, U.S.A., the mortality of

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large beech trees coupled with the reduced vigor of smaller stems has resulted in a 37% decline in beechnut production (Sage 1996). This decline in seed production has implications for wildlife because the fall weights of deer, reproductive rates of black bears, and overwinter survival of chipmunks and other small mammals are affected by the abundance of beechnuts (Rogers 1976; McLaughlin et al. 1993). Furthermore, stands with heavy mortality due to the disease alone or by subsequent salvage logging are often replaced by dense stands of susceptible beech sprouts that prevent the regeneration of other hardwood species (Houston 1975, 1994). Disease presence and progression within forested stands have been linked to a number of causal and contributing factors, including scale insect population densities, genetic resistance of the host, and the nutritional quality of beech bark (Houston 1994; Houston and Houston 2000). However, relatively little work has focused on how these factors change with the disturbance history of the forest to influence broader patterns of disease severity.

Beech bark disease is the result of an insect–fungal pathogen complex that is initiated by the infestation of beech bark by *C. fagisuga*. The insects feed by inserting their stylets into the bark parenchyma tissue (Wainhouse and Gate 1988). Amino N is of critical importance to the nutrition of such sucking insects (Dadd and Mittler 1965), and acquisition of key amino acids, such as methionine, can influence their growth and development (Mittler 1967; Dadd and Krieger 1968). The feeding activity of *C. fagisuga* colonies predisposes the bark of beech trees to infection by several species of Ascomycete fungi of the *Nectria* genus, primarily the introduced species *Nectria coccinea* var. *faginata* Lohman, Watson and Ayres and the native species *Nectria galligena* Bres. (Mahoney et al. 1999).

Although disease severity in North America, as in Europe, is most strongly linked to the patterns of scale establishment, the factors determining scale success remain largely unknown (Ehrlich 1934; Houston et al. 1979). Scale populations are often denser in the United States, perhaps due to the absence of natural enemies or coevolved defenses in American beech that limit the development of scale populations on European beech trees (Gibbs and Wainhouse 1986). A critical question in understanding the epidemiology of the disease is not what limits the spread of the fungal pathogens in these stands, but what limits establishment of the scale insect.

Stand-level factors that have been proposed to explain disease severity patterns include tree size, the species composition of a given forest, climatic variables, and the amino N content of beech bark (Houston and Valentine 1988; Houston 1994; Gore and Houston 1996). The severity of disease symptoms increases significantly with tree diameter, and this relationship holds in both old-growth and second-growth forests (Houston 1975; Mize and Lea 1979; Gavin and Peart 1993). As trees age and grow, they develop more spatially suitable habitat for the scale insects such as branch scars, bark fissures, and callused areas around wounds (Houston 1994). It is also reasonable to assume that there is a greater probability that more insects will infest a tree as its crown area increases and therefore captures more windborne larvae. However, due to low wind speeds in forests and the small size of the scale during the dispersal phase, there is greater probability that the first instars will be diverted around rather than deposited upon large trunks (Wainhouse and Gate 1988).

It has been shown that the bark of resistant beech trees has significantly lower concentrations of individual amino acids and total amino N relative to susceptible trees and that larger trees have higher levels of amino N in their bark compared with smaller trees (Wargo 1988). The quality of the feeding substrate, in this case the N content of bark tissue, is likely an important factor in determining how heavily infested a tree will become. Therefore, to better understand variation in regional and stand-level patterns of scale establishment and disease severity requires knowledge of the relationship between forest successional stage and the N content of the bark tissue on which the scale insect feeds as well as the collective effects of tree size, forest composition, and the duration of scale infestation.

The successional stage and (or) disturbance history of a forest are likely to influence its N capital, thereby determining the N status of individual trees and their response to chronic N deposition. As the total quantity of a limiting nutrient (such as N in temperate forests) amasses over time, supply eventually exceeds plant demand and the nutrient should no longer limit plant production (Vitousek and Reiners 1975; Gorham et al. 1979). Mature forests may be especially at risk of becoming saturated with N due to their low biotic demand for N relative to its supply (Aber et al. 1989, 1998; Hedin et al. 1995; Fenn et al. 1998). One common response of trees to increased N loading is greater incorporation of N into foliage (Nadelhoffer et al. 1995; Magill et al. 1996; McNulty et al. 1996). Fertilization experiments using ^{15}N-labelled nitrate additions have shown that American beech foliage accounted for as much as 50% of nitrate assimilation even when this species represented only 3% of the total foliar biomass (Nadelhoffer et al. 1995). Nitrogen enrichment of beech bolewood was also detected during the course of this 4-year experiment (Nadelhoffer et al. 1995). Nitrogen additions have resulted in dramatic increases of arginine, a N-rich amino acid, in the needles of Norway spruce (*Picea abies* (L.) Karst.) (Nasholm et al. 1997) and in grand fir (*Abies grandis* (Dougl. ex D. Don) Lindl.) where measured levels of N were toxic to balsam wooly adelgid (*Adelges piceae* (Raty.)) (Carrow and Betts 1973). Arginine is also one of the amino acids found to be higher in the bark of infested beech trees (Wargo 1988). However, whether N additions accelerate mortality due to beech bark disease currently is unknown (Magill et al. 1996).

Here, we report on patterns of beech bark disease severity in northern hardwood forests of New England and New York. Specifically, we tested the following hypotheses. (*i*) Disease severity at the stand level is related positively to tree size and the concentration of N in the bark. (*ii*) Old-growth trees have higher concentrations of bark N than do second-growth trees and this contributes to higher disease severity in old-growth forests as compared with second-growth forests rather than being an effect of disease presence. We also investigated how bark N levels were influenced by tree size. To assess whether disease presence increased bark N concentrations, we sampled bark from beech trees in regions not yet exposed to the disease.

(*iii*) Patterns of disease severity are a function of both the history of the forest and the duration of disease presence in the stand. We hypothesized that beech in old-growth forests would exhibit more severe disease symptoms compared with beech in second-growth forests the potential for old-growth forests to harbor N quantities in excess of biotic demand. (*iv*) Eight years of N fertilization should result in higher disease incidence and greater disease severity relative to a nearby, unfertilized forest.

