BEECH BARK DISEASE IN GREAT BRITAIN[1]

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Abstract.--The status of beech bark disease in Great Britain is summarised with respect both to historical perspectives and to the contemporary situation. Features of the disease which relate particularly to its occurrence in Great Britain are listed. Some tentative findings from recent observations and experimental work are presented.

INTRODUCTION

European beech (Fagus sylvatica L.) is an important constituent of the deciduous broadleaved forests of north-west Europe. About half of the beech in Great Britain is grown in southern England where it is the major hardwood planted on the chalk downlands. It will grow better on these calcareous soils than most other broadleaved trees.

Beech bark disease in its strictest definition is a complex disorder primarily attributed to the effects of a pathogenic species of the ascomycete fungus Nectria Fries, following infestations of the felted beech scale Cryptococcus fagisuga Lind. on the bark of Fagus species. These disease organisms appear to be endemic in stands of F. sylvatica in northwest Europe (Thomsen et al 1949). In Great Britain during the 19th century the death of many European beech was ascribed to the effect of the insect alone. British accounts at that time described it as a most destructive pest. Indeed, eighty years ago the Revd. Wilks (1902), commenting on the situation in southeast England, feared that "..... beech is doomed all over the country, and ..... the next generation will only know by pictures and reports how gloriously beautiful our forest beeches have been". Yet, although serious local outbreaks of the disease can occur, the fears of widespread devastation of beech in Great Britain have never been realised.

Beech bark disease appears to be most prevalent in pole stage beech i.e. with an age range of 20 to 45 years. Extensive plantings of beech in the 1930s fostered the major outbreaks of beech bark disease which built up in the 1960s. The peak of post-war beech plantings occurred in the 1950s, and it is these plantations which will be at their most susceptible during this current decade. Some commentators have suggested that the disease is cyclic in nature, with upsurges every 30 to 40 years. This may be explained in part by the natural tendency for an equilibrium to form in a host/pathogen relationship following an outbreak, particularly where the disease occurs in an endemic situation. However, it is more likely to be a reflection of the peak periods of beech plantings which subsequently give rise to a corresponding series of peaks in disease incidence at the time of maximum host susceptibility.

Variations in symptom expression between diseased beech in different localities can in part be attributed to differences in tree age and local site and climatic factors (Parker 1974a). The occurrence of similar symptoms on older beech trees initiated by adverse environmental conditions (Lonsdale 1979, 1980) also contributes to this variability.

HISTORICAL PERSPECTIVE

Following descriptions of a diseased condition of beech in Europe in the early 19th century, the first report from Britain seems to be that given by McIntosh (1849) who referred to a white rapidly spreading fungus which gave the bark of beech a snow-covered appearance. The latter part of this description would seem to apply to heavy C. fagisuga infestation, although

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as this usually builds up over a few years, the rapid growth could refer more appropriately to bark surface colonisation by the light coloured mycelium of the fungus Athelia arachnoidea (Berk.) Jül. which commonly appears on healthy beech trunks in years when the summer months are dull and wet. Nevertheless, infestations of C. fagisuga can on occasions appear to change from the imperceptible to the obvious during a two to three year period due simply to the effect of a logarithmic phase of growth that can occur with most plant and animal populations.

Despite much concern about the noticeable infestations of C. fagisuga and associated beech deaths during the latter part of the 19th century, and fears for the general demise of beech in Britain such as those expressed by Wilks (1902), the beech remains one of the leading species of broad-leaved trees grown in Great Britain.

Not all beech trees or beechwoods are seriously affected by the disease. Newstead (1903) referred to the sparse occurrence of C. fagisuga on a stand in the Cotswolds (limestone hills in southwest England). Seventy years later the owner of an estate there claimed that the virtual absence of disease in his beechwoods, which were of excellent form and growth, was the product of continued good management for well over a century (J. Workman, pers. comm.).

Yet the disease problem in the 20th century is a continuing one, and the overall picture now is perhaps not too different from the one seen by our predecessors a century or more ago.

