

BEECH BARK DISEASE

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INTRODUCTION. In forests of North America the beech bark disease (BBD) complex affects American beech, *Fagus grandifolia* Ehrh. BBD begins when bark tissues, attacked by the exotic beech scale insect, *Cryptococcus fagisuga* Lind. are rendered susceptible to killing attacks by fungi of the genus *Nectria* (Ehrlich 1934). The principal fungus, *N. coccinea* var. *faginata* Lohm. and Watson (Lohman and Watson 1943), was probably introduced also, but the native pathogen, *N. galligena* Bres., also attacks and kills bark predisposed by *C. fagisuga* (Cotter 1974; Houston 1994a; Mielke et al. 1982). The general framework for BBD's etiology:

BEECH TREES + *C. FAGISUGA* + *NECTRIA* SPP. => BBD

indicates the chronology of events required for disease development, and points out that although the effects of the insect are necessary, the disease is expressed only after *Nectria* spp. attack and kill scale-altered tissues.

Following its accidental introduction to Nova Scotia around 1890 (Ehrlich 1934), the beech scale spread westward and southward through forests of Canada and the United States. It now occurs throughout New England, New York, much of New Jersey and Pennsylvania, and is present in northeastern Ohio, northeastern West Virginia and northwestern Virginia (Fig. 1). Its recent discovery in the Great Smoky Mountain National Park along the Tennessee-North Carolina border (Houston 1994a,b) has prompted considerable concern.

Course of the disease and its effects: Generally, *Nectria* infections and tree mortality (Fig. 2) occur 1 to 4 years after heavy build-up of the insect on large trees (Fig. 3). The area of current heavy mortality is termed the killing front; regions where severe mortality occurred earlier comprise the aftermath zone (Shigo 1972). In aftermath forests the causal agents are established on small trees of both root sprout and seedling origin that often develop after death or harvest of their progenitors. Most of the new emerging trees and old survivors become cankered and rendered highly defective by the scale-*Nectria* complex (Fig. 4).

Thus, in North America, where the introduced causal complex is still advancing, there are two distinct phases of the disease. The first phase encompasses the effects resulting from the invasions and epidemic buildups of scale and pathogens (killing front); the second phase encompasses the effects of the established causal complex on the survivors and the young, small beech trees emerging in the aftermath of heavy tree mortality or salvage.

In phase one, scale populations build rapidly to high levels. Even though heavy infestations can reduce growth, and, by killing cells in the outer layers of the phellogen, cause bark fissuring, the

insect alone rarely damages the cambium (Lonsdale and Wainhouse 1987). Usually, however, infection of bark by one or both *Nectria* pathogens soon follows infestation. Bark exudation (“tarry spots”), which can result from many other causes as well, is often the first sign that bark has been killed by *Nectria*. Massive invasion by the pathogens of scale-infested trees usually ensues; often, more than 50 percent of the beech trees ≥ 10 inches in diameter are killed, and many more are severely damaged. Such losses can be significant. For example, as of 1977 the estimated loss in merchantable timber volume attributed to BBD in Vermont had reached nearly 300 million board feet (including trees dead, dying, or damaged beyond use) (Miller-Weeks 1983). Phase one is now occurring in eastern Pennsylvania and northeastern West Virginia and, no doubt, will occur soon in the recently affected stands in Ohio, Virginia, North Carolina, and Tennessee.

Opening up of stands by mortality or by salvage of diseased trees can lead to the development of dense stands from root sprouts and seedlings and has helped to create, over large areas, stands that are overly rich to beech and impoverished in associated species. Development of these stands ushers in phase two (Houston 1975). With time, the stems in these stands gradually acquire spatial habitats for the beech scale. Infestation of these habitats, which include bark figures and fissures, callused areas around injuries and patches of protective lichens and mosses results in scattered, isolated colonies. Bark beneath the colonies, altered by the insect’s feeding activity, may be attacked by *Nectria* spp. resulting in scattered, discreet cankers. The callus that develops around each canker provides additional refuges for scale. Aggregations of cankers develop over time, and trees become increasingly defective, but rarely are they girdled and killed quickly as were their progenitors in phase one.

