18

FACTORS AFFECTING DISEASE DEVELOPMENT

Philip Keane and Allen Kerr

Contents

18.1 The disease triangle

The amount of disease that develops in a plant community is determined by the host, the pathogen and the environment and can be depicted in the form of a disease triangle (Fig. 18.1). A fourth factor, namely 'human interference' (making a disease square) can be added, but, as the other three aspects have a degree of human influence, the disease triangle is sufficient as a framework for discussing the various factors that affect disease. It is important to note the arrows indicating interactions between the various factors in the disease triangle. It is the balance of these interactions that determines whether or not disease develops to destructive levels in a particular situation. Development of epidemics requires the interaction of a highly virulent pathogen and a susceptible plant host in an environment that favours the development of disease. The environment can affect both the susceptibility of the host (e.g. by predisposing it to infection) and the activity of the pathogen (e.g. by providing the conditions of leaf wetness required for spore germination and infection). The pathogen can affect the host and the host can influence the pathogen (e.g. by secreting chemical factors). Similarly, the host can influence the environment (e.g. by influencing the microclimate within the canopy). An understanding of these factors and their interactions for a particular disease in a particular locality allows prediction of disease outbreaks and intervention to reduce the amount of disease.

18.2 Pathogen factors

The main factor which determines whether or not disease occurs at all is the presence or absence of a pathogenic strain of the pathogen. In the outbreaks of eucalypt dieback in southern and south western Australia, the presence or absence of the pathogen is the overriding factor that determines whether or not disease occurs. The introduction of the pathogen, *Phytophthora cinnamomi*, into certain forests has resulted in outbreaks of the disease. Similarly the appearance

of new pathotypes of *Puccinia graminis* with virulence on cultivars of wheat that are resistant to the local pathotypes can also result in fresh outbreaks of wheat stem rust.

Quarantine regulations aim at excluding potentially destructive pathogens from Australia, New Zealand and the many other countries of the region which have been geographically isolated from major centres of crop and pathogen evolution in other parts of the world. Unfortunately, every few years a new pathogen finds its way into the region. Every time a quarantine barrier fails and allows entry of a pathogen which can attack a commercially important plant species, much of the competitive advantage of growing that species in a particular region is lost.

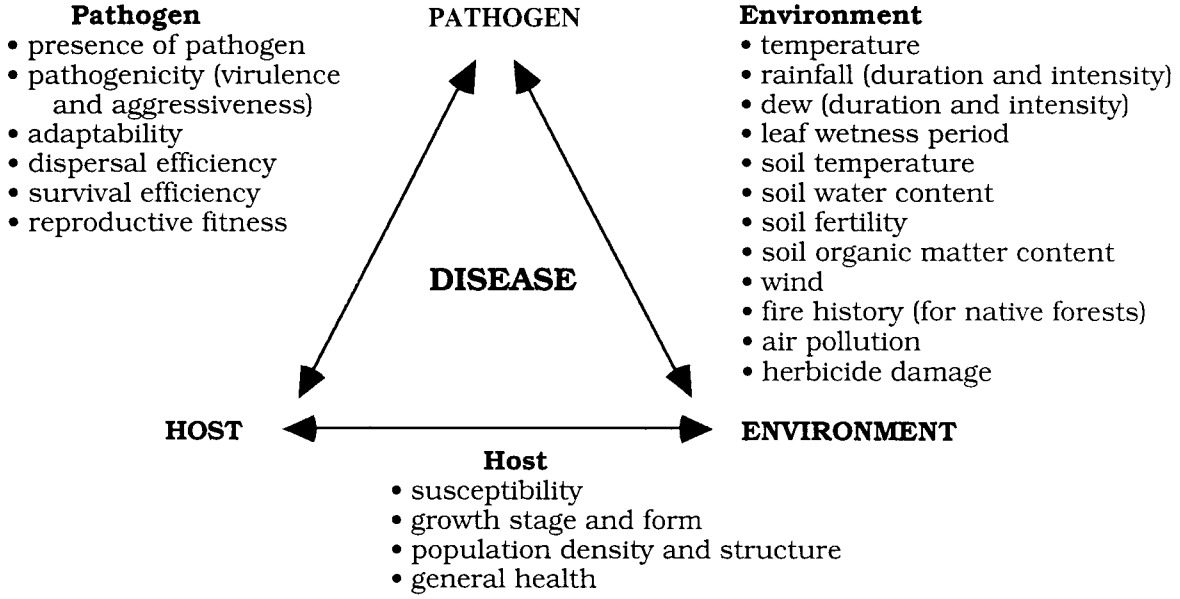

Figure 18.1 A generalised disease triangle showing the factors that affect the occurrence of a plant disease. Arrows show the possible interactions between these factors.