Materials and methods

Study areas

The study was conducted in forests in New York, Maine, and Michigan. Old-growth forests were defined as forests lacking any known history of logging, agriculture, or human settlement. To sample old-growth forests as near a natural state as possible, forests were chosen that were of sufficient size as to maintain natural disturbance regimes (Cogbill 1996). In practice, this size requirement was a minimum of 500 ha. Nonetheless, the forests studied are not "primeval" because they have been subjected to a variety of human-induced environmental changes, including exotic species invasions and N deposition. The second-growth forests were harvested 60–100 years ago. Adirondack second-growth forests were logged heavily for spruce in the mid-1800s and logged selectively for hardwoods about 90 years ago (McMartin 1994). The second-growth forests of Maine were logged selectively for hardwoods early in the century following an initial removal of spruce.

The New York study sites were located in the central portion of the Adirondack Park, mostly in and around the Five Ponds and Silver Lake Wilderness Areas. The Five Ponds Wilderness Area is one of the largest tracts of old-growth in the eastern United States, comprising nearly 20 000 ha of contiguous old-growth forests (Roman 1980; Leopold et al. 1988). The soils are primarily Spodosols underlain by metasedimentary rocks. Average annual precipitation is variable, ranging from 100 to >130 cm with 200–400 cm of snowfall (Leopold et al. 1988). Dominant canopy tree species include American beech, sugar maple (*Acer saccharum* Marsh.), red spruce (*Picea rubens* Sarg.), eastern hemlock (*Tsuga canadensis* (L.) Carrière), yellow birch (*Betula alleghaniensis* Britt.), and red maple (*Acer rubrum* L.).

In northern Maine, research was conducted in the Nature Conservancy's Big Reed Preserve and adjacent second-growth forests. The Big Reed Preserve is a 2025-ha tract, the largest area of mid- to low-elevation old-growth in Maine (Davis 1996). The composition of these mixed northern hardwood forests was similar to that in the Adirondacks, with the addition of balsam fir (*Abies balsamea* L.) as a canopy dominant.

To test whether disease severity was correlated with bark quality required the sampling of reference trees, i.e., healthy beech trees in stands free of disease. Reference trees for such a study are difficult to obtain because the disease is well entrenched in forests of the northeastern United States. Those trees free of insect colonization and of disease symptoms within severely diseased forests are thought to be resistant to colonization by the scale insect. Experimental studies have demonstrated that beech trees free of disease in infected areas are likely to be genetically resistant to scale colonization, and as such, trees remain disease free even when experimentally infested with *C. fagisuga* (Houston 1983, 1994). To control for the effects of individual resistance within forests undergoing the advance stages of the disease, we chose beech trees from forests in Michigan, where the disease is in its early stages and disease-free stands may still be found (McCullough et al. 2001). The Michigan forests were located in the eastern and central portions of the Upper Peninsula (UP), in the Hiawatha National Forest, Dunbar Experimental Forest, and the Jim Wells Forest. This area is part of the central lowlands geomorphic province that is marked by lacustrine deposits of sand, silt, clay, and (or) marl with locally exposed deposits of limestone and dolomite (Beyer et al. 1997). The region receives heavy snowfall and its climate is significantly modified by the Great Lakes. In addition to American beech, other dominant species include sugar maple, eastern hemlock, and yellow birch. All forests sampled in Michigan were second-growth, having been logged within the last 50–100 years.

To examine the relationship between bark N concentration and disease severity within the context of increases in anthropogenic N deposition, studies of fertilized and unfertilized beech trees were undertaken at the Bear Brook watershed located in eastern Maine (44°52′N, 68°06′W) on the southeastern slope of Lead Mountain (475 m elevation). Watershed fertilization treatments at Bear Brook began in November 1989 to assess the impacts of chronic S and N deposition on forest biogeochemistry. Soils are Spodosols (Typic Haplothods) underlain by till. The relatively young 40- to 60-year-old forests are dominated by American beech, red spruce, balsam fir, red maple, sugar maple, and yellow birch. Dry $(NH_4)_2SO_4$ was applied bimonthly to the 10.2-ha West Bear Brook catchment from 1989 through 1991 (Kahl et al. 1993). The loading rate was 25 kg $N \cdot ha^{-1} \cdot year^{-1}$ whereas estimated vegetation requirements were 80–120 kg $N \cdot ha^{-1} \cdot year^{-1}$ (Kahl et al. 1993). Unfertilized trees were sampled in the adjacent 10.9-ha East Bear Brook catchment, which serves as the control. Further information regarding the Bear Brook Watershed Manipulation project may be found in Norton et al. (1999).

Sample collection and analysis

To compare bark N concentrations and patterns of disease severity between forests with different histories or fertilization regimes, 0.2-ha plots (circular plots with a 25-m diameter) were located randomly within each forest. In June–July in 1996 and 1997, eight replicate plots were established at least 1 km apart in the Adirondack and Big Reed forests and 0.25 km apart in Bear Brook (due to the smaller overall size of the watershed). Plot surveys recorded the slope and aspect, and for all live and standing dead trees over 10 cm in diameter at breast height (DBH = 1.4 m from the ground), tree species and size were noted. All beech trees were scored for visible symptoms of beech bark disease (details below), and a random sample of beech trees stratified by tree size was selected for bark tissue analyses. The bark sampling procedure followed that of Wargo (1988), and three 2.5-cm-diameter discs of bark were removed per tree. The discs were taken from three randomly determined aspects and within 2 m of the ground. All sampled bark was free of scale

infestation, disease infection, physical injury, and foreign substances including lichens. Discs were dried and weighed before being pulverized for C and N analyses. An 8- to 10-g subsample was used for analyses on a Carlo Erba NA 1500 Series Nitrogen/Carbon Analyzer (CE Elantech, Milan, Italy).

We used a modified classification system of disease severity derived from that of Burns and Houston (1987). There were four classes of increasing disease severity based on visual external defects in beech bark. The 0 (none) class was assigned to individuals with no indication of bark defect attributable to disease. Class 1 (low) consisted of trees with small, discrete bark lesions (2–3 cm in diameter) resulting from scale and fungal infection. At this stage, the cambial tissue is only affected in localized areas. Individuals falling in class 2 (moderate) displayed obvious necrotic tissue with patches of blocky bark punctuated by sunken lesions. Trees with the most intense damage to vascular and cambial tissues were assigned class 3 (high). Long vertical fissures were present over >75% of the trunk bark surface and callus tissue had formed around old disease induced wounds. All beech deaths were assumed to be due to disease unless evidence existed to the contrary such as severe wind damage. Given the wide range of bark deterioration on standing dead individuals dependent upon time since death, dead stems were not assigned a disease severity class. The severity of beech bark disease was assessed in two ways: by calculating the relative number of diseased beech trees in each forest and by recording the frequency of beech trees within each disease severity class. To evaluate the previous extent of disease-induced damage, we examined the relative proportion of standing dead beech stems and basal area; however, standing dead individuals were not included in most statistical analyses with the exceptions noted below.

