PROTECTING EUROPE'S FORESTS: HOW TO KEEP OUT BOTH KNOWN AND UNKNOWN PATHOGENS*

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ABSTRACT

During the last century, the tree populations of Europe suffered from a number of serious epidemics resulting from the introduction of pathogens from abroad. Currently the principal approach to minimising the risk of introducing further exotic pathogens is through Pest Risk Analysis (PRA) in which measures are devised to prevent potentially dangerous pathogens from using their likely pathways of arrival. A relevant example is provided by *Ceratocystis fagacearum* (Bretz) J.Hunt, the cause of oak wilt in eastern North America, which was subject to critical evaluation in the 1970s and 1980s. Beneficial consequences can flow from such an exercise but the longevity and large size of trees may mean that crucial information can be obtained only from a research programme that takes many years to complete.

Moreover, there are other difficulties with the PRA approach when it comes to forestry. Firstly, most forest pathogens are classified using morphological criteria which are inadequate for the recognition of differences critical to the ability to cause disease. This means that the occurrence of a named organism in Europe does not mean that there is nothing to fear from further introductions of what is said to be the same species: witness the bitter experience with the fungi now identified as two species — *Ophiostoma (Ceratocystis) ulmi* (Buisman) Nannf. and *O. novo-ulmi* Brasier. Secondly, the PRA approach results in the development of highly specific quarantine measures which are likely to leave "loopholes" that unrecognised pathogens can use. There must be many organisms with great potential for damage in Europe that are living in ecological balance with their host trees in other forest regions of the world and thus can never be identified as candidates for a PRA. Therefore there is a strong justification for a quarantine philosophy that recognises the value of non-specific control measures for certain types of imported material — in particular, for unprocessed wood.

Keywords: disease epidemics; Pest Risk Analysis; Dutch elm disease; oak wilt; non-specific controls.

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INTRODUCTION

This paper falls into three parts. Firstly, an account is given of the characteristics of some damaging tree disease epidemics that have resulted from the introduction of exotic pathogens to Europe (Gibbs & Wainhouse 1986). Secondly, the current plant health philosophy of Pest Risk Analysis is described, and its strengths and weaknesses are examined with reference to the threat posed by the oak wilt pathogen *Ceratocystis fagacearum*. Thirdly, various arguments are presented to show that, whatever merits the PRA approach may have when applied to agriculture, there are fundamental weaknesses with its application to forestry.

EUROPEAN EXPERIENCE OF INTRODUCED TREE PATHOGENS

During the last 100 years, the tree populations of Europe have experienced a number of serious epidemics resulting from the introduction of pathogens from abroad. Examples include Chestnut blight, Cypress canker, and Dutch elm disease.

Chestnut blight is caused by *Cryphonectria (Endothia) parasitica* (Murrill) Barr. A good account of this stem-killing disease and its biology has been given by Griffin & Elkins (1986). It first came to prominence in North America where, during a 40-year period from 1904, the American chestnut, *Castanea dentata* (Marsh.) Borkh.) was destroyed throughout its native range. The disease was discovered in Italy on European chestnut *C. sativa* Mill. in 1938. Control efforts failed and within 25 years it was present in all the major chestnut-growing areas of Italy and had also spread to other parts of Southern Europe. Since 1950 there has been a natural remission of disease in many places owing to the effects of a cytoplasmically-transmitted dsRNA, but it remains a significant cause of damage and threatens unaffected chestnut further north in Europe. It has long been known that the fungus occurs in the far east and that Asiatic species of chestnut possess appreciable resistance. Infected planting stock is considered to be the most likely means whereby the disease was introduced into both North America and Europe. Molecular evidence indicates that Japan is a more likely source than China (Milgroom 1995).

A lively account of the characteristics and impact of Cypress canker, caused by *Seiridium (Coryneum) cardinale* (Wag.) Sutton et Gibson, has been provided by Graniti (1998). The disease began to cause damage to Monterey cypress (*Cupressus macrocarpa* Hartw.) in California in about 1915 although it was not described until 1927. It was first recorded in Europe in 1940 and quickly spread to the Mediterranean region where it has caused much stem and branch damage to the spire-shaped Italian cypress *Cupressus sempervirens* L., in the process impoverishing landscapes that have been commemorated in masterpieces of art since Renaissance times. Thus a survey in central Italy in 1978 showed that 18% of the 4 million cypresses in the area were either dead or severely affected by the disease. The geographic origin of *S. cardinale* is unknown but, given the high susceptibility of both North American and European cypresses, it is unlikely to be native to either continent.

