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Effects of climate change on fungal diseases of trees

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Introduction

Climate has been of great importance in the development of associations between trees and pathogenic fungi. In particular, the geographic range of each species of tree or fungus is delimited by factors such as temperature, moisture, snowfall and windiness which affect growth, reproduction and dispersal. Such factors affect the incidence of diseases by determining the distribution of a particular pathogen in relation to the geographic range of a potential host. Also, within a region where both host and pathogen are present, the severity of disease can vary with climate. Such variations can result from the direct effects of climatic factors on the pathogen, or from their effects on aspects of host physiology which determine resistance to attack. Other effects may involve other organisms with which either the host or pathogen interact.

In natural ecosystems, associations between particular tree and fungal species are often of great antiquity and have evolved in ways which tend to avoid mutual destruction. Environmental stability may have been a prerequisite for the development of many of these host-pathogen associations and, if that is the case, it follows that they will be perturbed by major climate change. Less stable relationships tend to occur in the simpler ecosystems that initially exist in man-made plantations, often involving new combinations of host and pathogen species that have artificially been transported beyond their natural geographic ranges. In such cases, it can be envisaged that climate change would encourage major changes in disease incidence and severity.

In an attempt to make a profitable evaluation of the effects of climate change in these diverse situations, we have narrowed our scope to consider only some of the most widely predicted changes – namely that

winter temperatures in the temperate regions of the world can be expected to be a few degrees higher than at present and that there will be greater climate instability, including in particular more frequent summer droughts in the middle latitudes (Kräuchi, 1993). Secondly, we have concentrated attention predominantly on diseases of the woody tissues, since it is the perennial nature of these tissues that marks the difference between woody and herbaceous plants (see Pettitt & Parry, Chapter 2).

Finally, we have excluded from consideration the whole topic of saprotrophic survival, as this would demand a chapter on its own.

Direct effects on the pathogens

Effects on the geographic range of pathogens

The geographic ranges of fungal pathogens are, to some extent, determined by the temperature ranges over which they can grow, although many species are prevalent only in regions where temperature and other climatic factors are sufficiently close to optimal values to allow rapid growth and reproduction during part of the year. A very wide range of pathogens could be expected to show alteration of their geographic ranges in response to climate change, and the potential for this is best exemplified by those that respond to the year-to-year fluctuations that already occur.

Leaf rust of poplars (*Populus* spp.), caused by *Melampsora allii-populina*, is an example of a disease which, near the edge of its present climatic range, appears only sporadically due to temperature fluctuation. It is a topical example, since poplar growing is now being encouraged in many European countries as an alternative to producing agricultural surpluses. Many of the new fast-growing clones that are favoured for this purpose were bred in Belgium, where they were screened for field resistance to rust in the 1970s (Pinon *et al.*, 1987). It appears that *M. allii-populina* was virtually absent from the trial grounds in central Belgium at this time. Thus, the clones were in effect screened only against another rust species, *M. larici-populina*, which, unlike *M. allii-populina*, is well established throughout Belgium and in much of northern Europe. In 1985, some of the clones were quite heavily infected by rust in Belgium, and the fungus was found to be *M. allii-populina* which, as shown in Fig.1.1, occurs regularly only in regions further south (Somda & Pinon, 1981).

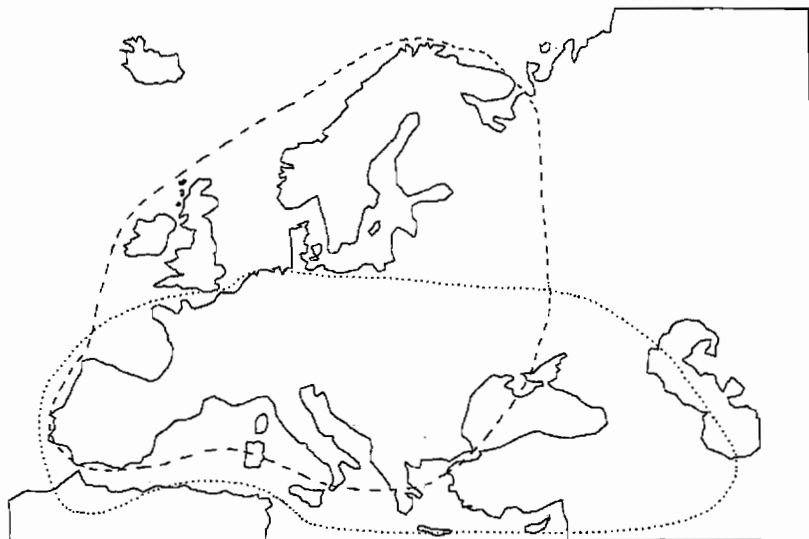


Fig. 1.1. European distribution of the poplar rust fungi *Melampsora larici-populina* (dashed line) and *M. allii-populina* (dotted line). (After Somda & Pinon, 1981.)