Derivation of probability functions and data analyses

Ordinal logistic models are designed to analyze the determinants of an ordinal (ranked) dependent variable. We used multinomial logits to determine the effects of tree size and bark N concentrations on levels of disease severity. All logistic regressions were performed using the SAS system for Windows, version 6.12 (SAS Institute Inc. 1989). The cumulative probability functions took the form

$$[1] \qquad \log\left(\frac{p_{js}}{1-p_{js}}\right) = a_s + c + \mathrm{DBH}_j$$

$$[2] \qquad \log\left(\frac{p_{js}}{1-p_{js}}\right) = a_s + c + \mathrm{BN}_j$$

where p_{js} is the probability that the disease severity of the jth individual is less than or equal to s, for $s = 0,..., 2$ disease classes, a_s and c are estimated parameters, DBH_j is the breast height diameter (cm) of the jth individual, and BN_j is the bark N concentration of the jth individual. As these probabilities are cumulative, to determine the probability of disease acquisition for a given level of s, the cumulative probability for level $s - 1$ is subtracted from the cumulative probability for level s. The ordinal model only needs to estimate three intercept terms (a) for the four disease levels because the probability that an individual has the highest disease class is simply 1 minus the cumulative probability that it has the next highest disease class.

To further examine the relationship between scale infestation and bark N concentrations, we tested the null hypothesis that there is no effect of the N concentration of beech bark on disease severity. Unfortunately, we cannot simply compare the average bark N concentrations in diseased and undiseased regions, as it is likely that concentrations vary for reasons other than disease (e.g., variation in atmospheric N deposition). Nonetheless, we may expect that the variation in bark percent nitrogen (%N) should not be significantly different for forests from regions with and without beech bark disease presence. If bark N concentrations are elevated by disease presence, we should expect less variation in bark %N measurements made in diseased forests relative to undiseased forests. Essentially, disease presence should elevate levels of bark N, thereby masking the natural variation. We compared variation in N levels of beech bark in two ways. First, we performed Bartlett's test of homogeneity of variance and computed coefficients of variation. Bartlett's test examines whether mean squares from two or more populations differ significantly (Snedecor and Cochran 1989). Having met the required assumptions, normal data distribution and sufficient degrees of freedom, we used Bartlett's test for samples of unequal sizes. Second, we calculated coefficients of variation to take account of the fact that the mean and standard deviation of a population often change together, a situation that could be attributed to edaphic or climatic differences between the regions. We did not include the Bear Brook fertilized stands in any of these analyses, as fertilization may be expected to decrease variation in the N concentrations of beech bark.

To better understand the relationship between bark N concentrations and tree size, we calculated the leaf and stem areas for a range of tree sizes using dimension analysis equations developed for beech trees in New Hampshire (Whittaker et al. 1974). To explore relationships between past and current forest structure by forest history and region, we used two-way analysis of variance (ANOVA) on normally distributed data. Most within-forest comparisons were made by one-way ANOVA.

Results

Disease severity as a function of tree size and levels of bark N

In both regions, the largest trees were, on average, the most severely diseased. Logistic regressions revealed that tree size was a significant ($P < 0.001$) predictor of the degree of disease severity for both old-growth and second-growth forests in the Adirondacks and in Big Reed.

Adirondack old-growth (Fig. 1*a*) and Big Reed old- and second-growth (Fig. 2) all demonstrated high probabilities of exhibiting severe disease symptoms over a wide range of tree diameters. This was in contrast with Adirondack second-growth in which small trees were most likely to be either undiseased or lightly diseased (Fig. 1*b*). Within Adirondack forests, fitted probability functions indicated that a median size tree of 15 cm had nearly a 20% higher chance of remaining disease free in the second-growth forests as compared with the old-growth (Fig. 1). Generally, old-growth

Fig. 1. Curvilinear functions for the probability of zero, low, moderate, or high disease severity across a range of tree sizes for Adirondack (*a*) old-growth and (*b*) second-growth. Broken vertical lines indicate tree sizes referred to in the text.

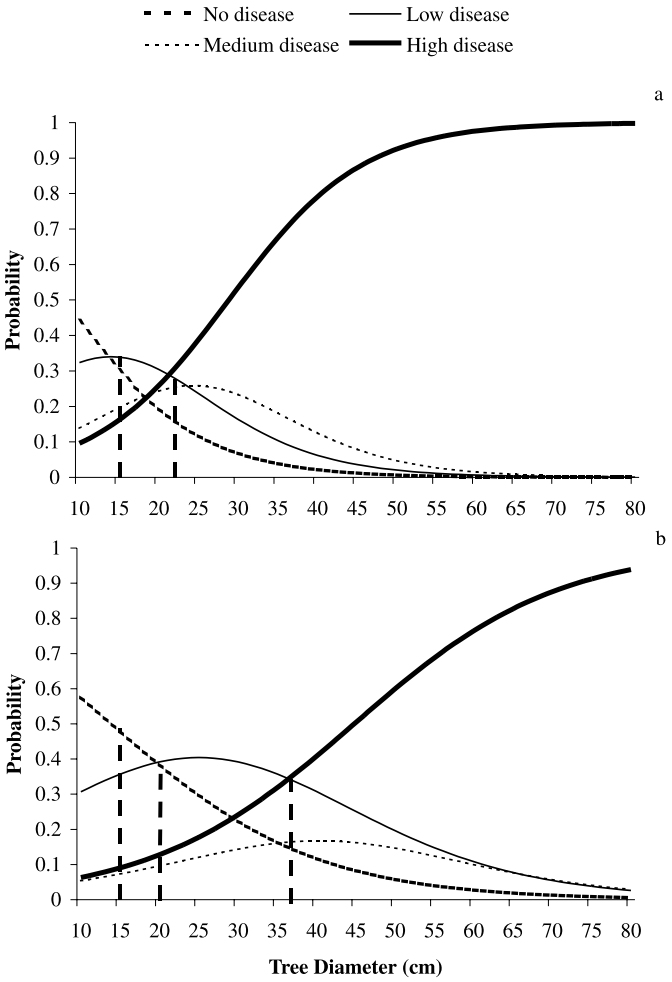
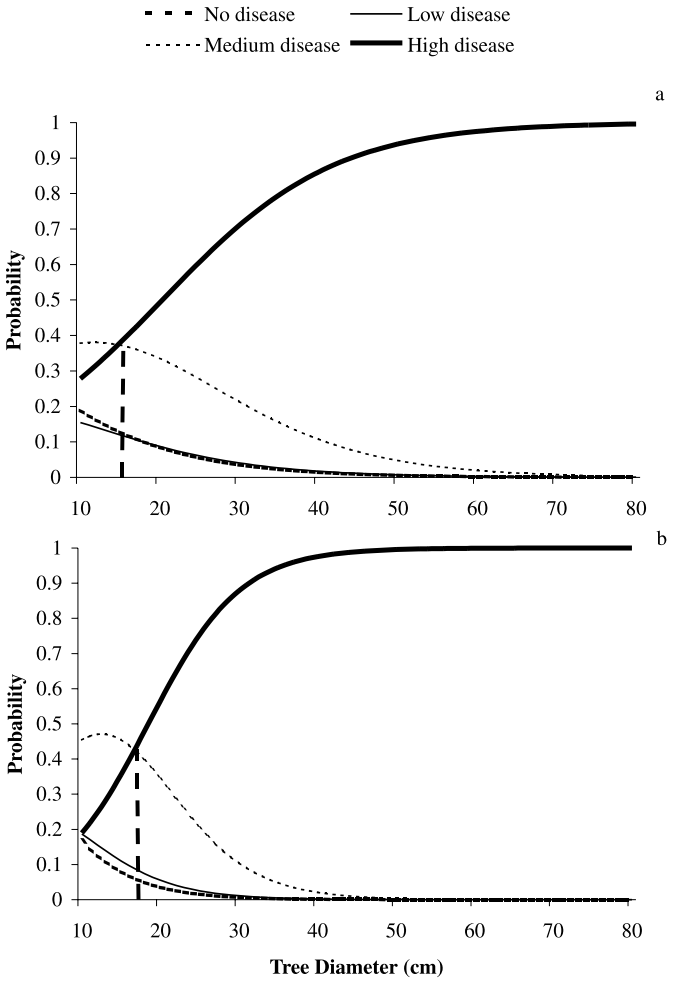


