

Keynote Address

BEECH BARK DISEASE: 1934 TO 2004: WHAT'S NEW SINCE EHRLICH?¹

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Some time in the mid-to-late 1800's, a ship from England arrived at the busy Canadian port of Halifax, N.S. On board was a consignment of plant material, including European beech (*Fagus sylvatica* L.) saplings, destined for the city's Public Gardens. These trees prospered in this northern city whose latitude and maritime climate were similar to those of England. Around 1890, some of the trees were found to be infested with the "felted beech coccus", *Cryptococcus fagi* Baerensprung.

Some 30 years later, large numbers of American beech (*F. grandifolia* Ehrl.) trees in forests around Halifax began dying of unknown cause, and in the late 1920's, John Ehrlich, a Canadian graduate student at Harvard, began a PhD study of the cause and consequence of the emerging problem. His work, published in 1934, (Ehrlich 1934) named the disease, described the causal insect/fungus complex, and laid the groundwork for nearly everything that has been learned since. Indeed, his research was so comprehensive that aside from the establishment of plots to monitor the disease, trials to control it using chemicals, and silvicultural attempts to slow its course or lessen its impact in forests, few critical examinations of or additions to his understanding of the disease system occurred in North America until the 1960's.

Ehrlich determined that the disease was caused by a complex of the felted beech coccus, (soon after named the "beech scale"), and a bark killing fungus of the genus *Nectria*. He provided experimental evidence that feeding by the scale predisposed bark to infection by the fungus. He did not name the fungus, but determined that it was closely related to *Nectria coccinea*. Ehrlich's framework for this complex can be stated as:

Beech + Beech scale + *Nectria* sp. = Beech bark disease

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So, at this conference on beech bark disease (BBD), convened approximately 120 years after the scale arrived on this continent, it seems appropriate to review the information that has been added to our understanding of this complex pathosystem in the 70 years since publication of Ehrlich's classic paper.

The Forest/disease Relationship

Ehrlich's study focused on how disease incidence and severity were affected by such forest stand values as beech abundance, basal area, position on slope, degree of slope, aspect, and length of time affected, and by such tree traits as DBH, crown class, and the presence of stem mosses and lichens. In the Canadian forests he studied, overall 54% of the stems were beech; nearly all trees in diseased stands were infested and 89.6% (of 4,483 trees over 3 inches dbh) were infected by the fungus. He found that degree of infection was a more reliable indicator of disease severity than scale infestation because evidence of infection persisted while the presence of wax was ephemeral. The best index of disease effect was mortality at time of observation, and mortality was strongly correlated with length of time of infestation/infection. Thus, while mortality can begin soon after infection begins, it may extend over a period of years: ca 20% in 4 years, 50% in 10 years (climate and other differences being equal).

Definite positive correlations were found between percent beech with *Nectria* infection and percent beech in the stand, and with position on ridge and steepness of slope. Severity of infection was correlated with tree size; mortality (% of trees killed over 3 inches) with diameter, crown class and position on ridge (bigger trees were more severely infested, infected and killed; trees growing on steep slopes and near tops of ridges were more heavily infected and killed).

Ehrlich's research arena was constrained both spatially and temporally by the geographic extent of the then newly emerging epidemic. In the ensuing 70 years the disease complex has continued to affect the forests he studied, and also has spread to forests differing in climate, species composition and structure, and use history. However, its relatively slow rate of spread (compared to other exotic diseases such as chestnut blight and Dutch elm disease) has provided opportunities to

observe both long and short-term forest effects and responses. Of interest today are what long-term impacts on stand composition and structure have occurred, whether silvicultural treatments designed to minimize impacts have been effective, and if differences exist in either the course of the disease or its effects in forests to the west and south.

Dynamic changes in beech abundance have occurred throughout post-glacial time (Cogbill, these Proceedings). Consequently, it may be important when evaluating current disease impacts, to do so against the backdrop of pre-BBD fluxes in beech abundance and to consider the influence of other stand disturbance factors, past and present. The papers by Cogbill, Canham, and Latty, (these Proceedings) describe early beech populations in different geographic regions, and emphasize the need to understand how other stressors might augment the effects of the BBD causal agents.