There have been similar outbreaks in southern England following the importation of these clones for commercial use.

As shown by Somda & Pinon (1981), *M. allii-populina* is more thermophilic than *M. larici-populina* at some stages of its life cycle – especially urediniospore germination – and, as a wind-dispersed foliar pathogen, it can become prevalent north of its usual range during years with warmer than average temperatures. Other poplar rusts are also quite temperature-sensitive, including one, *M. medusae*, which has been accidentally imported into south-west France and which has so far shown no sign of spreading from this region into other climate zones. However, the possibility of future climate change has important implications for poplar breeding programmes, as well as for plant quarantine controls.

The example of *M. medusae* raises a further issue in relation to certain pathogens which are climatically confined to certain regions but which could find suitable conditions elsewhere, if they were able to ‘vault’ natural geographical barriers such as mountain ranges. Barriers can, of course, already be circumvented by human interference, as has been the case with the introduction of *M. medusae* into Europe and with the recent appearance of *M. larici-populina* in North America (Newcombe & Chastagner, 1993). However, under conditions of climate amelioration,

some barriers might become less of an obstacle, allowing 'natural' spread to extensive areas formerly outside the geographic ranges of the fungi concerned.

The accidental transfer of plant pathogens to new regions of the world is always of concern but could become more significant in the face of climate change. An example of particular importance is that of *Phytophthora cinnamomi*, an oomycete that appears to have originated in the Pacific Celibes region (and perhaps also South Africa), but which now also occurs over wide areas of Australasia, North America and Europe (Brasier & Scott, 1994). This fungus has an extremely wide host range, causing a root and stem-base disease of broadleaved and coniferous trees. It has caused very severe damage to some Australian eucalypt and heathland ecosystems, and is also associated with major declines of Iberian oak forests (Brasier, 1992). Although the fungus can grow at 5°C, and now occurs in areas representing a very wide climatic range, its pathogenic activity is confined mainly to sub-tropical and warm-temperate climates. In cool-temperate maritime areas, such as Britain, it causes occasional disease and could be expected to become more prevalent with global warming (Fig. 1.2) (Brasier & Scott, 1994). This expectation is based both on the direct response of the fungus to temperature, which has been experimentally modelled using the 'CLIMEX' model for climate matching (Sutherst, Maywald & Bottomley, 1991) and also to changes in soil moisture and to the incidence of drought-induced susceptibility in the host. The question of climate change affecting host susceptibility will be considered in a later section of this chapter.

Effects on the reproduction and dispersal of pathogens

A wide range of pathogens, especially those that infect leaves or green shoots, show large annual fluctuations in their incidence and severity of attack, and these events can often be attributed to weather conditions. Many fungi are favoured by moist conditions during the growing season, due to an enhancement of spore production and, in many cases, dispersal by rain-splash. Others, such as some of the powdery mildews, are favoured by low humidity. Winter conditions are also important in determining the success of the saprotrophic survival of many leaf-infecting fungi.

In view of the effects of existing weather fluctuations on the severity of various foliar diseases, climate change could be expected to affect their