Fig. 2. Curvilinear functions for the probability of zero, low, moderate, or high disease severity across a range of trees sizes for Big Reed (*a*) old-growth and (*b*) second-growth. Broken vertical lines indicate tree sizes referred to in the text.



trees had higher probabilities of being more diseased than equivalently sized second-growth trees. For example, at diameters <22 cm in old-growth forests, trees were most likely to have no disease or low levels of disease. Trees >22 cm had a higher probability of being in the highest disease class. A similar shift from lower to higher disease severity occurred in the second-growth at a larger tree size, 37 cm. Trees 20–36 cm in second-growth were most likely to be moderately diseased; such a window in old-growth was narrower and at a smaller diameter, 15–22 cm. Similar trends were not observed at Big Reed. The highest probability (40%) of exhibiting moderate disease symptoms in Big Reed forests was for stems <15 and <18 cm for old- and second-growth, respectively (Fig. 2). For larger Big Reed trees, there was little chance (<20%) of remaining disease free or at low levels of disease regardless of forest history.

Logistic regressions demonstrated a significant effect of bark N concentrations on disease severity in all Adirondack and Big Reed forests (Table 1). In old-growth and second-growth forests in both regions, the mean bark %N of the most severely diseased trees was higher than for undiseased trees. Bark %N of acutely diseased tree was lower for trees at Big Reed than for trees in the Adirondack forests (Figs. 3 and 4). Higher probabilities of finding moderately diseased individuals occurred in the old-growth from both regions and the second-growth in Big Reed. Trees in the second-growth forests in the Adirondacks had the highest probabilities of exhibiting low levels of disease over the greatest range of bark N.

Bark N levels by forest history, tree size, and region

In both the Adirondacks and Big Reed, the average bark N concentration of old-growth trees was significantly higher than that of second-growth trees (two-sample t test: P = 0.0003 and P = 0.014, respectively) across all tree diameters. Concentrations of bark N were also positively correlated with tree size independent of region (Adirondacks or Big Reed) or forest history (old-growth or second-growth). A two-way ANCOVA revealed no significant effect of region (P = 0.278) or forest history (P = 0.714) on bark %N when tree size was used as a covariate. As there was a significant interaction between region and tree size (P = 0.033), we

Table 1. Results of logistic regressions testing the effect of N concentrations (% dry mass) of beech bark on disease severity.

	Adirondack Park, N.Y.		Big Reed, Maine	
	Old-growth	Second-growth	Old-growth	Second-growth
Intercept 1	1.16 (0.968)	3.75 (1.08)	5.01 (1.65)	11.7 (2.94)
Intercept 2	2.38 (0.990)	5.51 (1.15)	5.89 (1.68)	13.2 (3.01)
Intercept 3	3.54 (1.03)	6.01 (1.17)	7.54 (1.79)	14.8 (3.15)
Slope	–2.51 (1.19)	–5.20 (1.45)	–7.89 (2.01)	–18.2 (3.98)
χ^2 *P* value	0.0353*	0.0003*	0.0001*	0.0001*

Note: The SE of each estimate is in parentheses.
*Significant value.

Fig. 3. Curvilinear functions for the probability of zero, low, moderate, or high disease severity across a biological range of N concentrations of beech bark for Adirondack (*a*) old-growth and (*b*) second-growth. Broken vertical lines indicate concentrations of bark N referred to in the text.