Although the consequences of BBD are still developing, marked changes have occurred in forests long-affected. Shigo (1972) characterized three arbitrary temporal stages of the disease. The first two, the “advancing front”: areas recently invaded by beech scale where many large old trees support sparse populations, and the “killing front”: areas where high scale populations and severe *Nectria* infections are resulting in heavy tree mortality, would have been most familiar to Ehrlich. Less familiar to him, however, would have been the “aftermath zone”: areas where tree mortality has occurred in the past and which now possess a few large residual trees, and many stands of small trees—often of both seedling and root sprout origin (Shigo 1972; Houston 1975). As the trees in these stands develop in the presence of the causal complex, they often are rendered highly defective through the interactions of beech scale, *Nectria* fungi and, sometimes, another scale insect, *Xylococcus betulae* (Perg.) Morrison (Shigo 1962). Widespread mortality due to BBD is rare in the aftermath zone (Houston 1975). The aftermath zone is well developed in most of the beech forests of the Maritime Provinces and ME, in some forests of VT, NH, NY, and is developing in a few of the longest-affected forests of PA and WVA.

In terms of forest response and forest management considerations, the advancing and killing front temporal stages, which encompass the initial establishment and buildup of the causal complex and resultant high tree

mortality, comprise the “first phase” of the disease; the aftermath stage, where the now endemic populations of the causal organism complex cause accumulating defect in young, emerging stands, the “second phase” (Houston 1994a). A third temporal phase is now occurring in some longest-affected stands, especially those not subjected to recent harvesting disturbance. Here, severely affected trees, often in patches, lose vigor, slow in growth, and eventually succumb, survived by beech that are either less severely diseased or resistant, and by other species. Harvest operations in such stands can trigger anew the development of highly susceptible thicket stands (Houston 1975, 1994a).

From the foregoing, it is apparent that the options for managing a particular disease-affected beech forest will depend on the temporal phase of the disease. Dealing with current or impending high mortality in Phase I situations is little different now than in Ehrlich’s day, but managing the long-affected, highly defective, beech-dense aftermath stands has, in the interim, become ever more difficult as trees have become increasingly defective and slow-growing. Management approaches and harvesting systems have been proposed (e.g., Crosby and Bjorkbom 1958; Filip 1978; Mielke et al. 1986; Ostrofsky and McCormack 1986; Burns and Houston 1987; Ostrofsky and Houston 1989). Most of these approaches embrace the ultimate objective of increasing the proportion of disease resistant beech. In practice this has proven difficult, because American beech possesses a unique suite of ecological characteristics that renders it difficult to manipulate silviculturally. Together, these features (extreme shade tolerance, longevity, prolific seed production and aggressive root sprouting) insure that once established, beech as a species will survive and even increase with stand disturbance, and that its genes, including those for traits such as susceptibility, will be perpetuated.

The inability to effectively manage beech comprises a major stumbling block to managing the disease. Several papers and working group sessions at this symposium (these Proceedings) address the need for research on this problem. A number of studies have examined the relationship of forest stand composition and structure to BBD. In British beech plantations, trees 25 to 45 years old, growing on midslope, or downwind of old, large infested trees, often were the first affected (Parker 1975; Houston et al. 1979a, b). In North America, probability

of mortality was greatest among big trees of low vigor (Mize and Lea 1979). In Massachusetts and in New Hampshire, Twery (1983, 1984) found that mortality and defect attributable to BBD were significantly greater in stands dominated by hemlock (*Tsuga canadensis* (L.) Carr.). While BBD mortality resulted in a decrease in beech and yellow birch (*Betula alleghaniensis* Britton) and an increase in hemlock, understory composition (and consequently, that of the future forest) was little changed. In New York, harvesting of diseased or healthy beech often led to dense, nearly pure beech stands (Tierson et al. 1966). Studies now underway in the outbreak areas in Michigan (Storer et al. and Witter and Petrillo these Proceedings) and in the Great Smoky Mountains National Park promise to add to our understanding of disease effects in forests that may differ in origin and composition.