Dutch elm disease, a vascular wilt, was first described from Belgium and the Netherlands just after the First World War and by 1930 had spread as far as Italy and Bulgaria. It is estimated that this first epidemic, caused by *Ophiostoma (Ceratocystis) ulmi*, killed one-third of the 1.2 million elms in the Netherlands, whereas in the United Kingdom only

10–20% of the trees died between 1930 and 1960 (*see* Gibbs 1978a). In the latter years disease symptoms were only rarely seen. However, in the late 1960s a new and devastating epidemic began in Britain. An estimated 18 million trees had died by 1980 and in some areas the loss was total. The origin of this second epidemic was traced to the introduction on rock elm logs from North America (Brasier & Gibbs 1973) (Fig. 1) of what is now recognised to be a separate species of the pathogen (*O. novo-ulmi* Brasier). Very serious losses have now occurred right across Europe, in part caused by another form of *O. novo-ulmi*, in all probability of separate exotic origin (Brasier 1996). The ultimate origin of the fungus remains unknown. Some elms native to Asia possess significant resistance but, despite investigation, neither *O. ulmi* nor *O. novo-ulmi* has been found there. However, in 1993 a third species, *O. himal-ulmi* Brasier & M.D.Mehrotra, was found on *Ulnus wallichiana* Planch in Northern India in association with various native Scolytid beetles. There was no evidence of wilt disease but the fungus proved to be very pathogenic when tested on English and Commelin elms (Brasier & Mehrotra 1995).

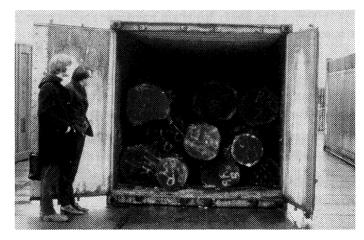


FIG. 1–John Gibbs (right) and Clive Brasier of the Forestry Commission examining a container of rock elm logs at Southampton Docks in 1973. It was just such a consignment, arriving a few years earlier, that resulted in the introduction of *Ophiostoma novo-ulmi (see* Brasier & Gibbs 1973).

PEST RISK ANALYSIS APPROACH TO DISEASE PREVENTION

Currently, the principal approach to minimising the risk of further such dangerous introductions is through Pest Risk Analysis (PRA) in which measures are devised to prevent specific exotic pathogens from using their likely pathways of arrival. The key organisations in this process are regional bodies such as The European Plant Protection Organisation (for an account of the work of EPPO, *see* Smith 1979). The responsibility for putting the measures in place falls to individual nations or to groups of nations — such as the European Union.

The process can be summarised as follows:

(1) The selection of a potentially harmful organism that is not present in the region in question;

- (2) The collation of information on key aspects of the organism's biology, ecology, and pest status e.g., geographic distribution, hosts, epidemiology, economic impact, and management; and
- (3) The production of a PRA with an assessment of the risk of entry, establishment, and potential damage the harmful organism may cause, together with options for management.

On the strength of this assessment, specific quarantine regulations can be drawn up to minimise the chance that the named organism will arrive from abroad.

Example of Ceratocystis fagacearum, the Cause of Oak Wilt

A relevant example of the PRA-type approach to protecting Europe's forests is provided by the assessments that have been conducted on the fungus *Ceratocystis fagacearum*. This is the cause of a vascular wilt disease that was first described in eastern North America during the 1940s (Henry *et al.* 1944). Initially it was thought to be present in only a few states in the upper Mississippi valley, but in 1950 it was reported from Arkansas and Pennsylvania. Surveys were hurriedly instituted and by the end of 1951 it had been recorded in 18 eastern and central states, which together contained more than half the growing stock of oak in the United States. A period of hectic research followed. *Inter alia*, it was discovered that red oaks (sub-genus *Erythrobalanus*) were very susceptible to the disease, while white oaks (sub-genus *Lepidobalanus*) were much more resistant.

After 1960, concern over the likely impact of the disease gradually decreased, largely because within the commercially important oak forests of states such as Missouri and West Virginia the number of trees becoming diseased each year remained roughly constant. Indeed, the data indicate that through much of the affected area only one tree dies of oak wilt per square mile (2.6 km²) of forest each year.

