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▶ To cite this version:

D. R. Houston. Beech bark disease: what we do and do not know. Annales des sciences forestières, 1980, 37 (4), pp.269-274. hal-00882219

HAL Id: hal-00882219 https://hal.science/hal-00882219v1

Submitted on 11 May 2020

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Beech bark disease : what we do and do not know

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Over the past 100 years, beech bark disease has spawned innumerable hypotheses of cause and effect and much conjecture about organism interactions. Yet this complex disease has received little substantive research at fundamental levels. The papers presented at this Colloquium are important for they add significantly to our understanding of beech bark disease. This paper presents, in brief and general terms, what we do and do not know about this disease.

What we know — The disease

We do know a great deal about the general « framework » of beech bark disease. We know that it begins when the felted beech coccus or beech scale, *Cryptococcus fagisuga* Lindinger (*C. fagi* Bäerensprung) infests the living bark of *Fagus sylvatica* L. and *F. grandifolia* Ehrh. We know that feeding by *C. fagisuga* results in reduced growth and some bark necrosis. While some of us feel that *C. fagisuga* can cause extensive bark necrosis (Braun, 1977), most of us feel that under normal growth conditions, death of bark attributable to the insect alone is relatively limited (Boodle and Dallimore 1911 ; Ehrlich, 1934 ; Hartig, 1878 ; Parker, 1974 ; Shigo, 1964 ; Thomsen et al., 1949).

We know that heavy feeding by *C. fagisuga* somehow renders bark tissues susceptible to successful colonization by normally very weakly pathogenic fungi, especially *Nectria coccinea* Pers. ex Fr. (Europe) and *N. coccinea* var. *faginata* Lohman, Watson and Ayers (North America). While some believe that these fungi are of relatively minor importance (Braun, 1977), the majority of us believe that they are primarily responsible for bark necrosis, and ultimately for tree mortality (Houston et al., 1979a).

The insect

Over the course of a century we have learned much about *C. fagisuga*. We know its life cycle and some of the ways that its probings affect the anatomy and physiology of beech bark (Braun, 1977; Ehrlich, 1934; Kunkel, 1968; Rhumbler, 1914). We

have cursory knowledge of its distribution and dissemination patterns (Houston et al., 1979b), its predators (especially Chilocorus spp.), but have no information on its parasites (Ehrlich, 1934; Schindler, 1962; Thomsen et al., 1949). We know that bark flora (lichens, mosses, algae) appear to influence the presence of C. fagisuga on individual trees (Ehrlich, 1934; Houston et al., 1979b) and that topographic relationships and probably other environmental factors affect the spread and development of insect populations (Ehrlich, 1934; Schindler, 1962; Parker, 1974; Thomsen et al., 1949; Lyr, 1967; Zycha, 1951; Houston et al., 1979b). While it appears that our knowledge about C. fagisuga is fairly broad, we know considerably less about the fungal components of the disease complex.

The fungus

We know that Nectria species are involved in beech bark disease but their exact identity is unclear. The fungi in Europe have been variously described as N. ditissima (Hartig, 1878), N. galligena (Thomsen et al., 1949), and N. coccinea (Ehrlich, 1934; Parker, 1974). In North America the fungus primarily associated with the disease was recognized as a variety of N. coccinea (Ehrlich, 1934) and later named N. coccinea var. faginata (Lohman and Watson, 1943). N. galligena has also been found to cause cankers on trees infested with C. fagisuga (Spaulding et al., 1936) and has recently been shown capable, together with C. fagisuga, of producing all of the symptoms of beech bark disease (Cotter, 1974).

Recent taxonomic studies comparing nutritional requirements and enzyme and protein patterns help clarify species relationships (Doyle, 1971; LaMadeleine, 1973; Stone, 1967). The sexuality and the cultural conditions for perithecia formation of *Nectria coccinea* and its North American variety have been investigated (Parker, 1976; Booth, 1966; Cotter, 1974). We know also, that these two *Nectria* species are attacked by the mycoparasite *Gonatorrhodiella highlei* A. L. Smith (Ayers, 1941; Blyth, 1949a, b).

Other organisms

We have found that many other organisms are involved in this complex disease (Shigo, 1964). Some, including mosses, algae, and lichens appear to offer protection for and thus favor *C. fagisuga* (Ehrlich, 1934; Houston *et al.*, 1979b), while the heavy stroma of the bark fungus Ascodichaena rugosa Butin appears to discourage *C. fagisuga* on European beech (Houston, 1976a, 1979b). Other organisms have been investigated, including bark and wood boring beetles and decay fungi that contribute to the rapid deterioration of injured and killed trees and to the familiar symptom of stem breakage called « beech snap ». In North America young beech trees developing in areas that were severely affected earlier by beech bark disease are often heavily attacked by another scale insect, *Xylococculus betulae* (Perg.) Morrison (Shigo, 1962). Wounds created by *X. betulae* render young stems highly defective and in turn provide colonization sites for *C. fagisuga* and *Nectria* spp. (Shigo, 1962; Shigo, 1964; Houston, 1975).