Occasionally, new encounter pathogens occur on crops introduced to a region. When cocoa was introduced to Papua New Guinea and South East Asia, *Oncobasidium theobromae*, probably a pathogen or endophyte of a native plant, infected the introduced cocoa tree, resulting in the occurrence of a serious new disease called vascular-streak dieback.

The amount of disease that develops is often determined by the **pathogenicity** of the prevalent population of the pathogen. The term pathogenicity comprises both the virulence of the pathogen (its ability to infect cultivars which have certain resistance genes) and its aggressiveness (the vigour with which it infects cultivars without resistance genes or cultivars whose resistance genes are not activated by the pathogen). Pathogenicity is one aspect of the reproductive fitness of a pathogen, which also includes aspects of dispersal and survival fitness. Control of diseases often involves monitoring the pathogenicity of the prevailing pathogen population as a guide to the breeding of resistant host cultivars.

The **adaptibility** of a pathogen is very important in determining its ability to overcome resistance in newly released cultivars or to adapt to changed environmental conditions (e.g. the introduction of irrigation or the planting of a crop only in drier parts of its potential range). Adaptability is governed by the genetic flexibility of the pathogen population and its reproductive efficiency. *P.*

graminis has high reproductive efficiency and, in spite of the absence of a sexual reproductive cycle in Australia, has the potential to develop new pathotypes by asexual means such as mutation, heterokaryosis and parasexualism. In contrast, *O. theobromae* on cocoa in Papua New Guinea has a much lower reproductive capacity and so is likely to adapt more slowly.

The ability of a pathogen to cause destructive disease epidemics depends on its ability to **disperse** rapidly over long distances. Herein lies much of the destructive potential of cereal rusts. The urediniospores of rusts can be blown vast distances in a few days, allowing epidemics to develop rapidly over very large areas. Nematodes and soil-borne pathogens, on the other hand, have limited dispersal ability and tend to cause localised disease outbreaks.

The ability of a pathogen to cause disease in successive seasons depends on its **ability to survive** from one season to the next. This is especially important for soil-borne pathogens. Some smut fungi and *Plasmodiophora brassicae*, the cause of club root of crucifers, form spores that enable the pathogens to survive in the soil for many years, thus reducing the effectiveness of disease control using crop rotation. The sclerotia of *Sclerotium rolfsii* and *Sclerotinia* spp. are also particularly effective in enabling these pathogens to survive for long periods in soil. The survival of cereal rusts between growing seasons is a critical factor in determining the likelihood of epidemics. A wet summer allows the growth of volunteer cereal plants on which the rust can survive through the summer and autumn, resulting in larger quantities of inoculum to initiate epidemics in the new growing season.

Pathogenicity, adaptability, dispersal and survival efficiency contribute to a fundamental property of the pathogen that determines the amount of disease, namely the **number of infective propagules** (e.g. fungal spores or sclerotia, bacterial cells, nematode eggs, individual nematodes, virus-carrying insects) available to infect plants. The most common relationship between the number of propagules and disease incidence is a straight line which levels off at high disease incidence (Fig. 18.2A). As the number of propagules increases, the amount of disease increases, but it increases more slowly as the number of uninfected plants or the amount of uninfected tissue decreases at higher levels of disease. This curve is similar to the basic disease progress curve used by Van der Plank in 1963 to describe disease epidemics (Fig. 18.2B and C). Disease progress curves basically describe a situation in which the amount of disease increases with the passage of time, again plateauing at higher levels of disease as the amount of tissue available for further infection declines.

Many of the other factors that influence disease development do so by affecting the number of propagules produced by the pathogen and/or their ability to infect the host (**infection efficiency**). In particular, environmental factors affect the survival of propagules, influencing the number of propagules available to initiate an epidemic and the proportion of these propagules that eventually initiate infection of the host.

18.3 Host factors

The main host factor affecting disease development is the occurrence of individuals in the host population that are **susceptible** to the particular pathogen. For a disease epidemic to occur, the host plant population must be largely susceptible to attack by the pathotypes of the pathogen in the vicinity. For example, in many forests in southern Australia, only certain species of eucalypt are susceptible to *P. cinnamomi*. Dieback is rare in forests consisting largely of eucalypts in the sub-genus Symphyomyrtus (smooth-barked species).