However, high infection rates were recorded in the north central states of Minnesota and Wisconsin where the trees, although of little timber value, can be very important for shade and amenity. These high rates were due largely to rapid transmission of the fungus from tree to tree via root grafts. The same process is also very important in the Southern state of Texas where *C. fagacearum* is now known to be the cause of serious mortality in stands of live oak (*Quercus fusiformis* Small and *Q.virginiana* Mill.). (For an introduction to the relevant literature see Gibbs & French 1980; Appel 1995, 2001.)

Ceratocystis fagacearum was identified as a potential threat in Europe at an early stage and quarantine regulations were enacted in some countries. However, concerns over the danger of an introduction increased greatly in the 1970s when, as described above, it was realised that the devastating second epidemic of Dutch elm disease owed its origin to an introduction from North America. In 1977, I was fortunate enough to be able to spend a year in Minnesota charged by my employers, The Forestry Commission of Great Britain, with investigating the oak wilt risk (*see* Gibbs 1978b, 1979) and several other European colleagues also made visits of evaluation. In 1980 these national initiatives were brought together, via what was then the European Economic Community (subsequently the European Union), to form "GROW" (Group for Research on Oak Wilt) — this positive acronym being chosen partly to contrast with the doleful "DED" (Dutch elm disease)! In this forum, expertise on the problem could be shared and agreement on the nature and extent of the threat achieved.

A useful account of the findings of GROW, supported by some short-term research conducted in part with EC funds, was produced by Gibbs *et al.* (1984). In addition to providing information on treatments to eliminate the pathogen from oak logs being imported into Europe for the production of veneer, this paper focused heavily on the potential importance of the European oak bark beetle (*Scolytus intricatus* Ratzeburg) as a vector of the pathogen should the latter ever arrive in the continent. The case was made that *S. intricatus* was likely to be a much more effective vector of *C. fagacearum* than the Pseudopithyophthorus bark beetles that are associated with it in the United States, this because of its much larger size and more aggressive breeding behaviour. It could be expected to form breeding galleries in the trunks of oaks that had just succumbed to the disease, and in the process to provide favourable conditions for the establishment of a link between the pathogen and the developing beetles. Moreover, when conducting its maturation feeding on small twigs on healthy trees, *S. intricatus* makes a large feeding wound that typically scars the xylem, thus facilitating pathogen transfer and infection (Fig. 2).

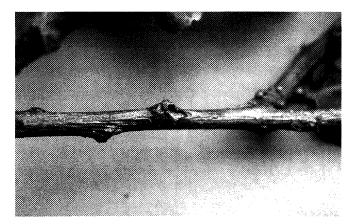


FIG. 2-One-year-old twig of *Quercus robur* in which a bud (centre) has been eaten away during maturation feeding by *Scolytus intricatus* to expose the xylem.

The information collated through GROW made a valuable contribution to knowledge and resulted in the development of new quarantine measures applicable across a broad geographic area. In addition, the identification of the potential capacity of *S. intricatus* to act as a vector of *C. fagacearum* has led to a recognition that this insect could transform the oak wilt situation in the major oak forests of North America, should it ever become established there. As a result, quarantine regulations have been enacted to exclude it.

These are all beneficial consequences of a PRA type of exercise. However, at the same time it must be noted that crucial information on the true threat posed by the pathogen has come to light only from the long-term research initiated by GROW: it could not have been predicted from the desk-studies and short-term research exercises that are conducted under the normal PRA procedures. During the 1970s and 1980s, advocates for the North American timber exporters, keen to see existing patterns of trade maintained, laid great

emphasis on the high resistance of North American white oaks to C. fagacearum. They contended that, since all the native European oaks were white oaks, there could be little risk that the arrival of the pathogen would result in major damage. Not satisfied with this argument, Jean Pinon of the Institut National de la Recherche Agronomique in France organised the collection of acorns from a range of European oak species and, with the invaluable co-operation of Bill MacDonald of West Virginia University and Frank Tainter of Clemson University, arranged for them to be imported into the United States. The resulting seedlings were planted out at sites at the two universities, together with North American white oaks (O. alba L. and O. prinus L.) and a red oak (O. rubra L.). To test resistance to a vascular wilt pathogen, a well-developed xylem structure is necessary and thus it was not until the trees were 12 to 14 years old that the critical inoculations could be conducted. The results (see example in Fig. 3) were remarkable. As expected, the North American white oaks showed little disease. However, the European white oaks developed very severe symptoms and most of the trees had died by the end of the season. Indeed, they proved to be almost identical in performance to the red oak O. rubra (MacDonald et al. 2001).