Eventually, in long-affected stands, severely affected trees lose vigor, grow slowly, and then die, out-lived by more vigorous, less severely diseased and resistant beech trees and trees of other species. In any particular forest the rate and pattern of this shift depend in large part on the relative density and frequency of the beech component, on the distribution patterns of resistant trees, and on management intervention. Thus, harvest operations, even in badly diseased and slowly declining beech stands can initiate once more, the formation of highly susceptible thicket stands.

Some Factors that Influence Disease Development

Stand composition and structure – Stand age and density, tree size, and species composition affect disease severity, especially in forests affected for the first time. Older stands with a high component of large beech trees are most vulnerable, and in such stands tree mortality can be very high (e.g., Valentine 1983). Large old trees with extensive decay, conks, or large broken branches are most at risk and often die rapidly as *Nectria* spp. becomes established (Mize and Lea 1979). One study of forests in Massachusetts and New Hampshire showed that stands rich in hemlock were especially vulnerable (Twery and Patterson 1984).

Environmental factors – Once established in a forest stand, scale populations tend to fluctuate at different rates and amplitudes related, in part, to the temporal phase of the outbreak. I monitored the general changes in annual populations of *C. fagisuga* on trees in plots from Maine to West Virginia (D.R. Houston, unpublished data). In newly infested stands, scale populations built up rapidly to high levels, whereas in long-affected stands, established populations typically were maintained at lower levels and exhibited less dramatic annual fluctuations (Fig. 5), presumably in response to local

climate change. Exceptionally cold winter temperatures and heavy autumn rainfalls, both highly correlated with low levels of BBD development, presumably adversely affected overwintering scale populations and the establishment of new populations in late autumn, respectively (Houston and Valentine 1988).

Predators and parasites – No invertebrate parasites of *C. fagisuga* are known. However, several predators are recognized of which the twice-stabbed ladybird beetle *Chilocorus stigma* Say is the most common. *C. stigma* is most abundant when scale populations are dense and, although it responds numerically to high scale densities, its predatory effectiveness is limited by its propensity to disperse, its failure to feed on all life stages of scale, and, especially, by the high rate of scale reproduction (Mayer and Allen 1983). Although scale populations on individual trees can be markedly reduced when populations of coccinellids are high, the overall effectiveness of these predators is limited.

Bark epiphytes – Some epiphytes growing on beech bark offer favorable spatial habitats for *C. fagisuga* (Ehrlich 1934, Houston et al. 1979). Colonies often develop initially beneath patches of moss and lichen. However, not all epiphytes enhance infestations. For example, in Nova Scotia, some stands on steep, south-facing slopes contain beech trees that are remarkably free of disease compared to others in the general area. These trees are heavily colonized by mosaics of crustose lichens, the predominant species of which are rarely colonized by *C. fagisuga* (Houston 1983b). Such preclusive lichens have thalli that are dense, smooth and epigenous in contrast to the loosely compact, granular-surfaced hypogenous thalli of readily colonized species.

Host resistance – In affected stands, some trees remain free of beech scale and disease (Fig. 6). Challenge trials have shown them to be resistant to *C. fagisuga* (Houston 1982, 1983a). Resistant trees occur in relatively low numbers (< 1.0 percent of the beech stems) and many occur in groups (Houston 1983a). The occurrence of resistant trees in groups is encouraging; groups of resistant trees are easier to recognize than isolated individuals, and potentially are easier to protect in forest management operations designed to discriminate against diseased trees. Isozyme genetic studies have shown that resistant trees in groups originate both from root sprouts (clones) and from seed (families) (Houston and Houston 1986, 1990).