The most important means of disease control is to plant host species or cultivars that are not susceptible to the prevailing population of the pathogen of concern. Wheat crops consisting of cultivars bred for resistance to the most common pathotypes of stem rust are little affected by the disease.

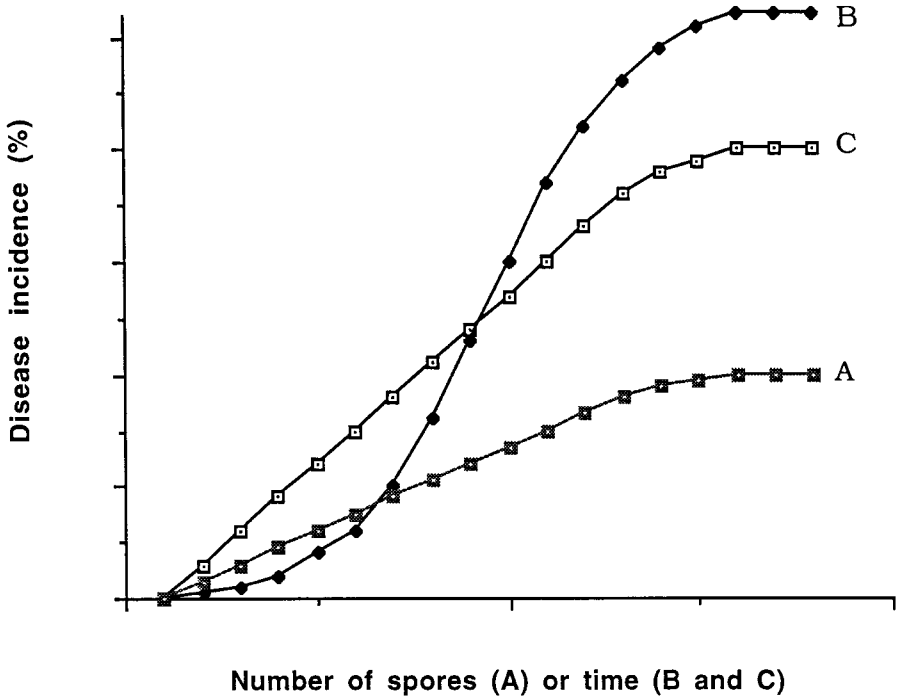

Figure 18.2 Dose response and epidemic progress curves. (A) The most common type of disease–inoculum response curve (or dose response curve) showing a decline in disease incidence at higher inoculum levels due to the reducing proportion of healthy tissue available for infection. Epidemic progress curves for pathogens that do (B) and do not (C) increase the amount of inoculum during the growing season of the crop.

The **growth stage** and **form** of the host can greatly influence the occurrence of disease. Some diseases such as damping-off are more common in seedlings, while others are characteristic of mature plants. The growth stage also determines the degree of closure of the canopy, which in turn affects the microclimate within the canopy. As the crop grows, foliage in adjacent rows meets and retains a more humid atmosphere under the canopy for much longer periods following rain (Fig. 18.3). Different cultivars of the same crop can produce different microclimates. The dense growth of dwarf wheat and rice cultivars produces higher relative humidities within the crops than occurs with the tall cultivars of the same plant. The incidence of splash-dispersed pathogens such as *Mycosphaerella graminicola*, the cause of speckled leaf spot of wheat, increased when semi-dwarf wheats replaced tall cultivars in the 1970s. The greater disease incidence in the shorter wheats resulted from two factors. The shorter distance between nodes resulted in the leaves being closer together. This changed the microclimate within the canopy leading to greater humidity and a longer period of leaf wetness favouring infection. In addition, the shorter distance between leaves increased the probability of infection by the splash-dispersed spores of the fungus. In dwarf cultivars the foliage is also closer to the soil which is the source of inoculum for some diseases.

Even if a host is susceptible to a pathogen, the characteristics of **population structure and density** in the host community will have a large bearing on the development of disease in the community. In particular the density of the main

host species and the proportion of non-hosts interspersed through the plant community will determine the rate and extent of disease epidemics. Many plant diseases are much more common in dense than in sparse plantings. Most human diseases are also of this type. Pathogens usually become more prevalent in larger, crowded host populations and the term 'crowd disease' is used to describe this type of disease. Increased density partly explains why diseases are often more serious in crowded agricultural or silvicultural plantings than in natural, diverse vegetation. The difference between disease incidence in natural vegetation and cultivated crops can also be partly explained by the way pathogens spread under the different conditions. In natural vegetation, individual hosts are widely spaced and separated by other species that interfere with the dispersal of pathogen propagules from host to host. Modern intensive agriculture and plantation forestry provide an ideal situation for development of plant disease because a cultivar or even clones derived from a single plant are commonly planted over a large area with few or no individuals of other (non-host) species or cultivars to interfere with inoculum dispersal.

