cially, are regulated by extremes of temperature and rainfall, and wind acts as an important dispersal agent between and within gardens. Compared with these groups, weather is a less important controller of vertebrate pest populations and behaviour. Ultimately, however, it is the host plants in the garden which determine the range of pests found there. Without these, many pest species would do relatively little damage and many would not thrive at all.

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Weather and plant diseases

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It is estimated that 30% of world food production is lost to pests and diseases (James 1998). This represents a major challenge for agricultural biologists if our food supplies are to be secured. Plant diseases are affected by weather conditions and each disease has its own specific weather requirements. It is unusual to see heavily diseased plants in nature and it is only when plants of similar genotypes are grown together for the production of food on farms or in nurseries that disease becomes a major economic problem (Ingram and Robertson 1999). As "all flesh is grass" (Isaiah 40:6), an understanding of plant diseases and their control is a vital component of our survival strategy. While plant diseases have been recognised since biblical times (Haggai 2:17), the first plant disease to be identified with an organism was that of potato late blight, caused by the fungus *Phytophthora infestans.* Its association with the disease provoked much comment at the time of the first reports in the UK in 1845. A debate raged in the columns of the *Gardeners' Chronicle* between John Lindley, Professor of Botany at University College London, and the Revd Miles Berkeley, a Northamptonshire cleric and keen naturalist (Anon. 1845). Lindley and others were of the view that the rotting of the potato was caused by excessive uptake of water during the wet seasons they were experiencing and that fungus was secondary. Berkeley was of the view that the fungus was the cause. Berkeley is considered to be the father of British plant pathology – the study of plant diseases and their cause.

Causes of disease

Plant disease can be defined as a departure from the normal physiological process; this can be caused by biotic or abiotic factors (Manners 1993). The abiotic factors include direct weather effects (light, heat, cold, drought, and excessive precipitation), pH, nutrition and chemical and mechanical injury. The biotic include fungi, bacteria, viruses and mycoplasmas. One can see why Lindley and others could be justified in their judgement on potato late blight when abiotic factors produce disease symptoms. One of the roles of the plant pathologist is to identify the causes of disease in order that they can advise on appropriate control strategies.

This article is concerned with the biotic causes of disease and in doing so it is best to consider how a pathogen reaches a host in the first place. It may be stating the obvious but in order for disease to occur there needs to be a coming together of a susceptible host and virulent pathogen.

Pathogen movement

In general, fungi produce spores which may be airborne and can be carried over vast distances in air currents; others have a more limited range and are moved short distances by rain splash (McCartney and Fitt 1998; Huber *et al.* 1998). Soil-borne pathogens are generally localised but are moved during cultivation and on equipment. Bacteria are limited in their range except when aided by rain-generated aerosols and carried by animals (Graham and Harrison 1975). Viruses are spread by vectors, such as aphids and nematodes, or by mechanical transmission on pruning knives and by animals brushing against infected plants. All pathogens can be moved on plant material in the course of trade (Rennie 1998).

Requirements for infection

Fungal pathogens split into three groups: the biotrophs, hemibiotrophs and necrotrophs (Ingram and Robertson 1999). The biotrophs include those fungi that can only survive on living tissue, such as the rusts and mildews, and as such live for a period without killing the host. The necrotrophs, in contrast, kill cells and produce rots and necrotic spots. Hemibiotrophs are intermediate, living in the tissues of the host before producing necrotic lesions. To infect, pathogens have to gain entry to their host. There are a number of ways in which this is achieved, but for airborne pathogens this process primarily starts when a spore lands on a leaf. Under optimum conditions of temperature and humidity the spore will germinate and then the infection process can begin. For biotrophs this means entry via stomata (rust fungi) or direct penetration of the epidermis (powdery mildew fungi), where a specialised branch of the mycelium penetrates the host cells to extract nutrients. Necrotrophs enter the host directly or via wounds and secrete enzymes which break down the cells, releasing nutrients required for their growth. The hemibiotrophs generally infect directly but then grow for a short period biotrophically before killing cells and producing lesions. However, plants are not susceptible to all pathogens that are available to infect. The reasons for this interesting phenomenon are the cause of much research, particularly in the field of genetics. Studies have indicated that there is signalling between host and pathogen and this is under genetic control (Crute *et al.* 1997).

Weather factors therefore play a major part in the initiation of disease, and each pathogen has its own optimum requirements for infection. Pathogens are generally spread by wind and rain but those that are transmitted by vectors will be dependent on conditions suitable for them. Some spores are released under drying conditions or in films of water arising from rain or dew on the leaf surface. High humidities, or the presence of free water on a leaf surface, are required for initiating the infection process. Temperature will speed or slow the infection process and disease development. Knowledge of the requirements for each pathogen will aid in their control, particularly where this can be achieved by the application of fungicides. It is generally uneconomic and environmentally unsound to apply fungicides as routine just to ensure that the plant is protected throughout its life. Knowledge of the specific conditions which will favour infection should enable control measures to be targeted more specifically. An understanding of weather and the pathogen allows pathologists to produce forecasts of potential disease epidemics.