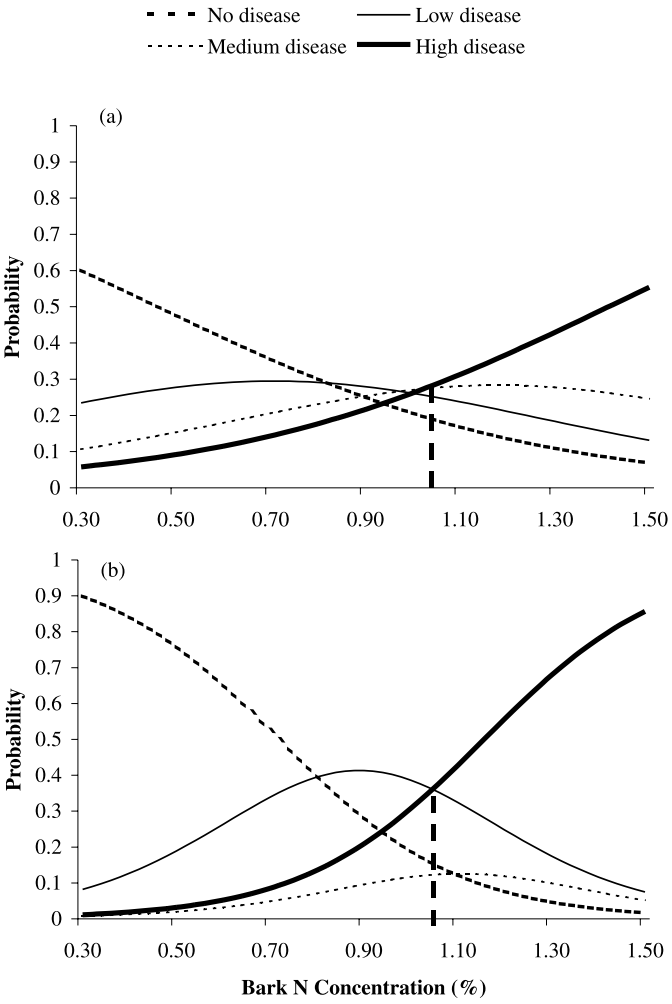
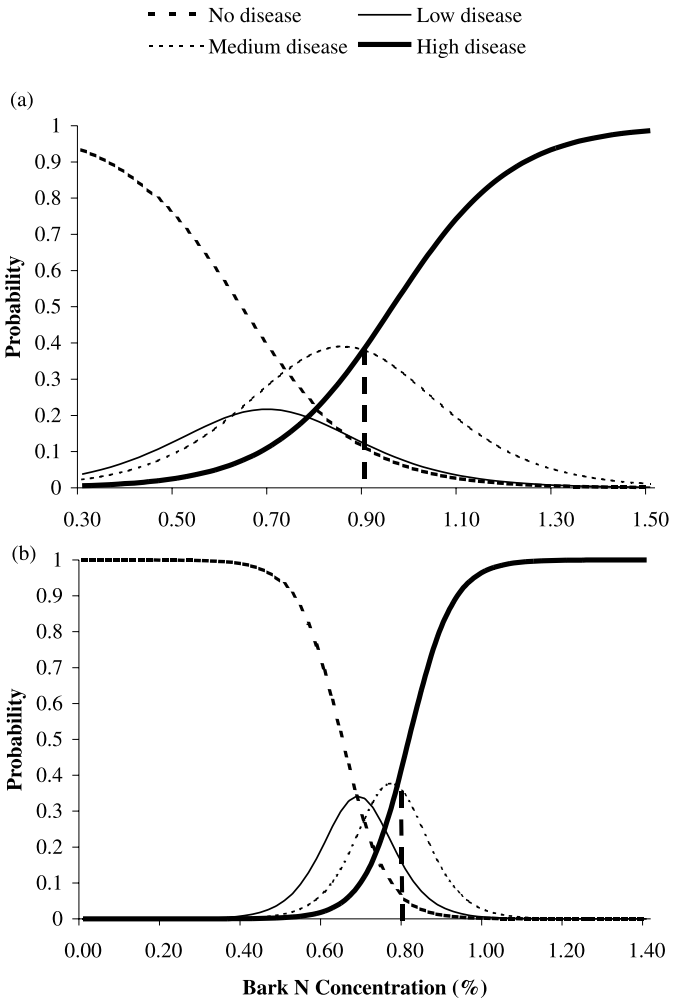


Fig. 4. Curvilinear functions for the probability of zero, low, moderate, or high disease severity across a biological range of N concentrations of beech bark for Big Reed (*a*) old-growth and (*b*) second-growth. Broken vertical lines indicate concentrations of bark nitrogen referred to in the text.



conducted a one-way ANOVA by region. The interaction term (forest type × tree size) was not significant for the Adirondacks ($P = 0.113$) or for Big Reed ($P = 0.949$). We pooled the data for forest types within regions and performed separate linear regressions of bark %N on tree size (linear regression: $n = 216$, $P < 0.001$, $r^2 = 0.054$ and $n = 128$, $P < 0.001$, $r^2 = 0.601$ for the Adirondacks and Big Reed, respectively). The dimension analysis equations demonstrated that the ratio of leaf to stem area increases as tree size increases (Fig. 5).

Variation in bark %N measurements taken from diseased stands in the northeast did not differ significantly from those

Fig. 5. Ratio of leaf area (cm^2) to stem area (cm^2) for beech trees over a range of tree sizes. Regression equations were adapted from Whittaker et al. (1974).

Table 2. Mean bark N concentrations (% dry mass) and coefficients of variation for beech trees in the three regions.

Forest	%N	Coefficient of variation (%)
Adirondacks, N.Y.		
Old-growth	0.807 (0.016)	20
Second-growth	0.735 (0.012)	18
Big Reed, Maine		
Old-growth	0.865 (0.018)	16
Second-growth	0.809 (0.014)	14
Bear Brook, Maine		
Unfertilized	0.815 (0.020)	16
Fertilized	0.810 (0.015)	12
Upper Peninsula, Mich.		
Second-growth	0.825 (0.015)	17

Note: The SE for the mean of each value is in parentheses. Bear Brook fertilized forests are included for comparative purposes but were not considered in the statistical analyses; see text for details.

measured in Michigan (Bartlett's test: $\chi^2 = 6.01$, $0.100 < P < 0.250$). The coefficients of variation ranged from 12% in the fertilized forests of Bear Brook to 20% in the Adirondack old-growth, with the disease-free second-growth forests from Michigan in the middle (coefficient of variation = 17%, Table 2). If the disease caused higher concentrations of N in bark, then we would expect reduced variation in bark N concentration. There also were no significant effects of the presence or absence of disease on bark N concentrations (GLM: $F_{[1,2]} = 0.746$, $P = 0.388$).

Among the three undiseased forests of Michigan, there were significant differences in bark N concentrations, and N concentrations were positively correlated with tree size. As there were significant effects of both forest and the forest × tree size interaction on bark %N (two-way ANOVA: df = 2, 1, 2, $P < 0.0001$ and $P < 0.05$, respectively), we performed separate linear regressions on each of the three Michigan forests. Significant positive relationships between tree diameter and bark %N were detected in two Michigan forests (linear regression: $n = 30$, $P < 0.05$ for the Dunbar ($r^2 = 0.486$) and Jim Wells ($r^2 = 0.453$) forests) but not in the Hiawatha forest ($n = 30$, $P > 0.05$, $r^2 = 0.104$), which also had the highest mean %N, 0.95% ± 0.017 SE (compared with 0.81% ± 0.021 SE and 0.73% ± 0.021 SE for the Dunbar and Jim Wells forests, respectively).