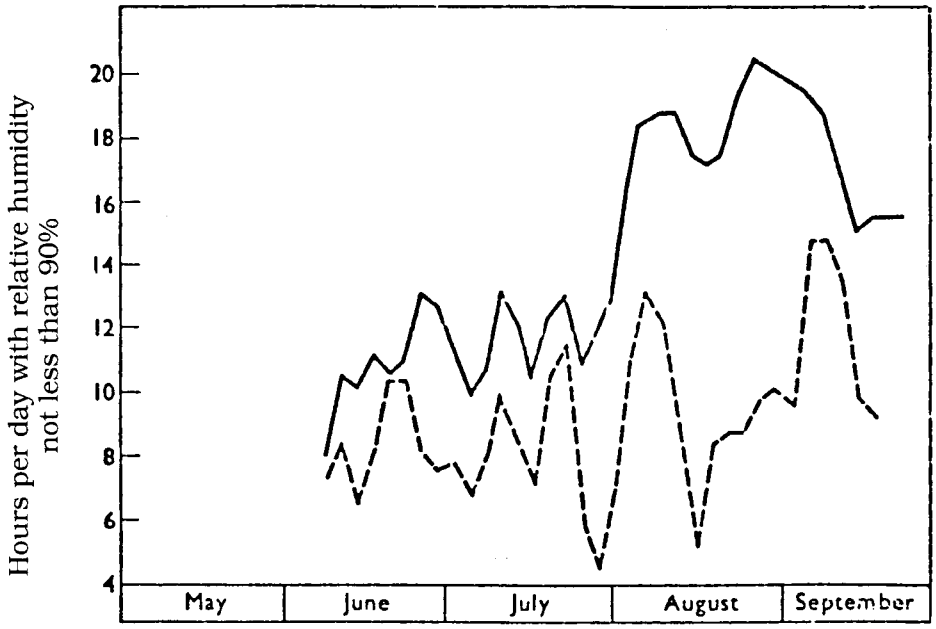

Figure 18.3 Hours per day when the relative humidity is 90% or greater within (——) and outside (- - - -) a growing potato crop. (After Hirst, 1958.)

Spectacular disease epidemics have occurred when susceptible host cultivars were planted over large areas or when a new pathogen was introduced to such plantations. Such a situation occurred with the introduction of wheat stripe rust, *Puccinia striiformis*, to southern Australia in 1979 and with the introduction of poplar rusts, *Melampsora medusae* and *Melampspora larici-populina*, to Australia in 1972 and 1973 respectively. These pathogens encountered monocultures with very little resistance and caused destructive epidemics. In recent years the use of more resistant cultivars has largely controlled wheat stripe rust. A similar situation occurred with the spread of *Phytophthora infestans* to Ireland in the 1840s and with the spread of coffee rust (*Hemileia vastatrix*) to Ceylon in the 1870s. Both pathogens encountered extensive monocultures of their respective hosts, potato and coffee, and caused destructive epidemics.

The epidemic in the USA in 1970 of southern corn leaf blight caused by *Cochliobolus heterostrophus* (anamorph = *Bipolaris maydis*) is an example of how the widespread planting of a susceptible host can result in a sudden and destructive disease epidemic. The basic causes of the epidemic were an

unexpected increase in the susceptibility of the host over a very large area, resulting from genetic manipulation of the plant and adaptation of the pathogen to the altered host (discussed in more detail in Chapter 1).

In many traditional agricultural systems (e.g. in Papua New Guinea), mixtures of many crop species and cultivars are planted, especially in the lowlands where conditions are ideal for pest and disease development. These mixed cropping systems suffer less disease than monocultures established under the same environmental conditions. For example, in Papua New Guinea and the Solomon Islands, taro blight caused by *Phytophthora colocasiae* is more destructive in monocultures of taro established for experimental purposes than in nearby mixed farms.