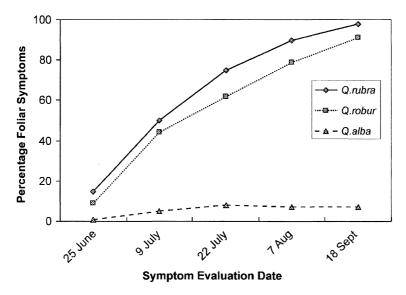


FIG. 3–Oak wilt symptom development in various oaks following branch inoculation in spring 1996 with *Ceratocystis fagacearum* (after MacDonald *et al.* 2001).

PROBLEMS WITH APPLYING THE PRA APPROACH TO FOREST PATHOGENS AND PESTS

The lesson to be learnt from the above exercise is that even when abundant scientific information is available and when significant research resources can be devoted to evaluation, the very longevity of trees and the length of time that is required for their mature structures to develop, may make it very difficult to ensure that the key information for an adequate PRA is obtained.

Moreover, there are further difficulties with the PRA approach when it comes to forestry. Firstly, the great majority of forest pathogens are classified using morphological criteria which are inadequate for the recognition of differences that may be critical to their ability to cause disease (Brasier 1997). Therefore, the fact that a particular fungal species has already been recorded in a continent such as Europe does not mean that there is nothing to fear from further introductions of what purports to be the same species. Here the classic example is provided by the pathogens causing Dutch elm disease. As outlined above, *Ophiostoma ulmi* caused significant but not catastrophic damage in Europe during the period 1920–60. Then began a highly destructive second epidemic that owed its origin to the introduction of a fungus that was subsequently named *O. novo-ulmi*. However, the "classical" morphological differences between the two species are extremely small and characterisation depends much more on the differences in culture, in genetic behaviour, and in molecular constitution (Brasier 1991, 1997).

Another example of the complexity that can exist within a named species is provided by *Mycosphaerella dearnessii* M.E.Barr, the cause of brown spot needle disease of pine. *Mycosphaerella dearnessii*, with a possible centre of origin in Central America, has now been recorded from North and South America, Europe, Australasia, and Asia. Molecular work (Huang *et al.* 1995) has confirmed earlier studies of physiological characteristics to show that in the United States there are two distinct populations of the fungus: a northern and a southern group. So far isolates collected in Europe seem to correspond to the northern group. However, it might well be that it is the southern group that has the attributes that would mean that it poses the greatest threat to the pines of Europe.

The second weakness of the PRA approach to forest pathogens is the sheer impossibility of identifying all the pathogens that are capable of causing damage if transported to a new environment. This is because many such organisms do no harm in their region of origin and are thus not candidates for critical examination. Chestnut blight provides a clear example of a fungus with a great potential for destruction abroad that causes little damage on its host trees in its native habitat. The story of Dutch elm disease, though not fully resolved, may well be similar. And there are uncounted numbers of micro-organisms living on and in the world's trees!

To understand the full significance of this point for forests and forest ecosystems, one must recognise the fact that, despite the use of plantations of exotic trees in some countries, most forests depend upon species that are growing within their native ranges. And the trees of one forest region are allied to those of other regions. This point can be well illustrated with reference to the Northern Hemisphere. Here there are four forest regions (in Europe, Asia, Eastern North America, and Western North America) which originated from a homogeneous Tertiary forest that once existed in what is now the Arctic Circle. During the Pleistocene glaciation these forests retreated southwards and, in the process, became separated from each other (*see* Fig. 4). As the oaks, birches, pines, and firs of each region evolved to give the species we know today, so also did their associated fungi, bacteria, and viruses. Who can say which of the array of micro-organisms now living harmlessly on or in the tissues of an Asiatic maple has the potential to be destructive to the maples of Europe!