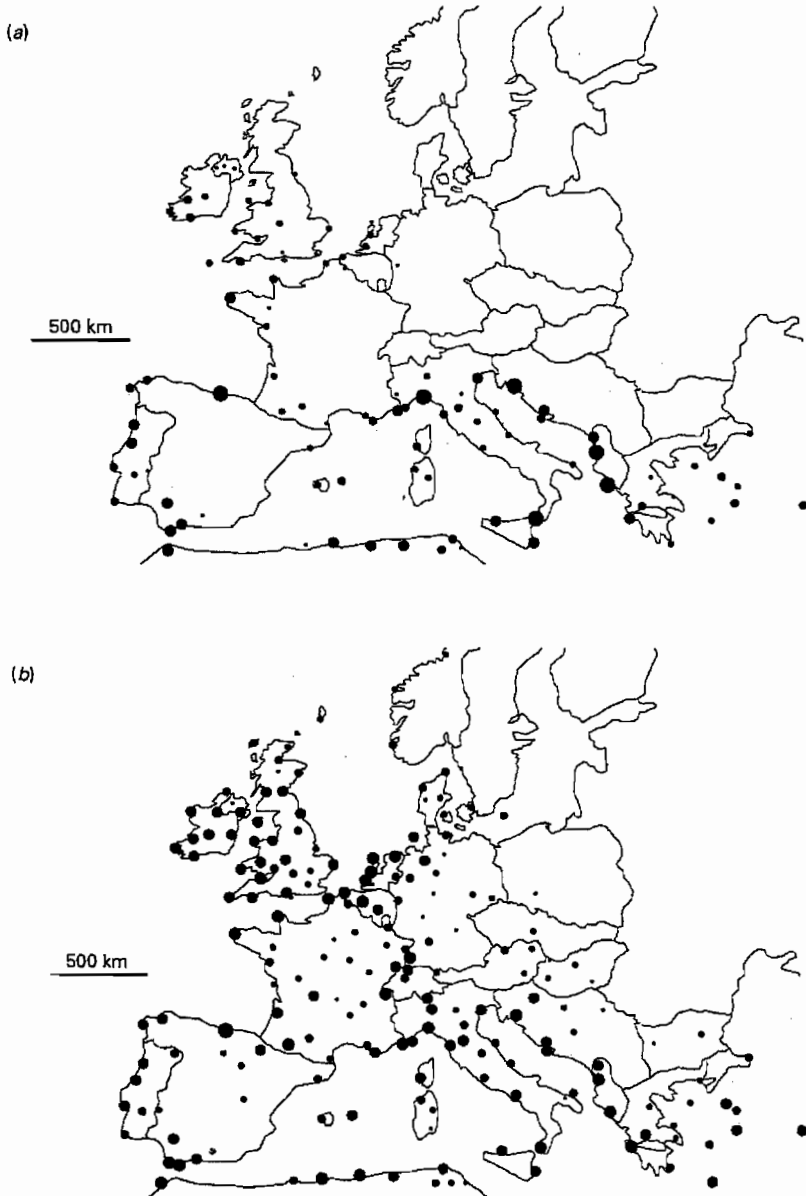


Fig. 1.2. (a) Current activity of *Phytophthora cinnamomi* in Europe, estimated from its distribution and present-day climate; (b) activity of the fungus predicted after a warming of 3 °C. Dot-size signifies relative suitability of climate for survival and growth of the fungus. Maps pre-date recent national boundary changes. (After Brasier & Scott, 1994.)

relative prevalence in the long-term. A reduction in the number of rain-days in the summer might, for example, decrease the dispersal of many leaf-spot fungi such as *Marssonina* species on poplars (Cellerino, 1979) and *Cristulariella pyramidalis* on black walnut (*Juglans nigra*) (Neely, Phares & Weber, 1976). However, wetter weather in the spring might encourage infection early in the growing season. Any predictions must be regarded as speculative, owing to the complex effects of climate on annual cycles of disease. There are, additionally, many microbial interactions that involve fungal pathogens, and the effects of climate change on these are virtually unpredictable.

Effects on the activity of pathogens in winter

In the dormant season, the host's physiological responses to temperature and day-length may to some extent inactivate its defensive reactions, but the temperature can be high enough to allow the pathogen to remain active. Thus, there are many diseases caused by weak parasites which develop mainly at this time of year.

Although many diseases are known to develop mainly in the dormant season, there are few quantitative data which demonstrate the onset or cessation of pathogenesis. One interesting example comes from the work of van Vloten (1952) on the bark-killing pathogen *Phacidium coniferarum* (syn. *Phomopsis pseudotsugae*). In Japanese larch, *Larix kaempferi*, this fungus can invade wounds made during the winter months, such as can be created by pruning operations, then developing until it is checked by the onset of renewed cambial activity in spring (Fig. 1.3).

Working in a stand of 13-year-old Japanese larch, van Vloten (1952) made wound inoculations with *P. coniferarum* at monthly intervals and measured the size of the resulting lesions. As the results in Fig. 1.4 show, the size of the lesions following dormant-season inoculation was proportional to the length of time available to the fungus for host invasion before the onset of the growing season. This work was conducted in the relatively maritime climate of Wageningen in the Netherlands during the mild winters of 1949/50 and 1950/51. The results might have been rather different during colder winters when the limiting effects of low temperature on the fungus might have been important.