In aftermath forests in Maine, thickets largely of root sprout origin developed after death or salvage of mature overstory beech (Houston 1975). Root sprouting in beech results when roots are wounded by disturbances such as frost heaving, browsing, logging, exposure to light and higher temperatures (Maini and Horton 1966; Held 1983; Jones and Raynal 1986, 1988). Sprouts arise from adventitious buds that originate within callus tissues associated with wounds (Jones and Raynal 1986). Jones and Raynal (1987, 1988) found that fewer sprouts occurred around isolated low-vigor trees compared to high-vigor trees, and on roots that were wounded in the fall compared to those wounded in the spring. Houston (2001) monitored the initiation and development of root sprouts and the development of seedlings around (a) resistant and susceptible beech trees that were (b) cut or left standing in stands (c) either clearcut or partially cut in (d) winter or summer of 1991. By 1994, significantly more sprouts occurred around the resistant trees. Interactions occurred between the amount of root disturbance (triggering root sprouts) and the amount of light available for growth. Thus, more sprouts developed after clearcuts than after thinnings, and sprout growth was best around resistant trees left standing and poorest around susceptible trees that were cut. Summer clearcuts sharply reduced the number of existing seedlings and prevented “new recruits”. Seedling growth was related primarily to the amount of light received as a consequence of the harvests or disease-caused crown thinning.

The impact of BBD on the growth and survival of beech has been examined in both Europe and North America. In young (age 20–45 years) or old (age 100+ yrs) European beech plantations, BBD-caused mortality was highest in stands where trees were stressed by competition (Peace 1954; Parker 1980, 1983). In England and in France, the disease was most severe in dense, pure stands and during times of water shortage, especially on sites with excessively drained soils and on thin soils over chalk (Peace 1954; Lonsdale 1980a; Perrin 1983; Parker 1983). In Europe and in North America, thinning of stands only temporarily reduced scale populations and the course of the disease (Parker 1980, 1983; Perrin 1983; Crosby and Bjorkbom 1958).

In North America, several studies have documented the effects of the disease on growth of beech stands and individual trees. Runkle (1990) found a 25% reduction in beech basal area (6.7% annually) over an eight year period in aftermath stands in the Adirondacks. Surviving trees, whose growth rates about equaled that of other species and was greatest in stands with highest tree mortality, seemed to benefit from the disease-caused reduction in competition. Mize and Lea (1979) used remeasurement data (1954 to 1976) to gauge decline and mortality of individual beech trees in an Adirondack forest. Mortality beginning in 1967 was mainly of trees of large diameter and crown class. Measured trees declined in diameter growth by 26% from predisease to aftermath periods. Gavin and Peart (1993) compared the effects of disease on trees in old growth and second-growth stands in New Hampshire. In both stands significant growth declines occurred that were related to severity of infection and, for the second-growth stand, with levels of internal stem defect. Significant differences in growth between undiseased and severely infected trees occurred earlier in the old-growth stand. Gove and Houston (1996) used increment core data to compare the growth of matched pairs of susceptible and resistant trees of different crown classes in two locations in Maine. In the more southerly site (88-year series: 1900 to 1987), susceptible codominant trees grew significantly better than resistant ones until the late 1930's. Decline in growth of susceptible codominant trees became significant in 1941, while it didn't occur in intermediate crown class trees until 1975 (35 years later). In the more northerly site (80-year series: 1909 to 1990) there was only a six year difference in decline onset between the two crown classes. Growth patterns were similar to those

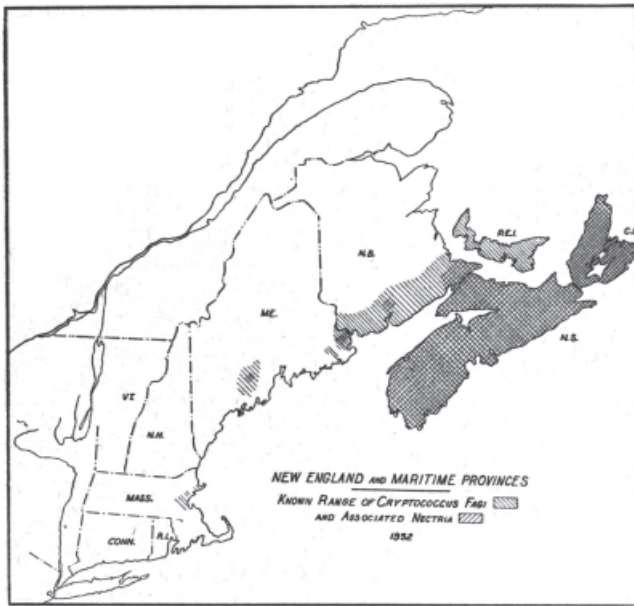


Figure 1.—The distribution of beech scale and *Nectria* as of 1932 (Ehrlich 1934).

in the southern site, with susceptible trees outgrowing their resistant counterparts until they began to decline in 1961 (21 years after onset of decline on the southern site). Differences in timing of decline onset and between the two crown classes probably reflected differences in climate, stand dynamics, and arrival time of the causal agents.