The prior **health** of the host is often important in determining the occurrence of disease. Necrotrophic pathogens are often more damaging on poorly growing than on vigorous hosts, while the opposite is the case for biotrophs. Older, over-mature trees are more prone to attack by certain leaf pathogens, mistletoes and heart rots, although the degeneration of the trees is also caused by the accumulated effect of these pathogens. Young, actively growing forests suffer very little from the effects of these pathogens, although highly specialised leaf pathogens such as *Mycosphaerella cryptica* and *Aulographina eucalypti* can be common in crowded, vigorously growing young foliage of eucalypts. Eucalypt dieback caused by *Phytophthora cinnamomi* is more common in mature, slower growing forests than in vigorous regrowth. This is partly due to the fact that the vigorously growing forest tends to remove more water from the soil than old forest, thus producing a drier soil environment which is less favourable for the activity of *P. cinnamomi*. Vigorous trees also have a greater capacity than mature trees to replace damaged roots.

18.4 Environmental factors

Environmental factors have traditionally been considered to have the major impact on disease development. Even if a susceptible host and a virulent pathogen are present in a certain locality, a common situation when the farmer has no choice but to plant the particular host, serious disease will not occur unless the environment favours its development. This includes both the aerial and soil (edaphic) environment. Cultural and chemical disease control measures usually involve some manipulation of the environment to make it less favourable for disease development.

Environmental factors may exclude a pathogen from, or greatly reduce its fitness in, a particular part of the potential range of a crop. This has been used as the major method of controlling foliar diseases of wheat in Australia. Beginning with the work of William Farrer in the late 1800s, Australian wheat cultivars have been bred for adaptation to the drier regions of Australia. These environments are less favourable for the leaf-infecting rust and powdery mildew diseases that are the scourge of wheat in Europe. Powdery mildew, *Blumeria graminis*, occurs on wheat sown in the wetter parts of Australia (e.g. coastal regions) but has never been a problem in the drier, inland wheat belts. Stem, leaf and stripe rust are a problem in only about one year in ten, when environmental conditions in the drier wheat belts are favourable for disease development. Australia is fortunate in being able to locate cropping areas to avoid disease, a situation not possible in smaller or more densely populated countries.

The aerial environment

Weather conditions have a great impact on disease development and have been intensively studied as predictors of disease outbreaks (see Chapter 19). Moisture (water availability) is the most important environmental factor influencing disease outbreaks caused by fungi and bacteria. It is important for nematodes also. *Anguina* spp. crawl up the plant in water films on the stems and parasitise above-ground tissue. The term 'moisture' covers rainfall (duration and intensity), relative humidity, dew and leaf wetness (duration and intensity). The influence of rain splash and running water on dispersal of pathogen propagules is also important.

While the two are related, the microclimate on the surface of the plant and especially at the infection court is more important in plant pathology than is the macroclimate in the general atmosphere. Thus, leaf wetness is a more accurate predictor of disease than rainfall and dew period, although these often determine the leaf wetness period and are used as practical predictors of disease because they are easier to measure than leaf wetness. Often the combination of leaf wetness (due to rainfall and dew deposition) and ambient temperature is critical in determining the proportion of pathogen propagules that infect the host. Most air-borne pathogens such as rusts and late blight of potato require a period of free water on the leaf surface for spore germination and infection. The germination and infection process takes time and it is therefore not surprising that the duration of leaf wetness has an important influence on infection. The intensity of leaf wetness is not often considered in disease development studies, but excessively heavy deposits of water on a leaf surface may not be as conducive to infection as a light deposit. The spores of certain pathogens (e.g. wheat stripe rust, *Puccinia striiformis*) may not establish effective contact with a leaf that is too wet. In general, germination and infection by powdery mildews is favoured more by high humidity than by leaf wetness.

The period of leaf wetness required for maximum infection is affected by temperature. Often the leaf wetness period required for a certain degree of infection is longer in cool than in warm weather. Germination and infection are usually accelerated by warmer conditions. A good example of how to quantify the effect of temperature on plant disease is seen in a widely used method of predicting the incidence of apple scab (*Venturia inaequalis*). 'Mills Periods' combine leaf wetness data with temperature data and indicate when infection is likely to occur (see Chapter 19). Longer periods of leaf wetness are required for infection at low than at high temperatures (Table 18.1).

Table 18.1 Some examples of 'Mills Periods' indicating the effect of temperature on the leaf wetness period required for infection by *Venturia inaequalis*.

Mean temperature over period (°C)	5.6	7.2	10	11.7	15
Duration of leaf wetness (h)	30	20	14	11	10

As well as influencing the time necessary for infection, temperature affects the **incubation** or **latent period** (the time between infection and the first appearance of disease symptoms), the **generation time** (the time between infection and sporulation) and the **infectious period** (the time during which the pathogen continues producing propagules). At higher temperatures the disease cycle is speeded up with the result that epidemics develop faster. Under cooler conditions, epidemic progress is usually slower so disease incidence and severity may not reach the threshold levels necessary to cause significant crop loss. With

stripe rust of wheat, a cool temperate rust, disease development slows at excessively high temperatures in late spring/early summer. In addition, the adult plant resistance of many wheat cultivars is expressed more strongly at higher temperatures, which also reduces the rate of epidemic development.