For pathogens like *P. coniferarum* that have little ability to overcome host resistance during the growing season, winter temperature is likely to be critically important. In climates where temperatures are too low during most of the dormant season to allow such fungi to grow within host

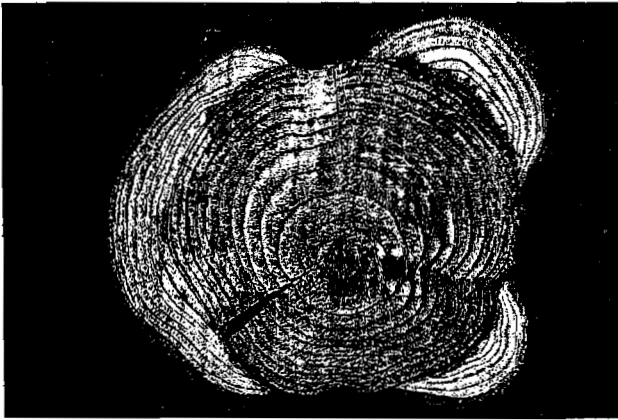


Fig. 1.3. Canker of Japanese larch caused by *Phacidium coniferarum*, a dormant-season pathogen.

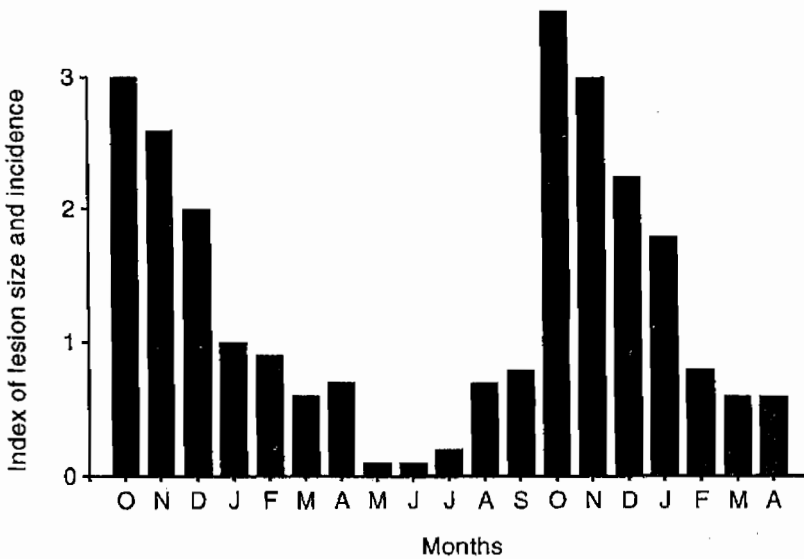


Fig. 1.4. Stem lesions induced by the dormant-season pathogen, *Phacidium coniferarum*, on Japanese larch inoculated at different seasons: bars represent an index of canker incidence and length. (After van Vloten, 1952.)

tissue, there is little opportunity for them to cause disease. However, there are geographic zones, mainly in temperate latitudes, in which winter dormancy of woody plants coincides with periods when temperatures are high enough for fungal activity. Thus, in these zones of 'asynchronous

dormancy', disease can be caused by fungi which would otherwise be largely non-pathogenic.

The poleward boundaries of the 'asynchronous dormancy zone' will obviously differ for different host-pathogen combinations. However, the 2°C isotherm for January in the Northern Hemisphere, and for July in the Southern Hemisphere (adjusted to sea-level) provide a possible demarcation for most diseases, although a more realistic line would need to be based on a detailed analysis of temperature records throughout the winter. Also, there are many cold upland regions within the zone which should be excluded from it. A suggested global 'asynchronous dormancy zone' based on the 2°C isotherm is shown in Fig. 1.5. Towards the equator, this zone is shown as including all regions with a distinct winter (that is, with the coolest monthly mean below 18°C). However, it could in reality be much narrower since, as shown here, it includes the subtropical zones, where there are many evergreen tree species, including some which should perhaps not be regarded as showing winter dormancy. This uncertainty applies particularly in much of the Southern Hemisphere, where genera such as *Eucalyptus* and *Nothofagus* are often dominant.

