

1

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



### 2 D. Lonsdale and J.N. Gibbs

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).



## Climate and fungal tree diseases

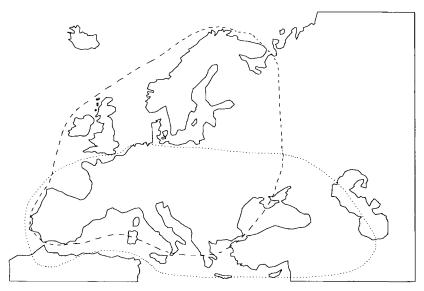


Fig. 1.1. European distribution of the poplar rust fungi *Melampsora larici-populina* (dashed line) and *M. allii-populina* (dotted line). (After Somda & Pinon, 1981.)

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,

3



#### 4 D. Lonsdale and J.N. Gibbs

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

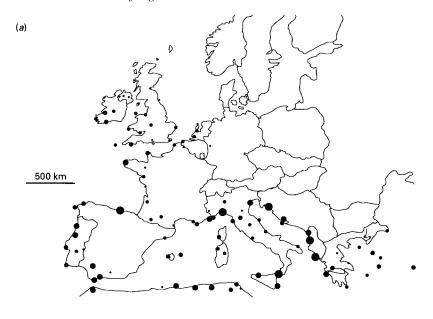
A wide range of pathogens, especially those that infect leaves or green shoots, show large annual fluctuations in their incidence and severity of attack, and these events can often be attributed to weather conditions. Many fungi are favoured by moist conditions during the growing season, due to an enhancement 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



Climate and fungal tree diseases





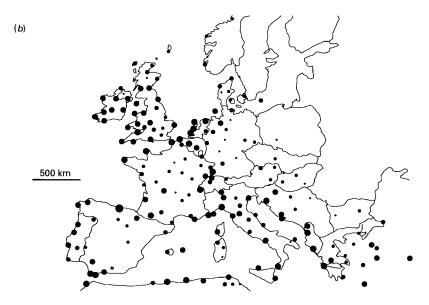


Fig. 1.2. (a) Current activity of *Phytophthora cinnamomi* in Europe, estimated from its distribution and present-day climate; (b) activity of the fungus predicted after a warming of 3 °C. Dot-size signifies relative suitability of climate for survival and growth of the fungus. Maps predate recent national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



#### 6 D. Lonsdale and J.N. Gibbs

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



## Climate and fungal tree diseases

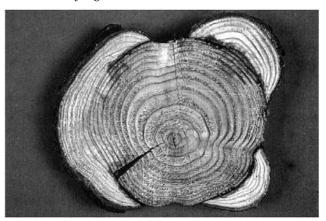


Fig. 1.3. Canker of Japanese larch caused by *Phacidium coniferarum*, a dormant-season pathogen.

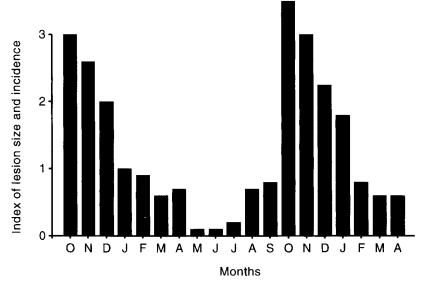


Fig. 1.4. Stem lesions induced by the dormant-season pathogen, *Phacidium coniferarum*, on Japanese larch inoculated at different seasons: bars represent an index of canker incidence and length. (After van Vloten, 1952.)

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

7



### 8 D. Lonsdale and J.N. Gibbs

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

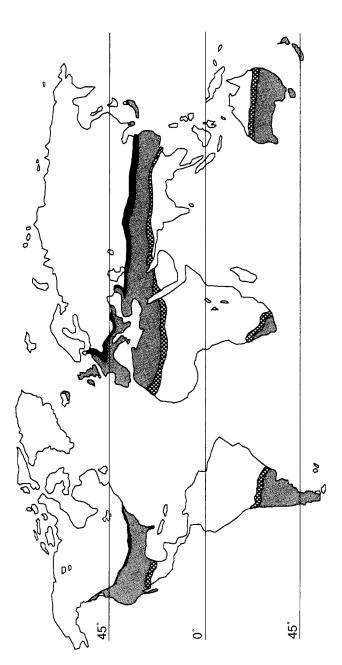
The poleward boundaries of the 'asynchronous dormancy zone' will obviously differ for different host-pathogen combinations. However, the 2 °C isotherm for January in the Northern Hemisphere, and for July in the Southern Hemisphere (adjusted to sea-level) provide a possible demarcation for most diseases, 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,



More information



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 winter inactivity, while in the cold zones trees and most pathogens are simultaneously inactive. The darkly stippled borders Fig. 1.5. The 'asynchronous dormancy zone' (ADZ) and its possible migration under predicted conditions of global warming or the mid-21st century. The land areas with the lighter stipple together with their cross-hatched borders represent the 'ADZ', stippled borders represent tropical and cold winter zones; in the tropical zones neither hosts nor pathogens haver periods of epresent a possible poleward extension of the 'ADZ'.



#### 10 D. Lonsdale and J.N. Gibbs

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.