Perhaps the most significant step in our understanding of beech bark disease was the publication of John Ehrlich's monograph in 1934, following his investigations in North America during the early 1930s. It is of interest to note that he subsequently became for a time a Research Fellow at the Commonwealth Mycological Institute, Kew, England, from where he was able to observe for himself the disease in British beech-woods. His initial reaction was that the endemic disease situation amongst British beech differed markedly from the effects of the beech bark disease epidemic occurring in northeast U.S.A. and eastern Canada. A possible explanation for this is that Ehrlich was shown mature beech stands, young plantations being infrequent at that time. Older beech trees are prone to problems somewhat similar to beech bark disease, but the underlying cause and the expression of symptoms differ perceptibly from beech bark disease as it occurs on younger beech. With hindsight Ehrlich's comments seem to be an over-statement of the case. Certainly differences do occur (these are listed in more detail in the next section of this paper) but in essence the beech bark disease complex seems remarkably similar on both sides of the north Atlantic (Houston et al 1979).

Day (1946) brought the subject of beech pathology into discussion once again. His account was followed by Forestry Commission reports of bark dieback and death of beech (Peace 1950-55 inclusive; Peace and Murray 1956 and 1957; Murray 1957). In the course of these studies Peace (1954) commented that some if not all of three factors were associated with the disease: (i) a thin freely-drained soil, (ii) an old crop, often over 100 years of age, and (iii) a history of underthinning, leading probably to great competition among individuals in the stand.

In the 1960s renewed concern was expressed within the Forestry Commission over outbreaks of beech bark disease in young beech plantations. This led to the inception in 1969 of the current research programme, to which Parker (1974a, b) contributed initially, and which was extended in depth from the mid-1970s by the work of D. Lonsdale and D. Wainhouse involving mycological studies, investigations of host tree anatomy and resistance and insect dispersal.

DISTINCTIVE FEATURES ASSOCIATED WITH BEECH BARK DISEASE IN GREAT BRITAIN

Houston et al (1979) noted many of the features of beechwoods and beech bark disease in Great Britain which differed from those observed in North America. In brief, these include:-

1. the different species of host tree and consequent likelihood of differences in bark anatomy;

2. the predominance of young beech plantations often planted closely (1 x 1 m) as a pure first rotation crop in the 1930s and 1940s, but thereafter usually in mixture with a conifer nurse species;

3. the common planting of beech on chalk downland, where there is often a steep scarp slope;

4. the bark flora, including the fungus Ascodichaena rugosa Butin which occupies part of the surface which otherwise could be available to C. fagisuga, and the abundance of the crustose lichen Lecanora conizaeoides Nyl. ex Cromb. which may favour the initial establishment and build-up of the insect;

5. the presence of large old beech trees which

may harbour sources of insect and fungal inocula;

6. the species or variety of Nectria associated with death of beech bark following C. fagisuga infestation - in Great Britain this seems to fit the description of N. coccinea (Pers. ex Fr.) Fries and its imperfect state Cylindrocarpon candidum (Link) Wollenw.;

7. the occurrence of bark pitting associated with the feeding activity of C. fagisuga, which is evident on some but not all, of the young beech trees which become infested by the insect;

8. the secondary organisms associated with the decay of diseased beech stems, in particular the white rot fungus Bjerkandera adusta (Fries) Karsten often linked with the phenomenon of beech "snap".

My previous observations (Parker 1974a, b) suggested that on some sites with good nutrient and moisture supplies producing beech crops of high yield class, the disease became apparent at an early age (15 to 25 years) and subsequently became severe, particularly where a first silvicultural thinning was late (i.e. around 30 years of age). Disease outbreaks were usually first noticed in scattered groups within a plantation. This feature may in part be explained by the localised dispersal of C. fagisuga larvae (Wainhouse 1980), although local soil disturbance may also cause similar expression of some disease symptoms, i.e. chlorosis and/or tarry spots but not of C. fagisuga infestation (Lonsdale et al 1979; Lonsdale and Pratt 1981).