According to a recent computer-generated model for the period 2058–2067 (Anon, 1992), global warming would cause the 'asynchronous dormancy zone' in each hemisphere to migrate slightly and irregularly polewards (Fig. 1.5). In Europe, it would also expand somewhat eastwards. These projected shifts in the zone are superimposed on the existing situation depicted in Fig. 1.5. The changes appear to be fairly small, compared with other effects such as the increased incidence of summer drought that could occur in many mid-latitude regions. However, the range of fungi which could respond to a 'window of opportunity' in mild winters is considerable, since it would include many of the commonest causes of stem cankers and shoot diebacks.

Even in regions of the world where winters are colder than in the zone demarcated in Fig. 1.5, certain fungi have evolved the ability to invade host tissues in the winter. Temperatures rarely fall far below freezing under snow cover, and certain low-temperature pathogens termed 'snow-moulds' have exploited this phenomenon. An example of such a fungus is *Phacidium infestans*, which attacks the needles and shoots of various conifers while they are covered by snow. The snow moulds appear to require the persistently high atmospheric humidity that occurs beneath snow cover, as well as insulation from extreme cold (Björkmann,

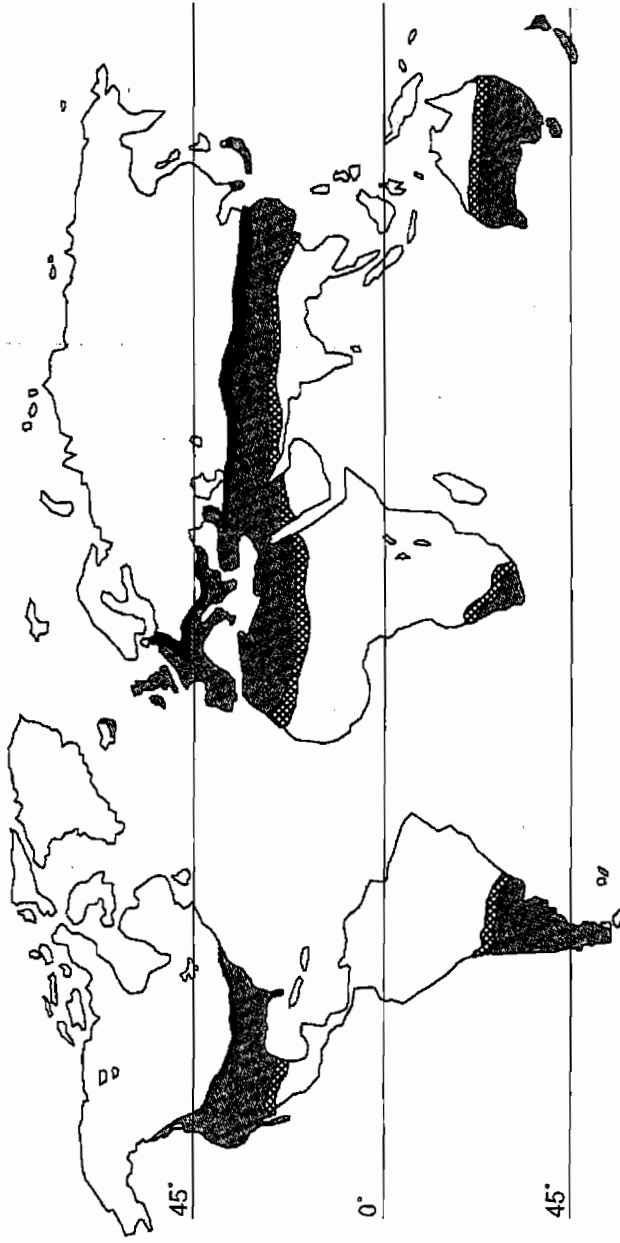


Fig. 1.5. The 'asynchronous dormancy zone' (ADZ) and its possible migration under predicted conditions of global warming for the mid-21st century. *The land areas with the lighter stipple together with their cross-hatched borders represent the 'ADZ', where bark and xylem fungi are often active during winter dormancy of their hosts. The cross-hatched borders represent a possible poleward extension of tropical zones, in which no distinct winter occurs. White land areas together with their darkly stippled borders represent tropical and cold winter zones; in the tropical zones neither hosts nor pathogens have periods of winter inactivity, while in the cold zones trees and most pathogens are simultaneously inactive. The darkly stippled borders represent a possible poleward extension of the 'ADZ'.*

1948), and so do not usually cause disease in mild climates where snow is absent from the host surface for most of the winter.