By definition the PRA approach results in the development of highly specific quarantine measures which are likely to leave "loopholes" that unrecognised pathogens can use. For this reason there is a strong feeling among forest pathologists that the international policies

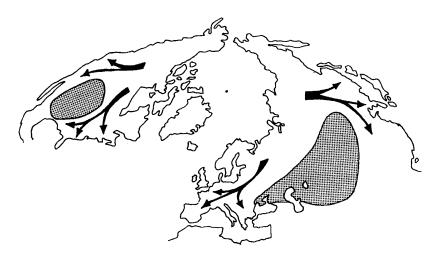


FIG. 4–Map showing the southward movement of the northern temperate Tertiary forest to give the four distinct forest regions of today. Stippling is used to indicate arid areas (after Gibbs & Wainhouse 1984).

that relate to the exclusion of exotic pathogens need to be re-examined. In particular, it is considered that certain types of imported material pose such a great risk that the requirement is for blanket "non-specific" control measures — so as to keep out the unknown pathogens as well as the known. This is the approach that was envisaged by officers of the American Phytopathological Society when they wrote to the then US Vice President Al Gore asking for all unprocessed wood products (including logs, dunnage, etc.) to be heat treated before they arrived in North America (Anonymous 1999).

REFERENCES

ANONYMOUS 1999: APS resolution on wood importation. Phytopathology News 33: 165.

- APPEL, D.N. 1995: The oak wilt enigma: perspectives from the Texas epidemic. *Annual Review of Phytopathology 33*: 108–118.
- ——2001: The basics of oak will biology and factors influencing disease incidence and severity. Pp. 71–81 in Ash, C.L. (Ed.) "Shade Tree Will Diseases". APS Press, St Paul, Minnesota.
- BRASIER, C.M. 1991: *Ophiostoma novo-ulmi* sp. nov., causative agent of the current Dutch elm disease pandemics. *Mycopathologia* 115: 151–161.

- BRASIER, C.M.; GIBBS, J.N. 1973: Origin of the Dutch elm disease epidemic in Britain. *Nature* 242: 607–609.
- BRASIER, C.M.; MEHROTRA, M.D. 1995: Ophiostoma himal-ulmi sp. nov., a new species of Dutch elm disease fungus endemic to the Himalayas. Mycological Research 99: 205–215.
- GIBBS, J.N. 1978a: Development of the Dutch elm disease epidemic in southern England: 1971–76. Annals of Applied Biology 88: 219–228.

——1978b: Oak wilt. The Arboricultural Journal 3: 351–356.

- GIBBS, J.N.; FRENCH, D.W. 1980: The transmission of oak wilt. USDA Forest Service Research Paper NC-185.
- GIBBS, J.N.; WAINHOUSE, D. 1986: Spread of forest pests and pathogens in the Northern Hemisphere. *Forestry* 59: 142–153.
- GIBBS, J.N.; LIESE, W.; PINON, J. 1984: Oak wilt for Europe? *Outlook on Agriculture 13*: 203–208.
- GRANITI, A. 1998: Cypress canker: a pandemic in progress. Annual Review of Phytopathology 36: 91–114.
- GRIFFIN, G.J.; ELKINS, J.R. 1986: Chestnut blight. Pp. 1–26 in Roane, M.K.; Griffin, G.J.; Elkins, J.R. (Ed.) "Chestnut Blight, other Endothia Diseases and the genus *Endothia*". APS Press, St Paul, Minnesota.
- HENRY, B.W.; MOSES, C.S.; RICHARDS, C.A.; RIKER, A.J. 1944: Oak wilt: its significance, symptoms and cause. *Phytopathology* 34: 636–647.
- HUANG, Z.Y.; SMALLEY, E.B.; GURIES, R.P. 1995: Differentiation of *Mycosphaerella dearnessii* by cultural characters and RAPD analysis. *Phytopathology* 85: 522–527.
- MacDONALD, W.L.; PINON, J.; TAINTER, F.H.; DOUBLE, M.L. 2001: European oaks susceptible to oak wilt? Pp. 131–137 *in* Ash, C.L. (Ed.) "Shade Tree Wilt Diseases". APS Press, St Paul, Minnesota.
- MILGROOM, M.G. 1995: Population biology of the chestnut blight fungus, *Cryphonectria parasitica*. *Canadian Journal of Botany 73 (Suppl. 1)*: S311–S319.
- SMITH, I.M. 1979: EPPO: the work of a regional plant protection organization, with particular reference to phytosanitary regulations. Pp. 13–22 in Ebbels, D.L.; King, J.E. (Ed.) "Plant Health, the Scientific Basis for Administrative Control of Plant Diseases and Pests". Blackwell Scientific Publications, Oxford.