Isozyme patterns unique to resistant trees have not been found (Houston and Houston 1994), and the control of resistance is probably multigenic. Resistant and susceptible trees differ in their bark chemistry. Bark of resistant beech has significantly lower concentrations of some amino acids and total amino nitrogen than does uninfested bark of susceptible trees (Wargo 1988).

Management/Control

Options available to reduce the effects of BBD are determined by the temporal phase of the disease, stand structure and composition, and the harvesting and silvicultural systems available (Mielke et al. 1986, Ostrofsky and Houston 1989). There are two major management situations or problems posed by the disease. The first is how to deal with forests that are about to become, or that have recently been, infested for the first time, and where heavy beech mortality can be expected within a few years. The second problem is how to handle aftermath stands where dense, disease susceptible, and defective stands have developed. In brief, managing recently-infested stands entails a) reducing

the proportion of beech, b) discriminating against large, overmature trees with roughened bark and signs of decay, c) removing heavily infested trees, and d) removing advance beech regeneration with herbicides where overstory beech is heavily infested (Mielke et al. 1986).

In killing front and aftermath forests, Mielke et al. (1986) propose that a) dead or declining trees with heavy beech scale populations be removed, and b) susceptible advance regeneration and understory beech be treated with herbicides. They recommend leaving beech with little or no scale or *Nectria* infection. Ostrofsky and Houston (1989) suggest using harvesting systems that by minimizing injuries to beech root systems, should reduce development of root sprouts from roots of susceptible trees.

It is clear that close surveillance of stand conditions and scale populations is important during all stages of disease development. In early stages of an outbreak, scale-free trees may not be resistant but merely "escapes." But, as the outbreak ensues, large trees that remain free of scale, or only very lightly infested, are good candidates for retention in the stand. Infested trees do vary in their levels of resistance to scale and possibly to *Nectria*. As a consequence, diseased trees persisting in aftermath forests may differ in how seriously they are damaged, e.g., on some trees what appear to be severe bark injuries, actually may be restricted to the outer bark leaving the cambium and wood unaffected (Burns and Houston 1987).

Increasing the relative number of resistant trees seems to be the most promising approach for reducing the impact of this disease in the long run. The results of trials to determine how various harvesting regimes affect the initiation, development, and survival of root sprouts are being analyzed. In addition, studies to determine ways to clone selected resistant genotypes have been conducted. Tissue-culture techniques which use sprouts from root segments and forced buds of mature resistant trees, have brought several genotypes through to rooting (Barker et al. 1995). Still needed are trials to develop ways to grow the tissue culture plantlets in soil and introduce them into the forest.

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Fig. 1. – Known distribution of beech scale (block areas) as of 1996, in relation to the range of American beech (hatched areas).