The importance of beech mast for wildlife especially for black bear (*Ursus americanus*) has been well documented (e.g., Beeman and Pelton 1980; Hugie 1982; Costello 1992; Schooley et al. 1994a, 1994b; Jakubas, these Proceedings). However, relatively little research has been done to determine the impacts of BBD on mast production. Costello (1992) monitored seed production on trees in different stages of disease and found that it dropped significantly only after trees became infected by *Nectria* sp. and had lost more than 25% of their crown. Very few beech nuts were produced by infected trees with crowns more than 25% dead. Using stand data from 1948, Costello calculated that by 1989, beech nut production decreased by 37%. The potential impacts of the disease on wildlife are discussed by Storer et al. (these Proceedings).

The Causal Complex:

1) The Beech Scale Predisposer

Ehrlich (1932, 1934) described the insect in North America, and gave the historical account of its discovery

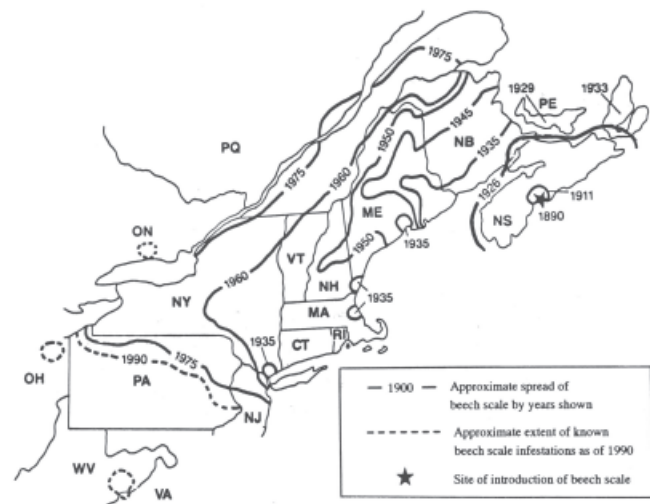


Figure 2.—Isopleths of years of discovery of the beech scale from 1890 to 1990 (Houston 1994a).

and description in Europe. Its distribution in North America as of 1932 was limited to the Maritime Provinces, and a few locations in Maine and Massachusetts (Fig. 1). He described the insect's seasonal development patterns, and factors that influence its colonization of individual trees (bark fissures, figures, mosses, lichens, aspect, position within the stand), and he speculated that its buildup could be influenced adversely by cold temperatures and by biocontrol agents, especially the coccinellid beetle, *Chilocorus bivulnerus* Muls.

Ehrlich described the nature and physical effects of the insect's feeding and how the drying and cracking of bark near killed cells allowed penetration and invasion by fungi otherwise unable to successfully infect intact beech bark.

Distribution

In the past 70 years, *C. fagisuga* Lindinger (as the scale is now named) has inexorably continued to spread west and south. Ehrlich's (1934) account of the insect's probable introduction to the Boston area around 1919, and records of its establishment near New York City by 1934, suggest that perhaps several separate introductions have occurred. Isopleths of discovery dates (Fig. 2) provide an approximation of the temporal—spatial movement. Imbedded in these now seemingly solid fronts are stands that earlier were outliers of infestation. The current

outbreaks in OH, MI, and NC/TN are examples, as once were those at Heart's Content Recreational Area on the Allegheny National Forest in PA and the Gaudineer Scenic Area on the Monongahela National Forest in WV.