Temperature has a much greater effect on disease development in temperate climates than in the tropics, where temperatures are relatively uniform throughout the year. However, in the tropics diurnal fluctuations in temperature, which are greater than the seasonal fluctuation, do affect plant pathogens. Many pathogens are induced to sporulate at night by the combination of the drop in temperature and the increase in humidity after nightfall.

The great epidemics of late blight of potato in Ireland in the 1840s resulted from the coincidence of a susceptible host, an aggressive pathogen and prolonged periods of warm, moist weather that maintained leaf wetness at ideal conditions for infection by the pathogen. Wet spring weather in Australia greatly favours development of cereal rusts. The period of leaf wetness, varying depending on the temperature, can be used to predict outbreaks in spring of brown rot of stone fruit caused by *Monilinia fructicola*. Such periods, termed **infection periods**, can be used to predict the ideal timing of fungicide sprays to control the disease (Chapter 19). A similar approach to disease control applies to downy and powdery mildews in vineyards throughout southern Australia.

Leaf wetness periods can be affected by wind. Leaves wetted by rain or dew will dry out faster under windy than still conditions. In a tall forest, the leaves in the more exposed upper canopy usually have shorter periods of leaf wetness and suffer less attack from foliar pathogens than leaves in the lower canopy or in the dense canopies of young, regrowth forest.

As well as influencing infection, moisture has an important effect on dispersal of pathogens. Free water or the impact of raindrops facilitates the liberation and dispersal of many fungi and nearly all bacteria. This is a very useful adaptation for a pathogen because when the propagules are dispersed, conditions are also likely to be suitable for germination and infection. For example, many coelomycetes (fungi forming pycnidia or acervuli) produce wet (slimy) spores (gloiospores) that are picked up by rain splash and dispersed. Many dry spores are liberated by the force of impact of raindrops. However, excessively heavy rain may wash spores from the air, reducing their dispersal distance.

Traditionally, environmental conditions in crops have been monitored using a range of mechanical devices, most commonly a thermohygrograph that measures temperature and relative humidity and records the data on a rotating drum covered with paper. Temperature is monitored by movement of a bimetallic strip and relative humidity by calculations from a wet and dry bulb thermometer or by expansion or contraction of a cord that is affected by humidity. A rain gauge has been used to record daily rainfall in traditional weather stations. An early attempt to monitor leaf wetness involved an adaptation of the humidity recorder. The cord was exposed directly to rain and dew and the expansion and contraction of the string was recorded on a rotating drum. The advent of electronic and computer technology has facilitated the continuous recording of microclimatic data that is potentially more useful in plant pathology than the gross weather data provided by weather stations. Temperature can be monitored at several points within a canopy or soil using electronic probes. Leaf wetness can be recorded using artificial leaves consisting of water absorbent fabric containing a wire grid whose resistance to a small electric current is affected by the wetness of the fabric. Rainfall can be monitored continuously by tipping bucket rain gauges. Wind speed and direction can be monitored by small anemometers. Data from these devices can be recorded electronically in computer memory chips, which can be

made very compact and weather proof for field use. The data can be downloaded from the field memory into a portable computer during occasional visits to the field site. While the technology is available to produce very useful microclimatic data for plant pathology, there is still much development work needed to make the equipment sufficiently reliable, compact and cheap for field use.

In recent times, a further aspect of the aerial environment has been observed to affect plant disease. In regions where aerial pollution associated with industrial development has been especially great, the concentration of pollutants such as oxides of sulphur and nitrogen can affect disease development and, in the form of acid rain, can directly damage plants.

The edaphic environment

Soil-borne diseases are affected mainly by environmental conditions in the soil (the edaphic environment). As with the aerial environment, wetness is often a critical factor. Some pathogens such as the pythiaceous fungi causing damping-off and eucalypt dieback require a period of soil saturation to allow germination of survival propagules and infection of roots. The zoospores of pythiaceous fungi require free water for their release and mobility. They also require a soil temperature above a critical level for optimum activity. Highland forests in Victoria, where the soil temperature rarely reaches the critical level of 15°C for *P. cinnamomi*, are never affected by eucalypt dieback even though the eucalypt species present are very susceptible to the disease.