Disease forecasting

In the 1940s, Beaumont, a plant pathologist located in the south-west of England, was examining ways in which the number of fungicide sprays that were being applied to potatoes for the control of potato late blight could be better timed. In order to ensure against the ravages of blight, farmers would begin spraying well in advance of a prospective epidemic. Beaumont investigated the forecasting scheme of a Dutch scientist Van Everdingen (1926) but found that it was too sensitive, triggering sprays well before they were required. The 'Dutch Rules' relied upon dew formation which was difficult to measure. Beaumont considered humidity to be more important a measurement than dew, as during periods of high humidity there is no wind and the nightly drop in temperature is most likely to result in dew formation and leaf wetness. He made adjustments to the Rules, and the revision became known as the Beaumont Period (Beaumont 1947). Its use persisted into the mid-1970s when the Smith Period became the UK standard. Smith, an agricultural meteorologist, tested the validity of using a shorter period of higher humidity than Beaumont and found that using a 90% humidity criterion for 11 hours in each of two days instead of 75% for 48 hours gave similar results (Smith 1956).

The Beaumont and Smith Periods relied on weather data from the Met Office synoptic stations. That these are not necessarily centred in areas of potato production and are located mainly on airfields, which do not mirror accurately the conditions in potato fields, indicates that there are opportunities to improve disease forecasts. The advent of microprocessors and the automatic logging and storage of data means that portable weather stations are available for siting in or near to crops and, hopefully, to increase the accuracy of predictions. A number of more sophisticated schemes have been developed since Beaumont for use in forecasting in a wide range of crop situations (Hardwick 1998). However, reliability still presents problems as, despite detailed research on

Fig. 1 Black spot of roses showing the spotting and subsequent chlorosis of the leaves

the understanding of pathogen behaviour, forecasting schemes tend to impose specific thresholds as triggers for action, be it temperature, or humidity level and duration. For example, a temperature of 10 °C will trigger a Smith Period, whilst 9.9 °C will not; plant pathogens are not so sensitive in their requirements. It is stating the obvious but the plant is the best indicator of risk, as the presence of a diseased plant indicates that weather conditions have been favourable and inoculum is present. Schemes that are based on careful crop observation and use of disease thresholds are therefore likely to be more successful. Unfortunately for some diseases, *e.g.* potato late blight, by the time they are discovered in a crop it is generally too late for control measures to be effective.

The traditional scourges for the gardener are black spot (Fig. 1) and powdery mildew of roses, scabs and cankers of fruit; and an understanding of the conditions favourable for infection by these pathogens can aid in control. For example, warm (above 18 °C), wet conditions favour black spot of roses. In contrast, powdery mildew of roses is active under warm (above 25 °C), dry conditions but requires periods of high humidity, in the absence of leaf wetness, at around 15 °C for infection. Applications of fungicide can be timed with these criteria in mind. Frequent applications may be required under prolonged periods of favourable weather. There may be a reluctance to use fungicide treatments and reliance then has to be placed upon the planting of disease-resistant cultivars. There is still much to learn, but where there are no alternatives to fungicide use environmental sustainable disease control will not be achieved without the development of robust forecasting schemes (Walters and Hardwick 2000).

Climate change

As disease is so influenced by the weather it is anticipated that climate change will have a major influence on diseases affecting plants in the UK. Little research into the impact of climate change has been undertaken on pathogens of crop plants (Rosenzweig and Hillel 1998), let alone those in gardens. Much of the literature on the effect of changes to climate in relation to pathogens covers the effects of raised CO_2, ultraviolet (UV) light and ozone levels (Chakraborty *et al.* 1998). Ambient ozone levels were thought not to affect pathogens but elevated ozone levels appear to reduce the impact of biotrophic and increase the effect of necrotrophic fungi by affecting the host. The effect on the host appears to be on the functioning of the chloroplast and nucleus. The effects are apparent in plant resistance mechanisms (Sandermann 1998). Elevated levels of CO_2 seem to compensate for the harmful effects of ozone but effects are complicated by the interaction of the nutrient status and age of the plants (Tiedemann and Firsching 1998).