Dispersal of the insect has been studied. Eggs, and especially crawlers, are transported passively in the airstream (Wainhouse 1980). The crawler's flattened shape reduces its terminal velocity facilitating its wind dispersal along a short-range steep gradient within stands. A small proportion of the population is wafted upward and is dispersed along a potentially long-range shallow gradient above the canopy (Wainhouse 1980). Infestation patterns within English beech plantations reflected both of these transport modes (Houston et al. 1979a). There is little doubt that the insect is also moved from place to place within stands by insects, mammals, and birds, and probably between stands and regions by birds and people. (See also, papers these Proceedings by Liebhold et al. that used kriging of forest inventory plot data to map the spread of BBD within the United States, and by Gardner, that used forest inventory plot data and range maps to model scale dispersal within a forest preserve in Eastern NY.)

Successful colonization depends on the genetic susceptibility of the trees, the nature and number of favorable spatial niches, and the host/insect/genetic relationship (Wainhouse and Deeble, 1980; Wainhouse and Howell 1983; Houston et al. 1979a). Although parthenogenetic, the scale apparently has some ability to adapt to the host it colonizes (Wainhouse and Howell 1983).

Resistance

It has long been recognized that some beech trees remain either uninfested and free of signs of BBD or continuously support only low populations (Ehrlich 1934; Thomsen et al. 1949; Shigo 1962, 1964; Wainhouse and Howell 1983; Houston 1983a; Houston and Houston 1987; Houston et al. these Proceedings). European beech that can support only low populations possess bark anatomical features that act as barriers to infestation (layers of difficult-to-penetrate sclerophyll cells that are thicker, more continuous, and nearer to the bark surface than in susceptible trees) (Lonsdale 1983a). Although comparable studies have not been done in North America, similar anatomical barriers to infestation probably occur also in American beech. Resistant

American beech possesses bark that contains significantly less total and amino nitrogen than that of susceptible trees (Wargo 1988). Low amino nitrogen concentration is known to limit establishment and growth of sucking insects (Dadd and Mittler 1965).

The proportion of scale-free trees in American beech stands is very low, usually around 1% or less (e.g., Houston 1983a). In recent years studies have been made of the distribution patterns of such putatively resistant trees and of their genetic relationships. "Clean" trees are especially evident in some aftermath forests where their smooth, uncantered boles stand in sharp contrast to the highly defective stems of their susceptible neighbors. Resistant trees can occur as single individuals, but often they are in groups (Houston 1983). Challenge trials have shown them to be resistant to beech scale attack (Houston, 1982, 1983), a consequence, perhaps, of their significantly lower bark nitrogen content (Wargo 1988).

Higher bark nitrogen content of old-growth forest trees, (presumably the result of nitrogen saturation) compared to that of second-growth forest trees, was correlated with higher disease severity in stands in New York (Latty et al. 2003; Latty these Proceedings).

Isozyme analyses revealed that within groups of resistant trees, some individuals are genetically identical (i.e., clonal, derived from root sprouts), while others have unique isozyme "genotypes" (derived from seed) (Houston and Houston 1987). Unique trees within groups appear closely related and probably are half- or full-sib families (Houston and Houston 1987). Spatial patterns of the unique individuals within groups (Houston and Houston 1987) appear to reflect the beech nut caching patterns of eastern blue jays, *Cyanocitta cristata* (Johnson and Adkisson 1985; Johnson et al. 1987). Such patterns were often clearly displayed in the easily discerned groups of resistant trees in many aftermath forests (Houston 1983a; Houston and Houston 1987). The use of isozyme analysis was extended to compare population genetic structure in beech stands in P.E.I., N.S., ME, MA, and WVA where groups of resistant trees had been identified. Analyses showed that both susceptible and resistant beech trees in these stands were substructured into mosaics of putative clones and trees of seedling origin (Houston and Houston 1994). Although no unique genotypes for identifying resistant trees occurred among the 17 enzymes (with 9

polymorphic and 14 monomorphic loci) analyzed, resistant tree populations exhibited consistent heterozygote deficiencies (Houston and Houston 2000).

Genetic markers for resistance would greatly enhance efforts to determine modes of inheritance and, perhaps, to identify resistant forest trees in advance of the disease. The search for such markers using molecular level approaches is now underway, as are also trials to clarify modes of inheritance via cross-breeding resistant and susceptible individuals (see papers by Koch and Carey, these Proceedings).