Disease severity patterns by forest history and region

There were substantial differences in disease severity between regions. There was a significantly lower percentage of live diseased trees (disease classes 2 and 3) in Adirondack forests relative to forests in Maine (two-way ANOVA: $F_{[3,39]} = 8.54$, $P < 0.001$) (Table 3). Within the Adirondacks, approximately 10% more of the trees in the old-growth were diseased relative to second-growth forests, a nonsignificant difference (one-way ANOVA: $F_{[1,25]} = 1.51$, $P = 0.23$). The relative proportion of diseased trees in Big Reed old- and second-growth were also statistically similar (one-way ANOVA: $F_{[1,14]} = 2.19$, $P = 0.16$).

A different measure of disease severity, the frequency distribution of trees across disease classes, also showed considerable differences between regions (Fig. 6). The majority of the Adirondack trees fell in the zero or low disease classes (Fig. 6*a*) whereas most were in the moderate to high disease classes in Big Reed (Fig. 6*b*). Within the Adirondack region, the disease severity distributions of old-growth and second-growth beech trees were significantly different from one another (two-sample Komolgorov–Smirnoff: $P = 0.004$). A higher proportion of trees in Adirondack old-growth were moderately and highly diseased compared with second-growth. In Big Reed forests, the old-growth and second-growth distributions were not significantly different (two-sample Komolgorov–Smirnoff: $P = 0.50$).

Standing dead tree surveys indicated that beech bark disease had been more lethal in Big Reed than it was in the Adirondacks (Table 3). Relative standing dead density was significantly higher in Big Reed than in the Adirondacks (two-way ANOVA: $F_{[3,39]} = 8.44$, $P < 0.001$). While old-growth forests in Big Reed had significantly higher percentages of standing dead beech density than did second-growth forests (one-way ANOVA: $F_{[1,14]} = 11.44$, $P < 0.001$), no such differences were detected in the Adirondack region (one-way ANOVA: $F_{[1,25]} = 0.27$, $P > 0.05$ and $F_{[1,25]} = 1.16$, $P > 0.05$, respectively) or for standing dead basal area in Big Reed (one-way ANOVA: $F_{[1,14]} = 2.15$, $P > 0.05$).

Disease severity patterns in fertilized and unfertilized forests

On average, 96% of the beech trees in the unfertilized and fertilized forests of Bear Brook were diseased. Individual beech trees were distributed similarly across disease severity classes in both forests (two-sample Komolgorov–Smirnof: $P > 0.05$ (Fig. 7). Size distributions of beech trees at Bear Brook were comparable with the other forests being compared (data not shown); again, as in other forests, the degree of disease severity increased positively with the mean tree diameter for forests that were both unfertilized (logistic regression: $\chi^2 = 13.26$, $P < 0.001$) and fertilized (logistic regression: $\chi^2 = 51.99$, $P < 0.0001$). Due to the low number of trees from the low disease severity class in the unfertilized forest, classes 1 and 2 were combined for statistical analy-

Table 3. Relative percentages of density and basal area for standing dead beech stems and the proportion of live beech trees exhibiting disease symptoms (n = number of stands).

Forest	n	Standing dead relative density (%)	Standing dead relative basal area (%)	Live diseased trees (%)
Adirondacks, N.Y.				
Old-growth	12	6.50 (0.02)	29.2 (0.09)	66.2 (0.05)
Second-growth	15	9.84 (0.03)	39.0 (0.15)	54.8 (0.07)
Mean		8.35 (0.02)	34.6 (0.09)	59.9 (0.05)
Big Reed, Maine				
Old-growth	8	22.9 (0.08)	29.3 (0.04)	87.2 (0.03)
Second-growth	8	12.5 (0.04)	18.9 (0.04)	91.4 (0.01)
Mean		17.7 (0.02)	24.1 (0.03)	89.3 (0.01)
P				
Region effect		0.0002*	0.4317	0.0001*
History effect		0.1386	0.9808	0.5630
Region × history		0.0055*	0.4243	0.2121

Note: All dead stems and live trees were >10 cm DBH. The SE for the mean of each value is in parentheses. Effects of region and history are presented as P values determined by two-way ANOVAs.
*Significant value.

Fig. 6. Frequency distributions of beech trees in the four disease severity classes (0 = none, 1 = low, 2 = moderate, 3 = high) for (*a*) the Adirondacks and (*b*) Big Reed.

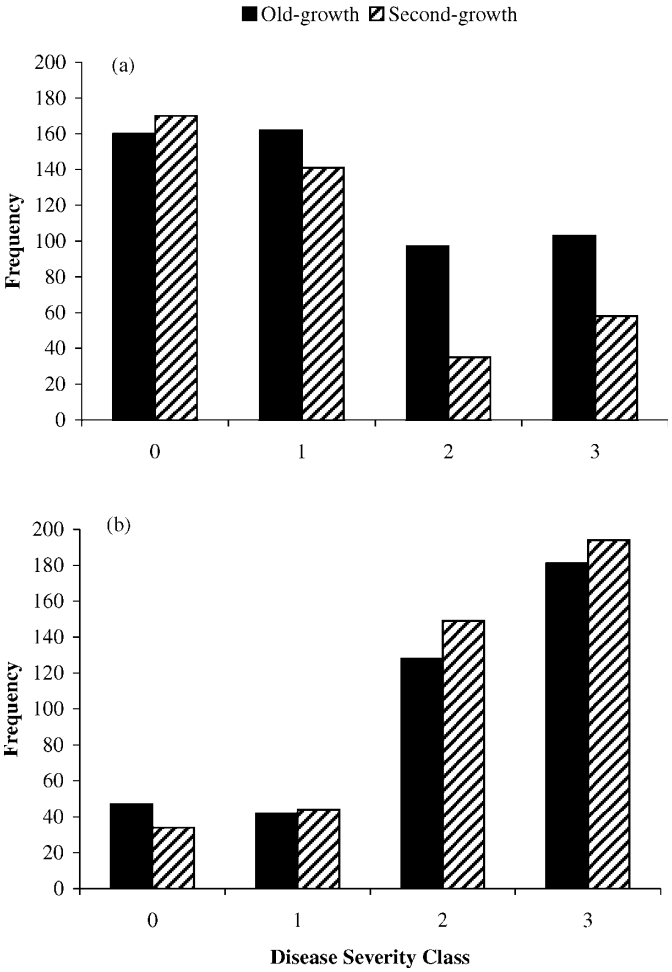
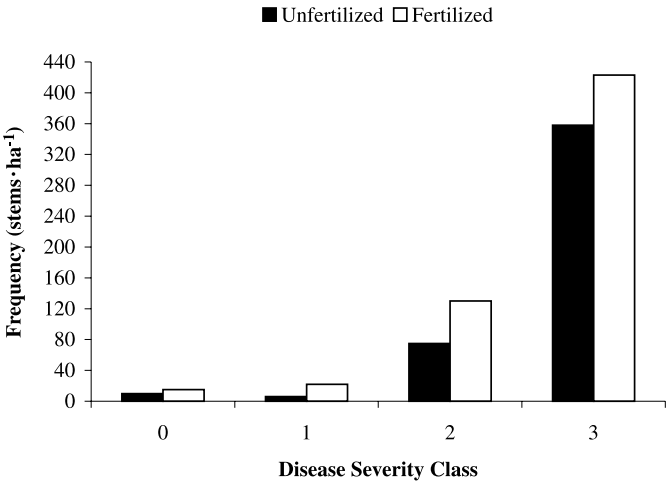