The incidence of damage due to snow moulds can be expected to change in response to global warming, since it is likely to involve changes in the amount and persistence of snowfall. This would probably be more important than change in temperature. However, it is interesting to consider the case of an important disease of conifers variously known as *Brunchorstia dieback* or *Scleroderris shoot blight*, in which temperature data have been recorded in relation to disease development beneath snow cover. The causal organism is an ascomycete, the teleomorph now most commonly being known as *Gremmeniella abietina* and the anamorph as *Brunchorstia pinea*. In pines, spore infection occurs as the shoot elongates in spring, but the fungus then ceases to develop until the end of the host's growing season, being confined to the dead cells of the epidermis and hypodermis of the shoots and the dead part of the bud scales (Siepmann, 1976). Once the growing season has ended, host invasion can begin, resulting in death of the shoots and buds by the following spring.

Marosy, Patton and Upper (1989) conducted an experiment on *Scleroderris shoot blight* at two locations in Wisconsin where seedlings of Red pine (*Pinus resinosa*) that had been inoculated in early summer were overwintered either under snow or without snow. The data in Table 1.1 show that there was far more disease in the seedlings kept under snow than in the others. The authors related this to the higher temperatures to be found there: the range below snow was between -6 and 0°C whereas

Table 1.1. *Effect of snow cover on the incidence of Gremmeniella abietina infection of artificially inoculated Red pine seedlings in Wisconsin*

	Percentage infection	
	With snow	Without snow
1984/85 experiment		
Blackhawk	76	0
Copper Falls	48	0
1985/86 experiment		
Blackhawk	83	36*
Copper Falls	62	9

Note: *In this year the seedlings in the no-snow plots spent the winter encased in ice as a result of a fall of wet snow in November.

Source: After Marosy, Patton & Upper, 1989.

the plants in the no-snow plots were exposed to temperatures as low as -36°C . From this and other work, Marosy *et al.* (1989) developed a concept of 'conductive days', which was based on the idea that a certain number of days between -6°C and $+5^{\circ}\text{C}$ is required for disease expression. In winters and locations with insufficient conductive days, the amount of disease would be greatly reduced.

Studies in Japan, where Scleroderris shoot blight occurs on *Abies sachalinensis*, also point in the same direction. Certainly it is known that, once the trees are tall enough for their tops to be above normal snow levels, attacks by *G. abietina* cease to be a problem.

It should be noted that, in the American studies, the upper temperature limit of 5°C was chosen arbitrarily to exclude days in spring and autumn when the host might be physiologically active and thus perhaps able to resist attack. In the winter, such temperatures are rare in Wisconsin, which has a continental climate, but are common in Britain.

Effects of climate change on the host

Increased climatic stress in the growing season

The evidence from diseases involving native hosts and pathogens suggests that pathogenesis can be exacerbated by stresses induced by extremes of weather, especially drought during the growing season. A classic example of this is the root disease caused by the honey fungus, *Armillaria* spp. Members of this basidiomycete genus are ubiquitous in long-standing woodland, existing in the soil as complicated networks of rhizomorphs sustained by food-bases in the form of colonised stumps and other woody debris. The fungus can establish numerous points of infection on the roots of healthy trees, from which it can extend further into host tissue, sometimes eventually overwhelming the host. Some members of the genus, such as *Armillaria mellea* and *Armillaria ostoyae*, are highly pathogenic (Rishbeth, 1982) but even they may be assisted by the impairment of host resistance through stress from drought and other causes (Wargo, 1984; Rishbeth, 1991). Invasion by less pathogenic species such as *Armillaria gallica* appears to be almost entirely dependent on host stress (Rishbeth, 1982) although it can sometimes be sustained indefinitely after the stress has abated. With an increased frequency of summer drought, therefore, damage caused by *Armillaria* spp may be expected to become more prevalent.

Another disease process that is very much linked to drought stress is the formation of strip-cankers, associated mostly with ascomycetes of the families Xylariaceae and Diatrypaceae. A number of studies (Bassett & Fenn, 1984; Carroll, 1988; Chapela & Boddy, 1988; Hendry, 1993) have indicated that such fungi can exist for many years in the healthy xylem of various broadleaved trees as latent invaders or as endophytes until drought stress, or other damage to the host, allows them to extend by pathogenic growth within the sapwood and into the overlying bark. This pathogenesis is dramatically revealed by the production of very extensive stromatic fruiting structures on the surfaces of the branches or stems that these fungi have helped to kill. An important example is that of *Biscogniauxia mediterranea*, which has caused serious damage to oak species such as *Quercus cerris* and *Q. suber* following droughts in southern Europe (Estanyol & Molinas-de-Ferrer, 1984; Vannini, 1987). In Britain, similar strip cankers on beech (*Fagus sylvatica*) are caused by *Biscogniauxia nummularia* and *Eutypa spinosa* (Lonsdale, 1983; Hendry, 1993), and these were particularly prevalent in southern Britain after the 'double' drought years of 1975/76, 1983/84 and 1989/90.