Recent discussion has focused on the methods of beech silviculture (Aldhous 1981; R A G Coxwell, unpublished). Certain observations and results of experimental work suggest that beech bark disease in Great Britain is worse in an overcrowded crop where competition develops between individual trees, and in particular between the developing crowns in the woodland canopy (Parker 1980, R A G Coxwell, unpublished). After a decade or more of observations on the beech bark disease problem in the young beech plantations of southern England it does seem that there is a short phase in the life of a beech plantation during which a "contracted epidemic sequence" of beech bark disease expression occurs. This is centred around a peak of severity at about 30 years of age, i.e. the current scheduled time for the first silvicultural thinning of these crops under Forestry Commission management. Initial incidence starts at about 20 years of age and by 45 years after one or two silvicultural thinnings (which at times may have been partly selective with regard to diseased stems), affected crops appear to be in an aftermath stage. Indeed in some compartments where a decade ago the disease appeared to be at peak severity, the beech crop now looks remarkably healthy, aesthetically pleasing, and with every indication of producing a good final crop of mature trees. An associated factor in the recovery of diseased beechwoods is the ability of the remaining trees to make compensatory growth and to close any gaps in the canopy caused by the death of neighbouring trees within a few years.

What seems to have occurred is a "natural thinning operation" in places where appropriate forest management has been delayed, usually on economic grounds. Only in very exceptional circumstances have affected areas had to be clearfelled and replanted. With regard to market considerations, the death of young pole-stage beech means that the volume available for pulpwood is reduced, but this is an area of uncertain and fluctuating economics. It is indeed difficult to estimate the loss of timber volume or economic return before the basic complexities of beech bark disease and the interaction with silvicultural management are evaluated.

If there were to be an increased movement towards the selection of good crop trees at an early stage, together with heavier and more frequent thinning of the beech crop to produce more high quality timber (Matthews and Newton 1982), then it is possible that beech bark disease may become much less of a problem in the future.

RECENT OBSERVATIONS

In order to gather detailed sequential data on host characteristics, symptom expression, and disease development both on individual trees and on a plot basis, a series of long-term observation plots each containing about 400 beech trees was established early in 1979. In selecting the crops for this experiment consideration was given to ensure representative combinations of age class, site type, and phase of disease. Each of the individually numbered beech trees is assessed annually for a range of host and disease characters. For each such character, descriptions and score classes have been closely defined in order to reduce the degree of error due to subjective assessment. It must be stressed that the data given below (which are mainly concerned with the incidence of C. fagisuga) are on the basis of only three years assessments and must inevitably be preliminary. The experiment in the first instance is scheduled to run for eight years before any major consideration is given to revision of the current procedure.

Initial C. fagisuga establishment on young trees and later the occurrence of stem pitting seems most favoured on the main trunk at a height of about 1 to 3 metres; a fact which relates in part to the pattern of passive dispersal and deposition of the insect larvae and may be partly due to the host bark characteristics on that portion of the young beech stem.

In the early phase of the disease when the crop is about 20 years of age, over half of the trees are still completely free from C. fagisuga infestation (over 75 per cent were "clean" in the 21 year old plot where beech is in mixture with a conifer nurse), the remaining trees usually bearing a trace or very light infestation of the insect. Moderate C. fagisuga infestation was present on less than 3 per cent of these young trees (with virtually no heavy infestation), whereas in the plots which were at about peak disease severity between 12 and 20 per cent of the crop bore moderate or heavy C. fagisuga infestations. In this peak phase of the disease when the trees are about 30 years old, uninfested trees are virtually absent. In many cases the more heavily infested trees are likely to die as beech bark disease takes its course, although in some cases recovery occurs. In the aftermath stage, by about 40 to 45 years of age, few moderately or heavily infested trees can be seen.

There is a marked tendency for the heavier C. fagisuga infestations to occur in trees of the dominant or co-dominant classes, rather than on sub-dominant or suppressed trees. This is perhaps because the bark of these faster growing trees more quickly reaches the stage when it may be anatomically and physiologically suitable for C. fagisuga infestation.

While in general the rate of increase of C. fagisuga infestations on polestage beech is relatively slow, some observations have indicated the possibility of a rapid and heavy build-up on a very small proportion of the crop. Thus, among forty trees under observation in a 35 year old pure beech plantation showing generally low colonisation by C. fagisuga, two initially uninfested trees became heavily infested within seven years (Parker, unpublished data); and one uninfested tree to which C. fagisuga inoculum was artificially introduced developed a heavy infestation after only four years (Houston et al 1979).

CONCLUSIONS

The normal healthy physiology of beech can be disturbed by abnormal external ("stress-inducing") factors of which, in the case of young pole-stage crops, the most important is a heavy infestation of C. fagisuga leading to the subsequent development of beech bark disease. To the owner or manager of a young beech plantation in which the disease can build up to a peak of severity by the time the crop is 30 years of age, the apparent extent of destruction and economic loss induces pessimistic thoughts about the wisdom of growing beech. However, the signs are that even in severely affected young stands the surviving trees will grow on to form a mature beechwood.