So, if it is true that we know all of this, we might logically ask the question : Why have a Beech Bark Disease Colloquium ? The answer can be stated very succinctly : What we do know about beech bark disease is not enough.

We cannot stop this disease — we still do not have enough understanding to prevent or reduce massive disease losses.

What we do not know (Or, what we still need to know)

About the forest stand

We do not know if we can manipulate the forest to reduce disease losses. Efforts to reduce the rate of increase in the causal agents or of disease development by stand thinning have thus far proved of little value (Crosby and Bjorkbom, 1958). Results from similar experiments in Great Britain are reported at this Colloquium. One factor not yet explored critically is the effect of various species mixtures and densities on disease development.

About the host trees

We need studies to clarify the potential influence of environmental factors in predisposing beech trees to infestation by *C. fagisuga* and perhaps to infection by *Nectria* spp. Whether fluctuations in insect populations observed in Europe and in North America are due to influences of environment on host tree susceptibility, on the insect itself, or both is not clear — and is very difficult to ascertain. This situation is confounded further by the probable occurrence of natural host resistance (at least in *F. grandifolia*) to *C. fagisuga*, and in North America to *Xylococculus betulae* and host tolerance to the associated *Nectria* species (Shigo, 1962; Shigo, 1964; Houston, 1975; Camp, 1951).

Studies are needed to clarify the validity and nature of apparent host resistance, and how such resistance is influenced by environment, by tree age, and by biotic factors that appear to either enhance or reduce local tissue susceptibility. Such factors could include wound agents (e. g. X. betulae in North America, N. ditissima in Europe) and bark coverings such as lichens (e. g. Lecanora conizioides in parts of Europe); or fungi (e. g. Ascodichaena rugosa in Great Britain).

About the insect

Even though we recognize that *C. fagisuga* is the necessary precursor to severe *Nectria* infection, we lack a good understanding of the factors that predispose trees or tissues to its attack, and of environmental factors that limit or encourage its development. We also have little insight into how the insect alters bark tissues biochemically in favor of *Nectria* spp. In fact, it is still unclear whether the fungi enter trees directly via stylet wounds, or indirectly through cracks created as bark dries out as a consequence of insect feeding. And, although a number of predators are known, their role in population regulation and their potential for manipulation (in many parts of the world) is unclear.

About the fungi

The role of fungi in beech bark disease is still controversial. Individual researchers have vacillated in their conception of *Nectria*'s role — one time describing it as a necessary part of the causal complex, and another time as a strictly secondary organism (e. g. Rhumbler, 1914; Nusslin and Rhumbler, 1922; Rhumbler, 1931). This latter view has surfaced again recently (Braun, 1977). The potential role of other *Nectria* species in beech bark disease is not entirely clear. In North America both *N. coccinea* var. *faginata* and *N. galligena* have been associated with the disease (Spaulding et al., 1936; Cotter, 1974), and in the United States and in Europe, the isolation of other *Nectria* species from those suspected of having beech bark disease is sufficiently frequent to warrant a close look at possible relationships.

A major question still unanswered is whether N. coccinea var. faginata is native to North America or was introduced along with C. fagisuga. Aside from one report (Spaulding et al., 1936) the fungus has not been isolated from areas free of C. fagisuga in North America (Cotter, 1974). But since it also is not known to occur in Europe, its origin is still in doubt (Parker, 1974).

And finally, the mycoparasite, G. highlei, first studied in Great Britain (Blyth, 1949a, b) and then in North America (Ayers, 1941, Ehrlich, 1942, Gain and Barnett, 1970) was recently found associated with beech bark disease in Great Britain (Houston 1976b) and in France (Perrin, 1977). The effects of G. highlei on Nectria in nature, and its potential for exploitation, are unknown.

The Look Ahead

We cannot effectively cope with beech bark disease with only current information. Aside from recommendations to avoid growing beech on high-yield sites, especially on slopes, or to salvage diseased trees as soon as possible, we can offer the forest manager nothing that will enable him to prevent the disease or to reduce losses in the long run. It is clear that answers to many of the questions posed above are necessary before sound recommendations can be made for coping with the disease through forest management practices. Especially appealing to many of us is the possibility that with increased understanding one or more natural controlling factors may be exploitable, especially genetic resistance, predators of the insect, or parasites of the pathogen.

The promises, just over the horizon, are what have brought us together for this Colloquium. I hope our interactions here will not only benefit our respective current research programs but will also trigger future cooperative research ventures between our countries and continents. It is highly probable that the solutions to this international complex problem will be arrived at only through an international research thrust.

Reçu pour publication en décembre 1979.

Résumé

Maladie de l'écorce du hêtre Un bref commentaire sur nos connaissances et ses lacunes

Nous savons que la maladie de l'écorce du hêtre commence par l'infestation de la cochenille *Cryptococcus fagisuga* dont l'action nutritive rend l'écorce vulnérable à un parasite de faiblesse Nectria coccinea (Europe) ou sa var. faginata (Amérique du Nord). Les connaissances acquises sur l'insecte comme sur le champignon sont considérables. Cependant notre compréhension des facteurs prédisposant l'arbre ou les tissus à la maladie, comme celle de l'influence de l'environnement sur son développement, reste insuffisante.

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