With climate change in mind, it is interesting to look at the European distributions of *B. mediterranea* and *B. nummularia*. The former causes strip-cankers only in relatively warm climates, such as occur in the Mediterranean countries, while the latter is common as far north as southern Britain but becomes rare in the cooler and damper north and west of Britain, and has its northernmost outposts in southern Scandinavia, where its host is *Prunus* rather than *Fagus* (Granmo *et al.*, 1989). Interestingly, *B. mediterranea* has also been found in southern Britain, though not as the cause of a strip-canker (Spooner, 1986). Increased summer temperatures and droughtiness could be expected to help shift the distributions of these fungi northwards within the range of potential hosts, or at least to increase the geographic range over which they behave as pathogens.

Another interesting stress-related disease akin to the strip cankers is sooty bark disease of sycamore, *Acer pseudoplatanus*, caused by *Cryptostroma corticale* (Fig. 1.6). Here again the fungus is latent or endophytic within the tissues of the healthy tree (Bevercombe & Rayner, 1984; J.N. Gibbs & J. Rose, unpublished observations). In hot dry summers, it can rapidly develop within the xylem and subsequently within the bark (Young, 1978; Dickenson & Wheeler, 1981). Curiously, this disease has been found only in south-east England and northern France (J.N. Gibbs & J. Rose, unpublished observations), even though

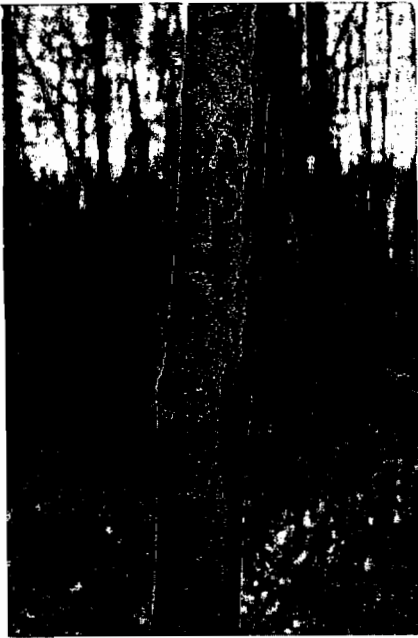


Fig. 1.6. Sooty bark disease of sycamore, caused by *Cryptostroma corticale*, a pathogen dependent on host stress.

sycamore occurs as a native over a much wider area of Europe, including the mountains of the south.

In forests where a more frequent incidence of summer drought exacerbates stress-related diseases, there can be a resulting increase in tree mortality which leads to an opening up of the canopy structure. This, in turn, enhances transpirational stress in the remaining trees, due to their increased exposure to insolation and wind.

An exception to the examples described so far appears at first sight to be provided by Dutch elm disease (DED), in which drought conditions suppress the development of foliar symptoms in both the English elm, *Ulmus minor*, and the American elm, *Ulmus americana* (Gibbs & Greig, 1977; Smalley & Kais, 1966; C.M. Brasier, personal communication). However, when in such cases the xylem has been examined, the presence of extensive vascular streaking has indicated that fungal invasion was not prevented, even though foliar symptoms were suppressed (C.M. Brasier, personal communication). This suppression of wilting may be due to stomatal closure during periods of water stress, but there remains some possibility that the fungus could be directly affected by the heat associated with summer droughts. The aggressive DED pathogen,

Ophiostoma novo-ulmi, has a temperature optimum for growth of only *c.* 20–22 °C, as compared with *c.* 30 °C for *Ophiostoma ulmi*, the non-aggressive pathogen (Brasier, Lea & Rawlings, 1981). It is also possible that vascular wilt fungi have a limited ability to colonize drought-stressed trees because of the presence of vessel cavitation (Zimmerman, 1983) which could obstruct their normally rapid invasion of hydraulically functional sapwood.