There are various characteristics of beech bark disease in Great Britain which differ from those observed in the epidemic which continues to advance westwards and southwards in North America, but the basic theme of the Cryptococcus/ Nectria association is similarly expressed. Current British research into the problem is aimed at elucidating the fundamental mechanisms of disease etiology and epidemiology. Once these basic mechanisms are more fully understood we will be in a better position to advise forest managers on preventative or remedial silvicultural practices aimed at reducing losses due to beech bark disease, and therefore to producing a greater volume and a higher quality of beech timber for use in the 21st century.

ZUSAMMENFASSUNG

Berichte über das Auftreten der Buchen-Rindennekrose in Grossbritannien in der Vergangenheit wurden zusammenfassend dargestellt. Die Befürchtungen, die von den Waldbesitzern und Bewirtschaftern erkrankter Buchenwälder in den 70er Jahren geäussert wurden, sind mit denen aus dem 19. Jahrhundert vergleichbar. Trotz der offensichlich örtlich schweren Krankheitsausbrüche in 20-40 Jahre alten Beständen ist Fagus sylvatica nach wie vor eine der wichtigsten Laubbaumarten in Grossbritannien.

Das Krankheitssyndrom ist in Europa, wo es endemisch, und in Nordamerika, wo es epidemisch auftritt, im Wesentlichen ähnlich ausgeprägt.

Die ökologischen Bedingungen für den Wirt und die spezifischen Mikroorganismen der Rinde bestimmen das Erscheinungsbild der Krankheit in England. In der Regel beginnt der Aufbau der Lauspopulation (Cryptococcus fagisuga) in rund 20jährigen Beständen. Der Höhepunkt der Infektion durch Nectria ist bei etwa 30 Jahre alten Buchen zu beobachten, worauf es zu Ausfällen kommt.

Im Alter von etwa 45 Jahren ist dann eine Phase erreicht, in der die Krankheit nur noch in geringer Intensität vorkommt. In den meisten Fällen weisen die überlebenden Bäume ein vergleichsweise besseres Wachstum auf und die betroffenen Bestände scheinen durchaus zu hiebsreifen Beständen heranwachsen zu können. Es gibt Hinweise dafür, dass die Krankheit bei zu grossem Dichtstand der Bäume verstärkt auftritt, besonders wenn Konkurrenz zwischen den sich entwickelnden Kronen herrscht. Unter Umständen könnten geeignete waldbauliche Massnahmen zu einer Verringerung der Krankheitsintensität und letztendlich zu besserer Holzqualität führen.

RÉSUMÉ

La physiologie normale d'un hêtre sain peut être modifiée par des facteurs externes anormaux ("provocateurs de stress") qui, dans le cas de peuplements de perchis de hêtres sont représentés par les infestations du C. fagisuga conduisant au développement de la maladie de l'écorce du hêtre. Pour le propriétaire ou l'aménagiste d'une jeune plantation de hêtres dans laquelle la maladie peut atteindre une culmination maximum au moment où le peuplement atteint 30 ans, l'importance apparente des dégâts et des pertes économiques conduisent à un pessimisme évident concernant la production du hêtre. Cependant, il semble que même à partir de jeunes peuplements fortement affectés, les arbres survivants puissent croître assez pour former une futaie de hêtres.

Il existe plusieurs particularités de la maladie de l'écorce du hêtre endémique en Grande-Bretagne. Elles diffèrent évidemment de celles de l'épidémie qui continue de s'étendre vers le sud et l'ouest de l'Amérique du Nord, mais la situation fondamentale de l'association Cryptococcus/Nectria s'y retrouve intacte. Actuellement, la recherche britannique dans ce problème essaie d'élucider les mécanismes fondamentaux concernant l'étiologie et l'épidémiologie de la maladie. Une fois ces mécanismes de base bien compris, nous serons dans une meilleure position pour conseiller les aménagistes forestiers sur des pratiques sylviculturales préventives ou correctives pour réduire les pertes dues à la maladie de l'écorce du hêtre, et ainsi à produire un volume plus grand et une meilleure qualité de bois de hêtre pour utilisation dans le 21ème siècle.

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