One strategy to raise the proportion of resistant genotypes within a given stand includes augmentation using rooted cuttings or plantlets derived from resistant trees. Vegetative propagation of beech, as with other members of the Fagaceae, has proven difficult. Indeed, even though micropropagation techniques to derive plantlets from beech bud tissues have been developed (Barker et al. 1997), and some rooted cuttings have been obtained from root and stem tissues (Loo et al. these Proceedings), rooted beech plantlets have not been successfully overwintered. This problem remains a major hurdle for programs attempting to restore high quality American beech.

Biocontrol

No insect parasites of *C. fagisuga* are known in Europe or North America despite repeated searches, including recent ones in Asia (Wainhouse and Gate 1988; R. Reardon, pers. comm.). A number of predators are known. The most common is the twice-stabbed lady beetle, *Chilocorus stigma* Say, (identified by Ehrlich as *C. bivulnerus*). Although both larvae and adults of this native coccinellid beetle prey on the sedentary life forms of *C. fagisuga*, its effectiveness is limited by its propensity to disperse following eclosion, its apparent failure to feed on the scale's crawler stage, pupal mortality, and high rates of host reproduction (Mayer and Allen 1983). Other coccinellids (*Exochomus* spp.) and a cecidomyid fly (*Lestidiplosis* sp.) are also common, and all are usually confined to trees with moderate to heavy scale populations (Wainhouse and Gate 1988). While predators have been shown effective in reducing scale populations on individual trees, their influence on the course of the disease is of little consequence.

The entomophagous fungus, *Verticillium lecanii* Viegas, was found to be associated with colonies of *C. fagisuga* in Great Britain (Lonsdale 1983b). Studies suggest that the spread of the fungus between insects on individual trees may depend on scale colonies increasing to levels where they coalesce. Observations and isolations suggest that the fungus, after reducing heavy scale populations, persists within the resultant isolated colonies and effectively maintains them at low levels (Lonsdale 1983).

Bark epiphytes including mosses and foliose lichens provide favorable habitats for establishment of *C. fagisuga* (Ehrlich 1934; Houston et al. 1979a). Not all lichens are favorable, however. On some steep, south-facing slopes in Nova Scotia, the stems of beech trees remain remarkably free of beech scale and of defect caused by BBD compared to trees on other nearby sites. These 'clean' trees were heavily colonized by dense mosaics of compact crustose lichens—the majority of which have thalli that are thick, elevated above bark surfaces (epigenous), and have smooth surfaces that provide little spatial habitat for *C. fagisuga* (Houston 1983b). Conditions which favor development of these 'preclusive' lichen mosaics may include the relatively low level of lichen-damaging air pollution, and slow tree diameter growth, attributable to reduced moisture availability on the steep slopes, that may allow growth of lichen thalli to maintain complete coverage of the lower stems.

The bark-inhabiting fungal parasite, *Ascodichaena rugosa* Butin, attacks both *F. sylvatica* and *F. grandifolia* (Butin 1977). Studies in Great Britain showed that patches of *A. rugosa* stroma on beech trees supported far fewer colonies of beech scale than did stroma-free bark (Houston et al. 1979). Butin and Parameswaran (1980) studied the ultrastructure of *A. rugosa* and its effects on tree bark. Invasion by the fungus triggered cork cambium to produce thicker layers of cork periderm. Speer and Butin (1980) found that stylets of *C. fagisuga* could penetrate through thickened periderm to the bark parenchyma below, and except for cases where secondary periderms were formed, actually favored the insect. Stromatic patches of *A. rugosa* on *F. grandifolia* are less frequent, less continuous and less compact than on *F. sylvatica*, and the stroma, often thinned and fractured as stems increase in diameter, appears to constitute a favorable spatial niche for the scale.