Effects of altered winter temperatures on the host

During winter dormancy, direct effects of climate on the host are generally less important than those involving the pathogen. However, frost damage has been reported to encourage the development of certain fungal cankers. Examples for which this has been demonstrated include Botryosphaeria canker of rowan, *Sorbus aucuparia*, caused by *Botryosphaeria dothidea* (Wene & Schoeneweiss, 1980) and canker of *Pinus resinosa* caused by *Diplodia pinea* (Palmer, 1991). In areas such as Britain, where climate-change modelling predicts a decrease in the incidence of frost (Anon, 1992), diseases of this type could become less prevalent.

Effects involving interactions between trees or pathogens with other organisms

Interactions with vector organisms

The requirement for an insect or other vector in certain fungal diseases complicates predictions about the possible effects of climate change on the geographic range of disease incidence. Dutch elm disease provides an interesting example, since *Scolytus scolytus* and *Scolytus multistriatus*, the most important of the beetle vectors in western Europe, do not readily fly at a temperature below about 22 °C (Fairhurst & King, 1983) and are therefore less able to act as vectors in cool conditions (Redfern, 1977; Harding & Ravn, 1982). In some parts of the range of elm species, this temperature requirement may have delayed the northward extension of the epidemic until the occurrence of warm summers in particular years and the build-up of beetle breeding habitats in the outbreak areas.

In regions where a pathogen already occurs, weather conditions may favour outbreaks of its vectors in certain years, suggesting that climate change could influence the long term prevalence of the disease. An

interesting case is that of the fungus *Ceratocystis laricicola* (Redfern, Stoakley & Steele, 1987), which has been described only in recent years. It infects the bark, cambium and sapwood of larch trees, causing death and dieback, and appears to be transferred between trees by an insect vector, the bark beetle *Ips cembrae*. Redfern *et al.* (1987) found that this disease occurred mainly in areas affected by drought, which favours bark beetle attacks by inducing host stress.

For many fungal diseases involving vectors, the effects of climate and weather on the development of outbreaks and epidemics have not been studied in detail. However, the importance of such effects is to some extent self-evident, since the geographic ranges of insect vectors are determined largely by climate, while their activity and abundance are influenced by the vagaries of weather. Climate change, involving an increase in temperature or in the incidence of drought, could extend the range of any such diseases into areas where the host and fungus can already both exist but in which vectors are not yet operative.

Effects on mycorrhizas

There is evidence that mycorrhizal fungi can protect trees against certain root pathogens (Marx, 1970; Chakravarty & Unestam, 1987; Buscot, Weber & Oberwinkler, 1992), and that some species can be much more effective than others in this respect (Malajczuk, 1988). The incidence of certain root diseases may therefore be influenced by the outcome of inter-specific competition between mycorrhizal fungi. Periodic drought tends to provide opportunities for competitive replacement within mycorrhizal communities because it leads to the death and subsequent regeneration of non-woody roots, and thus to the loss and renewal of mycorrhizal associations. Although there appear to have been no studies involving the effects of mycorrhizal replacement on the suppression of disease, replacement *per se* has been observed following environmental change. For example, when seedlings of *Pinus caribaea* were planted in the dry zone of Sri Lanka, their mycorrhizal symbiont, a *Boletus* sp., was totally replaced by the drought-tolerant mycorrhizal fungus *Cenococcum* sp. (Muttiah, 1972). Mycorrhizal replacements might occur not only in seedlings planted out in a new site, but also in established trees exposed to an increased frequency of drought, as has happened during recent years in some countries. It is interesting to ask whether such an effect might partly explain the reported decline in the occurrence of various mycorrhizal fungi in Europe (Arnolds, 1988; Jansen, 1990).

Summary and conclusions

The effects of possible climate change on fungal diseases of trees can, to some extent, be judged by analysing the existing role of climate and of fluctuations in weather. For pathogens whose geographic ranges or pathogenic activity are clearly affected by temperature, the effects of climate warming are probably predictable. These pathogens include both those that are favoured by relatively high summer temperatures and also those that require mild temperatures in the dormant season because it is only then that they can attack the host.

The effects of any increase in the frequency of summer droughts is also reasonably predictable, since the role of host stress in allowing attack by many pathogens, especially root pathogens, is well known. In particular, it can be predicted that such a climate change would alter the stability of associations between tree species and various members of their endophytic mycofloras, some of which would be triggered more frequently into curtailing such associations through pathogenesis.

Prediction is more difficult in the case of pathogens whose reproduction and dispersal is strongly affected by rainfall and atmospheric humidity. This is also true of pathogens that are strongly affected by interactions with other organisms, such as insect vectors or protective mycorrhizal fungi.

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