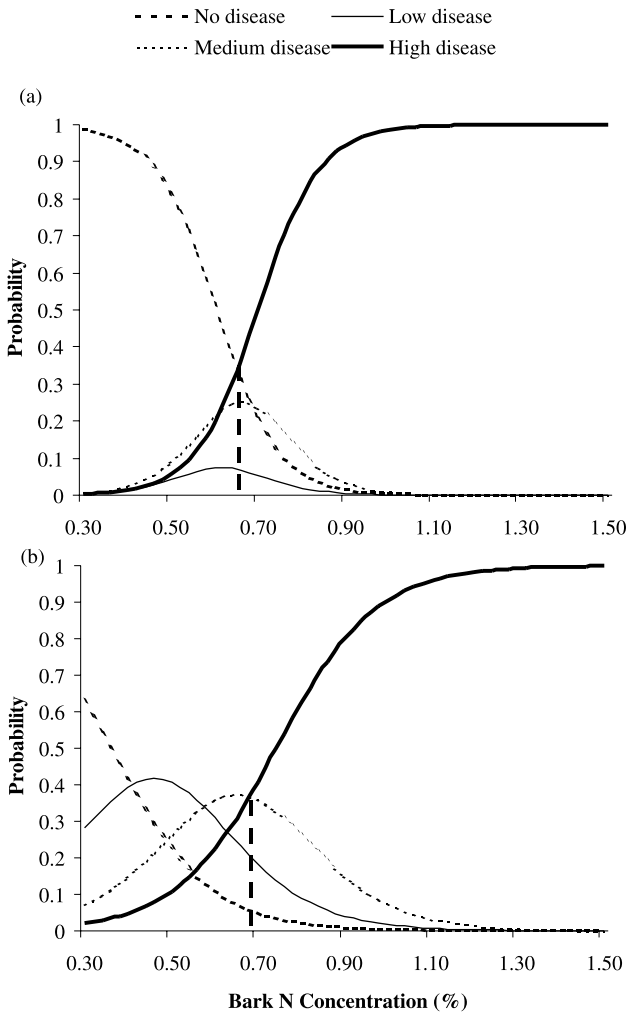
Fig. 7. Distribution of beech trees into four disease severity classes (0 = none, 1 = low, 2 = moderate, 3 = high) for unfertilized plots (n = 448 beech trees) and fertilized plots (n = 590 beech trees) from Bear Brook.



ses. The probability curves indicated that even the smallest individuals had a greater than 40% chance of being severely diseased, regardless of fertilization treatment (data not shown). These same individuals were about 20% more likely to be severely diseased in the unfertilized plots, but the probability of being undiseased was similar in both forests.

The mean N concentration in the bark of fertilized trees was 0.810% and for the unfertilized trees was 0.815%. The difference was not significant (one-way ANOVA: $F_{[1,8]}$ = 0.001, P = 0.982). Therefore, we combined data from the control and fertilized plots in analyzing the relationship between tree size and bark %N. As in other forests, there was a significant positive relationship between tree size and bark N concentration (linear regression: F = 41.25, r^2 = 0.35, P < 0.0001). There were also significant effects of bark N concentrations on disease severity in the unfertilized (logistic re-

Fig. 8. Curvilinear functions of the probability of zero, low, medium, or high disease severity across a biological range of N concentrations in bark for (*a*) unfertilized and (*b*) fertilized watersheds at Bear Brook. Broken vertical lines indicate concentrations of bark N referred to in the text.



gression: χ^2 = 7.30, P < 0.01) and fertilized (logistic regression: χ^2 = 4.61, P < 0.05) forests. Functions depicting the probability of being diseased over a range of bark N concentrations were different for unfertilized and fertilized plots, particularly at low levels of bark %N (Fig. 8). In the unfertilized plots, trees were most likely to be undiseased at low levels of bark %N and severely diseased at bark %N levels >0.65%. At bark %N levels <0.70%, fertilized trees had the highest probabilities of falling within disease classes 0–2.

Discussion

Disease severity was positively related to tree size, and our modelled probability curves demonstrated that the largest diameter trees in our study sites had nearly a 100% chance of exhibiting severe disease symptoms, regardless of region or forest history (Figs. 1 and 2). Other Adirondack studies also have found that beech trees killed by the disease tended to be larger than the survivors (Mize and Lea 1979). In the White Mountains of New Hampshire, similar patterns have been noted in old- and second-growth forests where tree size was again correlated with the severity of external disease symptoms (Gavin and Peart 1993). Bark N concentrations provide at least a partial explanation for this relationship. In each of the regions that we sampled, there was a significant positive relationship between tree size and bark N concentration. The relationship between bark N and tree size may be explained by a change in the ratio of leaf area to stem area as trees grow. If as a tree reaches canopy stature, its total leaf area increases faster than the stem surface area, then more photosynthates would be funneled through proportionally smaller phloem pathways, potentially resulting in high concentrations of photosynthate constituents, including N in bark. Our calculations demonstrated that leaf area increases more rapidly than stem surface area, supporting this hypothesis (Fig. 5). The resulting increased bark quality of larger trees appears to result in more heavily diseased trees.