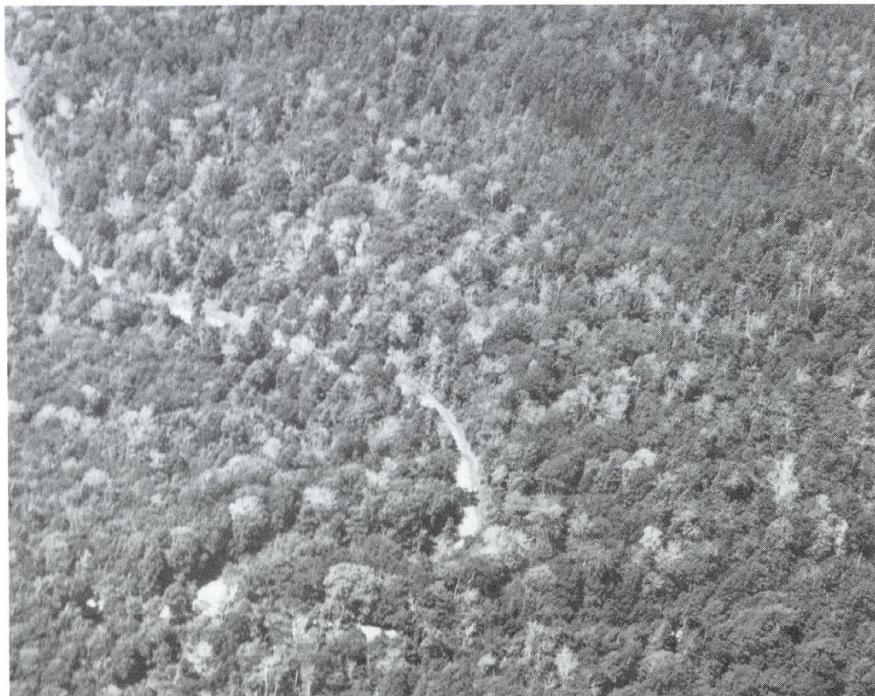


Fig. 2. – High tree mortality can occur when forests are affected by the causal complex for the first time. Over 75% of the large beech in this Vt. forest were dead or dying (bare and gray crowns) in 1971.



Fig. 3. – Heavy infestations of beech scale can cover tree boles with white wax.

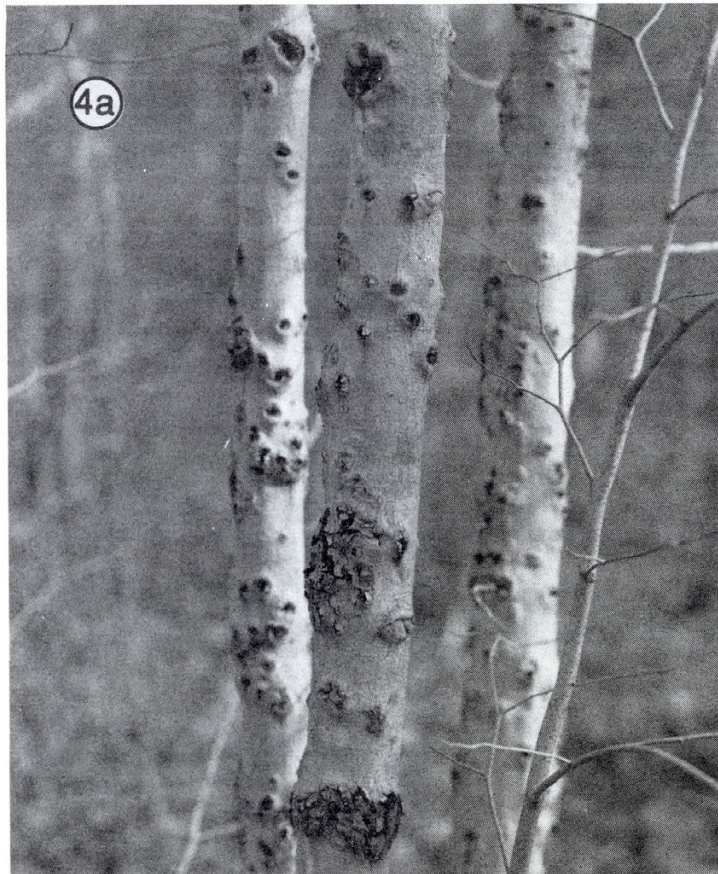
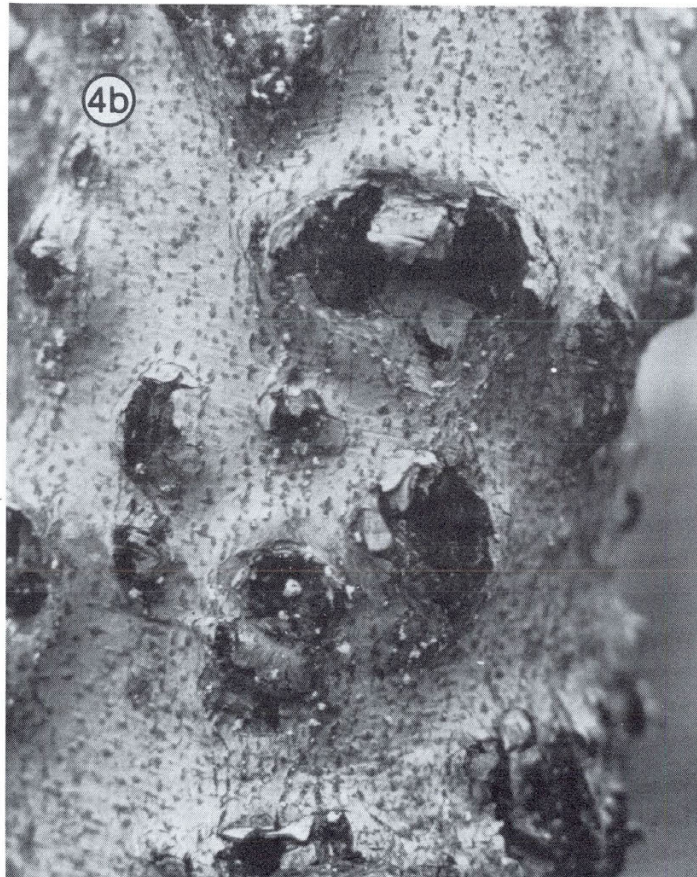