The Causal Complex: 2) The *Nectria* Pathogens

Ehrlich recognized that the fungus associated with BBD belonged to the *N. coccinea* group, but was different enough from those known from North America and Europe to be considered a variety. Lohman and Watson (1943) named it *Nectria coccinea* var. *faginata* Lohman, Watson and Ayers, and it was recently renamed *Neonectria coccinea* var. *faginata* (Rossman et al. 1999). (To avoid confusion in this paper, *Nectria* is used to designate the genus.) Studies of its population genetics revealed it to be more closely related to the *N. coccinea* spp. of Europe than of North America, and strongly suggest that it was introduced, probably about the same time as was the scale (Mahoney, et al. 2001). It is likely that isolates of *N. coccinea* var. *faginata* (Ncf) vary in pathogenicity as do those of *N. coccinea* var. *coccinea* (Pers.) Fries (Ncc) in Europe (Perrin 1979; Lonsdale 1980b). In addition, *Nectria galligena* Bres. (Ng), which only rarely infects healthy beech, was shown to readily infect beech scale-infested bark (Spaulding et al. 1936; Cotter and Blanchard 1981). In forests where beech scale is introduced ahead of Ncf, inoculum of Ng from non-beech hosts, if present, can infect scale-infested beech trees and cause “full blown” BBD (Mielke, et al 1982; Houston 1994a). When Ncf arrives on the scene, it eventually replaces Ng and becomes the dominant pathogen (Fig. 3) (Houston 1994b).

Another *Nectria* species, *Nectria ochroleuca* (Schwein.) Berk. (now named *Bionectria ochroleuca* (Schwein.) Schroers and Samuels (Rossman and Samuels 1999), was found associated with dead trees and dying, scale-infested trees in several stands in WV, PA, and in woodlots near Toronto, Ontario (Houston and Mahoney 1987). While sometimes found together with Ng, it was often the only *Nectria* present on dying trees. LaMadeleine (1973) frequently isolated the anamorph of this fungus, *Gliocladium roseum* (now named *Clonostachys rosea*) from the bark of chlorotic trees in the initial stages of the disease. Indeed, *G. roseum* was the most common fungus isolated from the few trees he sampled from stands in PA and NY where the killing front was in its early stages. The role of the fungus is unclear, as it was significantly less pathogenic than either Ncf or Ng when inoculated into dormant, scale-free beech logs. Further study is needed to determine if this *Nectria* species can successfully invade bark predisposed by scale attack (Houston and Mahoney 1987).

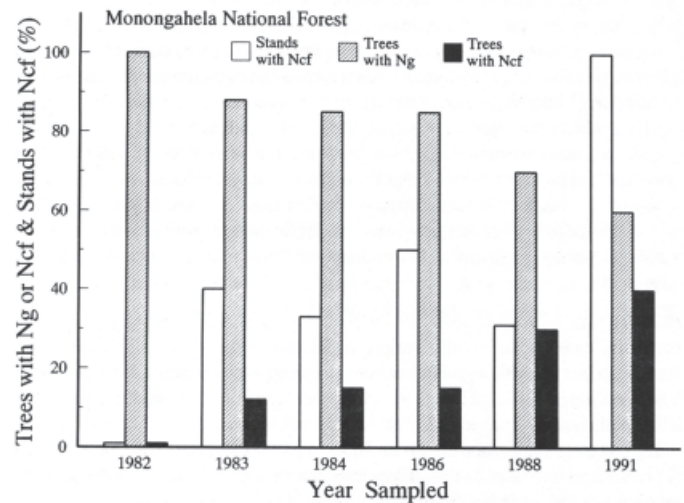


Figure 3.—The shift in percentage of stands and trees on the Monongahela National Forest, West Virginia, that yielded Ng or Ncf from 1982 to 1991 (Houston 1994a, b).

Many *Nectria* species and several other fungi are infected by the biotrophic contact mycoparasite, *Nematogonium ferrugineum* (Pers.) Hughes (*Gonatorrhodiella highlei* A. L. Smith) (Blyth 1949a,b; Gain and Barnett 1970). In North America, Ncf on beech and Ng on beech and other tree species are hosts (Ayers 1941; Ehrlich 1942; Houston 1983c). While parasitized isolates of Ncf and Ng produce fewer spores (Shigo 1964; Houston 1983c) and both species are rendered markedly less pathogenic (Houston 1983c), the effects on BBD development in individual trees or stands appear to be of little consequence.

The Beech Scale-*Nectria* Relationship

Ehrlich demonstrated through exclusion and removal experiments that infestation immediately prior was required for infection by *Nectria* to occur. In Nova Scotia, infection usually occurred within 1 to 5 years after infestation and, while tree mortality resulting from bark death could occur as soon as 1 year after infection, it sometimes took many years for trees to die.