Several lines of evidence suggest that elevated bark N concentrations are a cause rather than a result of disease occurrence. Wargo (1988) showed that in the absence of beech bark disease, large trees can have significantly higher concentrations of amino N in their bark relative to small trees. Our measurements from nondiseased trees in Michigan confirmed this and revealed also that variation in bark N concentration was similar in all regions regardless of disease status. Furthermore, levels of bark %N were not higher in diseased forests relative to undiseased forests, and the bark N levels from the most diseased forests were not consistently higher than those from less diseased forests. Others have also concluded that elevated bark N is a predisposing factor of disease, not the result of infection (Wargo 1988). The tendency of larger trees to harbor more insects is likely due in part to more suitable spatial habitat. However, it is also likely that the more nutritious bark of larger trees results in greater scale fecundity, which yields higher infestation densities, greater fungal infection, and more severe disease development.

In general, the Bear Brook and Big Reed forests of Maine were more diseased than the forests of New York. The number of individuals exhibiting signs of beech bark disease in our study forests ranged from none in Michigan to >95% of the beech trees in the forests of southeastern Maine. The simplest explanation is the spread of the scale insect from its point of introduction in Halifax, Nova Scotia. The progression of beech bark disease across regions has been subjectively divided into three stages: the advancing front, the killing front, and the aftermath zone (Shigo 1972). By the 1950s, the disease front had advanced throughout the state of Maine, and forests in the southern part of the state had progressed to the killing stages of the disease. The advance front spread through the western Adirondacks in the late 1960s followed by the killing front in the early 1970s (Houston et al. 1979). The disease reached the Big Reed area of northern Maine between 1945 and 1950, 15–20 years prior to sweeping through the Adirondacks; thus, those forests in which the disease has been present longer are also those forests that are most diseased. However, other differences among these forests, such as the effects of forest history and N deposition on bark quality, are important in determining variation in patterns of disease severity at a more local scale.

In the more recently affected forests of the Adirondacks, small-diameter trees in old-growth forests had relatively high probabilities of being heavily diseased whereas small-diameter second-growth trees had lower probabilities of exhibiting heavy disease loads (Fig. 1). These small trees also had lower levels of N in their bark relative to similarly sized old-growth trees.

Insects may be proliferating on trees with a smaller average diameter in old-growth relative to second-growth due to higher bark quality for a given tree size in the old-growth. The high quality of old-growth trees could be a result of these trees having access to larger N pools in the soil (Latty 2001). According to steady-state dynamics, one could expect old-growth forests to have larger N pools than previously logged forests due to the removal of biomass and (or) nutrient leaching due to logging practices (Bormann and Likens 1979). Logging resulted in lower N concentrations in the bark of Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco) trees compared with bark from trees growing in an uncut control 18 years after the treatment (Mitchell et al. 1996). Moreover, old-growth forests, particularly in the northeastern United States, are expected to be closer to N saturation (Hedin et al. 1995), a consequence of which is having elevated concentrations of N in biomass (Aber et al. 1998). Thus, forest history may help to explain the greater incidence of beech bark disease in old-growth forests of the Adirondacks.

In Big Reed forests, despite the higher bark quality of the old-growth trees, second-growth trees in a given disease severity class were not consistently larger than the old-growth trees. We attribute this lack of a forest history effect in Big Reed to the timing of the disease onset. During the earlier stages of insect establishment, bark quality is likely to be critical in determining the size of initial scale populations. Over time, as the amount of uninfected living bark diminishes, bark quality matters less than the quantity of uninfected bark in limiting scale establishment. In the long-diseased Big Reed forests, disease-free bark is likely more important in determining scale establishment than bark quality; thus, old-growth trees containing elevated levels of bark N are not obviously preferred by the scale. Taking a regional perspective of these forests, trees become more severely diseased at lower bark N concentrations as the duration of disease presence in a stand increases. The bark N concentration at which the probabilities shift to the greatest chance of being severely diseased is lower in Big Reed (<0.90%) and Bear Brook (<0.70%) than it is in the Adirondacks (>1.0%) (Figs. 3 and 4). The elevated bark N concentrations of old-growth trees in the Adirondacks may be causing these recently infected stands to more closely resemble the long-diseased stands of Maine, in terms of disease severity patterns by tree size.

Bear Brook forests were most severely diseased and have the longest history of disease presence of all of the forests in this study. As the N concentrations of beech foliage and bolewood increase in response to N fertilization (Nadelhoffer et al. 1995), we expected similar trends for bark tissue. Rather than cause bark N concentrations to increase, 8 years of fertilization treatments have not elevated bark N concentrations in Bear Brook, Maine. It has been suggested that even the reference watershed at Bear Brook is approaching N saturation (Aber et al. 1998). In this case, additional N amendments would have little effect on the quantity of N that is being stored in bark, since trees may already be subject to excess N supply. High rates of atmospheric N deposition could swamp the natural variation in concentrations of N in bark, thereby eliminating any resistance to the disease conferred by low %N in the bark. In keeping with the lack of bark N response to fertilization treatments, we saw no differences in overall disease severity between the unfertilized and fertilized forests (Fig. 7), and in both cases, the majority of the individuals were highly diseased. It is likely that most trees in this long-diseased area had significant disease development prior to the initialization of the fertilization treatments, a time when bark %N was probably similar between the fertilized and unfertilized stands, which may explain the comparable patterns of disease severity in these forests.

Conclusions

At a regional scale, proximity to the site of scale introduction is critical in determining current patterns of disease severity. The effects of beech bark disease were more acute in Maine forests than in New York forests. However, at a local scale, in areas in which disease presence is limited to approximately 30 years, tree size and N concentration of the bark are important in determining the severity of infection. Our results suggest that land use history is important in determining bark quality. High bark quality, in turn, predisposes beech trees to severe infection during the earlier phases of disease progression before space available for scale infestation becomes the limiting factor. Since 1974, forests of the western Adirondacks have contained well-established populations of *C. fagisuga.* Within a span of 7 years, the pathogen complex proliferated throughout these forests, tripling the beech mortality rates (Mize and Lea 1979). Disease occurrence has now been documented as far west as Michigan and sited as far south as Tennessee (Mielke et al. 1985; Houston 1994; McCullough et al. 2001). Our results suggest that forests, such as the Hiawatha forest of Michigan, that possess high mean bark N concentrations should be monitored carefully as the advance front of beech bark disease migrates west. Furthermore, in all forests, the effects of the disease should be considered in the context of regional N deposition. The high degree of disease infestation in the forests of Bear Brook indicate the potential for losing disease resistance conferred by low bark N levels when forests are subjected to elevated N deposition.

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