Nematodes require adequate soil water content for their motility (see Chapter 8). On the other hand, nematodes and many pathogenic fungi are adversely affected by prolonged periods of soil saturation, which reduce the concentration of oxygen in the soil. Thus while *P. cinnamomi* requires a period of saturation for zoospore release and infection, prolonged saturation can greatly reduce the activity of the fungus.

Soil fertility can affect development of both soil- and air-borne disease. Some facultative pathogens cause much more disease when plants are growing poorly under conditions of nutrient deficiency than when they are growing vigorously. The defences of the plant that exclude minor pathogens from healthy plants are weakened by nutrient deficiency. On the other hand, biotrophic pathogens such as rusts and powdery mildews are often more common on vigorous well-fertilised plants than on poorly growing plants.

The organic matter content of soil has an important influence on some soil-borne pathogens. For example, eucalypt dieback is common in sandy or gravelly soils low in organic matter. Successful control of *P. cinnamomi* in avocado groves has been achieved by greatly increasing the organic matter content of the soils. In southern Australia, bare patch disease of cereals caused by *Rhizoctonia solani* is more common in poorer, sandy soils than in more fertile soils. In some instances, soils high in organic matter contain larger populations of antagonistic microorganisms which reduce the survival time of plant pathogens and the subsequent incidence of disease.

As with the aerial environment, pollutants (e.g. herbicide residues) in soil can affect disease, or even directly damage plants.

18.5 Interactions among factors

Although the disease triangle can be described as three sets of two-way interactions (pathogen–environment, host–environment and host–pathogen), this is an over-simplification. All three groups of factors interact in any field situation, often in non-linear ways that are difficult to quantify and predict. Attempts have

been made to model the complexity of these interactions using computers. Examples of such programs are EPIDEM, EPIMAY and EPIVEN for *Alternaria solani*, *Bipolaris maydis* and *Venturia inaequalis*, respectively. Much research is needed to provide data on all the interactions involved. Often, from a practical point of view, it is sufficient to concentrate on the few key interactions that govern disease development (e.g. temperature and duration of leaf wetness for foliar pathogens).

The disease triangle for wheat stem rust in Australia

The Australian wheat growing environment is very different from that in parts of the Northern Hemisphere where wheat and stem rust co-evolved. In the Northern Hemisphere the rust must survive freezing conditions in winter, while in Australia the wheat crop is grown during the mild winter and the rust faces the problem of surviving the long dry summer-early autumn period.

Critical factors determining the extent of rust epidemics in Australia are shown in Figure 18.4. The degree of susceptibility of the host depends on the resistance genes in the major wheat cultivars and the degree of horizontal resistance of the cultivars (see Chapter 26). The ability of resistance genes in the wheat population to recognise the prevalent rust population is also critical. Environmental factors that determine disease development include the amount of rainfall in summer which determines the amount of rust that is able to survive and sporulate on volunteer wheat plants whose growth is stimulated by summer rainfall. In the following spring, the amount of leaf wetness, which determines the proportion of urediniospores that are able to infect the main wheat crop, and the temperature, which determines the rate of development of the rust infections, have major influences on disease development. The main control measure involves monitoring the virulence structure of the rust population and breeding wheat cultivars resistant to the prevailing rust pathotypes. In some localities early sowing and the use of early maturing cultivars enable the crop to avoid the most destructive part of the rust epidemic in late spring.