In Europe, Ehrlich's causal complex hypothesis was supported a decade later in a noteworthy study by Thomsen et al. (1949). In spite of what must have been harrowing times (1939-1943), these authors tracked an apparently unprecedented rise and decline of the beech scale and of BBD in Danish European beech plantations. Of interest here is that the associated *Nectria*, as determined by Wollenweber in Germany on the basis of

ascospore size (as we do today), was Ng. (Today, throughout Europe, the causal fungus is considered to be Ncc.) Slime fluxes, or exudates, “which disclose the disease” were, as today, considered non-specific, i.e., they can result when bark is killed by a variety of abiotic or biotic factors. The role of ambrosia beetles, notably *Xyloterus domesticus* L. and *Hylecoetus demestoides* L, as well as many decay fungi, were well described.

Other, more recent studies have reconfirmed the scale-fungus causal relationship. Perrin (1980) in France, and Lonsdale (1980b) in England, showed that wound inoculation of bark with Ncc caused cankers, the nature and size of which were related to the severity of *C. fagisuga* infestation. However, these studies also showed that infestation by *C. fagisuga* does not simply provide entrance courts for infection as Ehrlich hypothesized, but also lowers resistance to fungal invasion. Heavy scale infestation results in reduced growth and in restricted callus formation that favors rapid and unrestricted canker enlargement. In these inoculation trials, isolates of Ncc varied greatly in their virulence and trees varied in their susceptibility.

Yet, conclusive “proof” of this scale-*Nectria* interaction remains difficult to obtain or demonstrate. The conditions that either allow or limit infection and invasion by weakly pathogenic, facultative organisms may vary widely from place to place and from time to time (e.g., Houston 1992). Because such conditions are often difficult to identify, and impossible to reproduce, verification of the scale-*Nectria* relationship has depended in large part on careful observation of the timing and consequences of the organism association, and on experiments to clarify portions of the relationship. Many ‘variations on the theme’ have resulted—a consequence, perhaps, of studies being made at different times in forests of different ages, origins, disturbance histories, and stages of the disease (e.g., Houston et al. 1979b).

The temporal pattern of canker development (the beech/scale/*Nectria* interaction) was examined in two eastern Maine aftermath forests (Houston and Valentine 1987). Increased rates of cankering occurred over time, and year-to-year fluctuations in canker incidence were synchronous between trees and stands. Cankering was negatively correlated with October rainfall and the number of cold days (< 15 F) in December through March of the previous fall and winter. Low rates of

cankering presumably resulted from the adverse effects of these variables on the establishment and survival of *C. fagisuga* and/or the development of, or infection by, *Nectria* spp. Temperatures of -30 F (-34 C) are known to be lethal to *C. fagisuga* (Barter 1953). In Denmark, declines in scale populations were associated with unusually cold winter temperatures (Thomsen et al. 1949), and in France, low rainfall in October was followed by increases in scale populations the next year (Perrin 1979).

Conclusion

It is clear that much knowledge has been gained in the 70 years since publication of Ehrlich’s paper about the beech scale-*Nectria* spp causal complex, as well as the beech host and the forests in which it grows. Yet, it is apparent also that much more information is needed if BBD is to be controlled at the forest level. The following list contains a few of the most obvious needs; many more items are identified in the papers and discussions that follow in these Proceedings.

The host-causal complex relationship: the beech scale

1. Determine the nature of host resistance to beech scale. Confirm that low bark nitrogen content conveys resistance.
2. Explore the genetic mode of inheritance of resistance.
3. Identify genetic markers of resistance.
4. Continue the search for scale predators, especially in Eastern Europe and Asia.
5. Investigate the role, if any, of the entomophagous fungus, *Verticillium lecanii* in North America.

The host-causal complex relationship: the *Nectria* fungi

6. Clarify the mechanisms by which the beech scale predisposes beech bark to infection by a seemingly congeneric fungal complex.
7. Clarify the role of *Nectria ochroleuca* in BBD, especially in early stages of the disease.

The forest-disease relationship

8. Develop practical silvicultural approaches, with and without herbicides, to effectively manage

beech populations, and especially, to manipulate the initiation of root sprouts.

9. Develop protocols to vegetatively propagate resistant beech genotypes.
10. Develop protocols to introduce and establish resistant beech genotypes in a variety of forest situations.
11. Develop management systems to exploit existing resistant trees.

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