Fig. 4. – Trees in aftermath forests can become severely defective. Initially, cankers are scattered and discrete (a);



...with time cankers provide habitat for scale and new cankers develop and accumulate (b);



...extreme defect sometimes results (c).

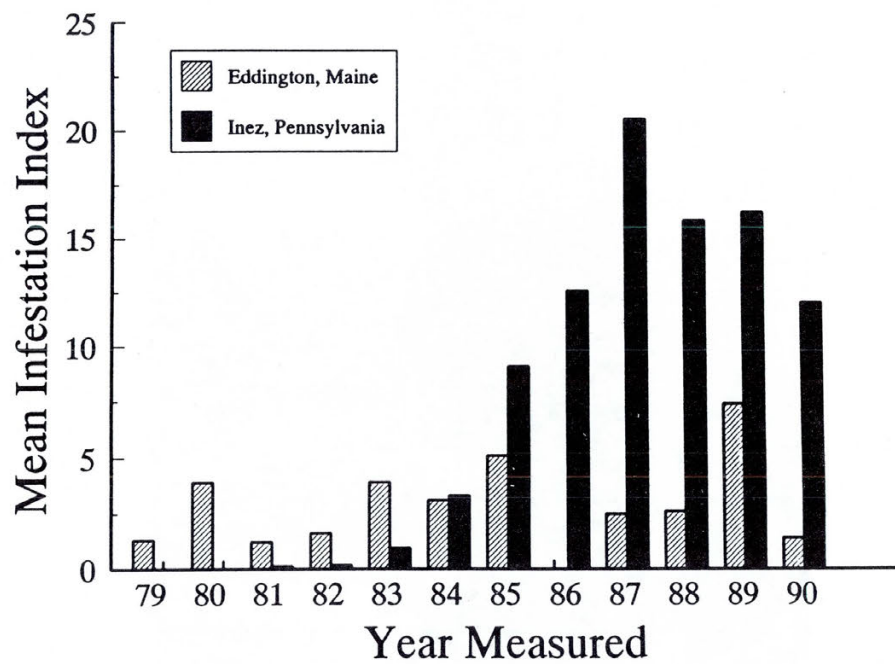


Fig. 5. — Annual population levels of *C. fagisuga* from 1979 to 1990 in the first decade of infestation (Inez, Pennsylvania) and in the sixth decade after initial infestation (Eddington, Maine). The infestation index was calculated as a weighted average of infestation scores for approximately 200 trees per plot. Trace populations were scored as 1, very heavy as 40 (Houston 1994a).



Fig. 6. – A few trees are resistant to beech scale and remain free of disease (center) in contrast to their susceptible neighbors.