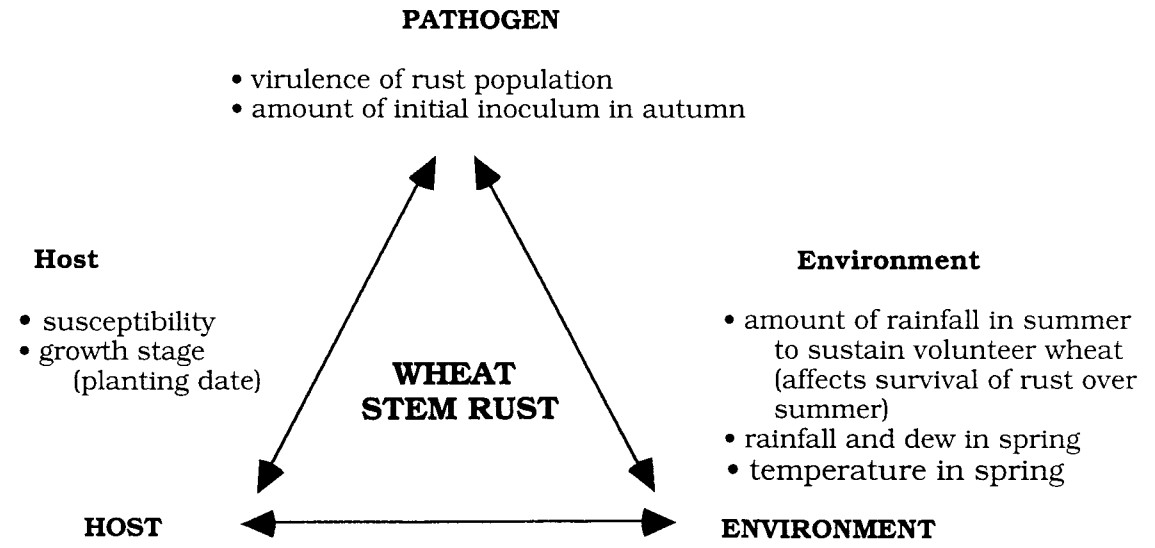

Figure 18.4 Disease triangle for wheat stem rust (*Puccinia graminis tritici*) in Australia, showing the main factors of the pathogen, host and environment that affect the amount of disease that develops in a season.

The disease triangle for eucalypt dieback caused by Phytophthora cinnamomi *in southern Australia*

The most critical factors determining the occurrence of eucalypt dieback are the introduction of the pathogen to the particular forest, the occurrence of susceptible eucalypt and understorey species in that forest and the occurrence of periodic soil saturation during the warmer months of the year (Fig. 18.5). Only one subgenus of eucalypts, Monocalyptus, which includes the stringybarks, ash species and jarrah, is highly susceptible to *P. cinnamomi*. However, the presence of highly susceptible understorey species such as *Xanthorrhoea australis* (in Victoria) and *Banksia grandis* (in Western Australia) presents the pathogen with an ideal food base on which to build up its population. The occurrence of *B. grandis* is determined by the fire history of the forest. Regrowth of this species, at the expense of more resistant *Acacia* species, is encouraged by cool, fuel-reduction fires deliberately lit in the cooler months as a fire control strategy. While manipulation of the fire regime to allow hotter fires that would encourage the proliferation of acacias has been considered as a control measure, it is largely impractical. The main control measures for the disease are prevention of the spread of the pathogen into uninfested forests and re-establishment of vigorous young regrowth forests on affected sites.

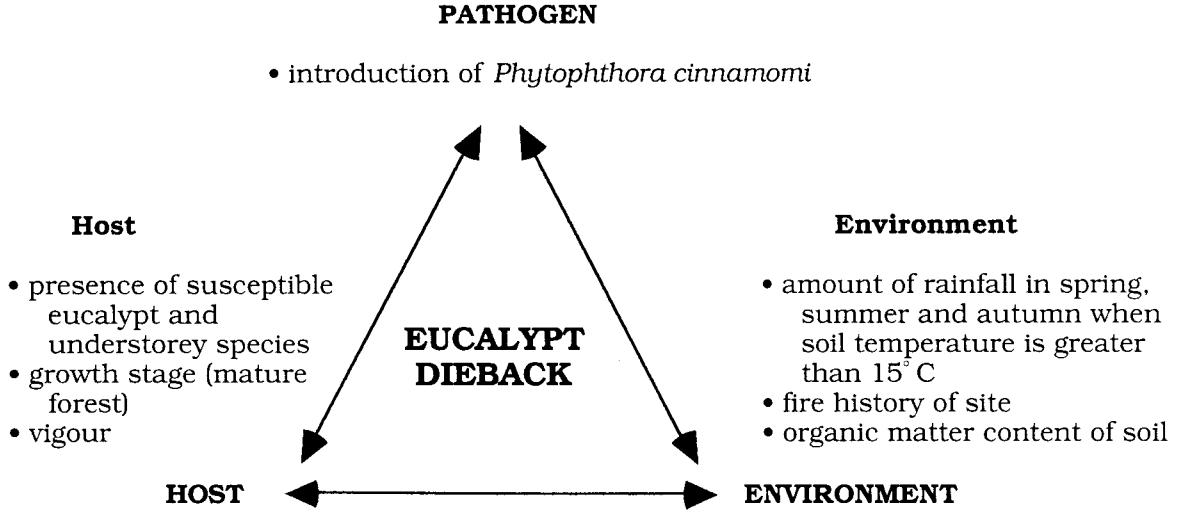

Figure 18.5 Disease triangle for eucalypt dieback caused by *Phytophthora cinnamomi* in forests of southern Australia, showing the main factors of the pathogen, host and environment that affect disease development.

18.6 Further reading

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