The effects of UV light have been studied extensively and the longer-range UVA (315–400 nm) and short UVB (290–315 nm) bands have been shown to stimulate fungal spore production. UV effects also impact on survival of fungal spores during dispersal and the early stages of infection (Paul *et al.* 1998). Interest in the effects of UVB has been stimulated because such radiation is likely to increase at ground level if stratospheric levels of ozone are depleted. There are indications from field experiments, for example, that increased UVB radiation could decrease the severity of *Septoria tritici* on wheat, although at the levels predicted it was thought unlikely to have an agronomic impact (Paul *et al.* 1998).

It is the intricate relationship between the weather and diseases that makes predictions difficult. An understanding of the complexities of the interrelationships between the environment, the pathogen, and its host are required. The disease tetrahedron is the classic way of understanding the interaction between the main elements which combine to produce disease (Zadoks and Schein 1979). Disease will not occur where there is no pathogen, no receptive host or no environment favourable for infection by pathogens. People influence each to a varying degree in the breeding of disease-resistant cultivars, moving pathogens in the course of trade and affecting the environment by release of greenhouse gases (Fig. 2). These factors provide a starting point to understanding the potential impact of climate change on diseases of crop plants. New hosts will attract from the indigenous pathogen bank

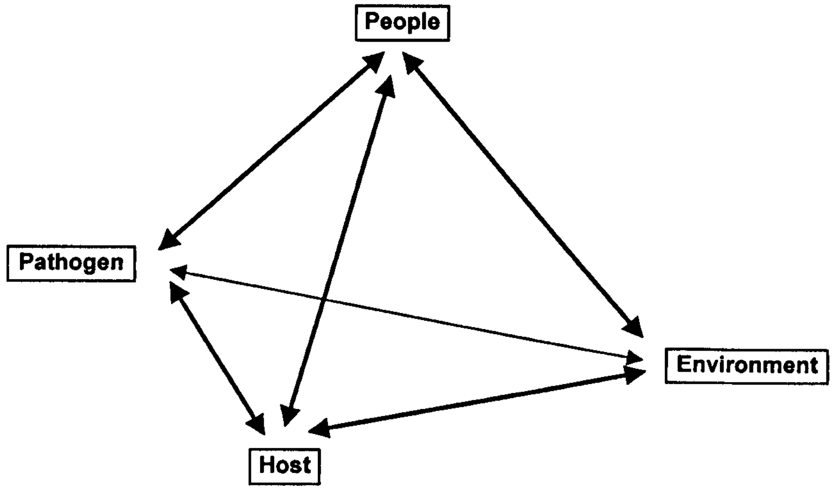

Fig. 2 The disease tetrahedron

both 'alien' (in the sense that they have not previously been exposed to that species) and familiar pathogen species, as some plant pathogens are capable of surviving in the soil for several decades (Coley-Smith *et al.* 1990). Wild plant species serve as alternative hosts for many diseases of crop plants (Ingram 1999) and the reservoir of potential pathogens amongst the natural flora has only been touched upon by plant pathologists. There are two main aspects of climate change which will affect plant diseases: first, it will alter the balance of existing pathogens and, second, it will favour the survival of alien introductions, assuming a susceptible host is present.

Some examples of those pathogens that gained entry into the UK and were able to establish are tobacco blue mould (*Peronospora tabacina*), which was first found in England on *Nicotiana* spp., grown under glass in 1958. The outbreak was suspected to have arisen from an escapee which was originally imported under quarantine for research. The disease was then not recorded again in the UK until 1974, the fungus probably surviving on amateur tobacco growers' premises or in gardens (Dale and Hardwick 1976). The disease is now widely established on ornamental nicotianas. Chrysanthemum white rust (*Puccinia horiana*) entered the UK on infected plants imported from Japan in 1963. A successful campaign eradicated the disease but there were subsequent outbreaks in 1976 originating from infected cuttings imported from the Continent. These are cases where weather conditions and the presence of a suitable host already occur in the UK and resources are aimed at preventing such introductions.

Of great interest are situations where the disease spectrum alters with a change in climate. As an example, there are three main rust diseases that affect cereals, each having its own temperature requirements. The dominant rust in the UK is yellow rust (*Puccinia striiformis*). Brown rust (*P. recondita*) is a problem late in the season as temperatures rise, and also in hotter years. Stem rust (*P. graminis*) is an occasional late visitor to crops on the south coast of England. The origin of the outbreaks is thought to be from wind-blown spores from north Africa or southern Europe, mainly the Iberian peninsula (Jones and Clifford 1983). The three cereal rusts of wheat have differing temperature optima and ranges for infection (Fig. 3). It can be seen from the graph that a significant rise in mean summer air temperature could change the dominant rust species affecting winter wheat in the UK.

The predicted changes in climate for the UK are still within the current ranges of temperature and rainfall requirements for most of our current pathogens. Although the spatial and temporal fluctuations in disease incidence and severity with the weather will continue, any long-term effects of a shift in climate are likely to bring changes in specific disease dominance.

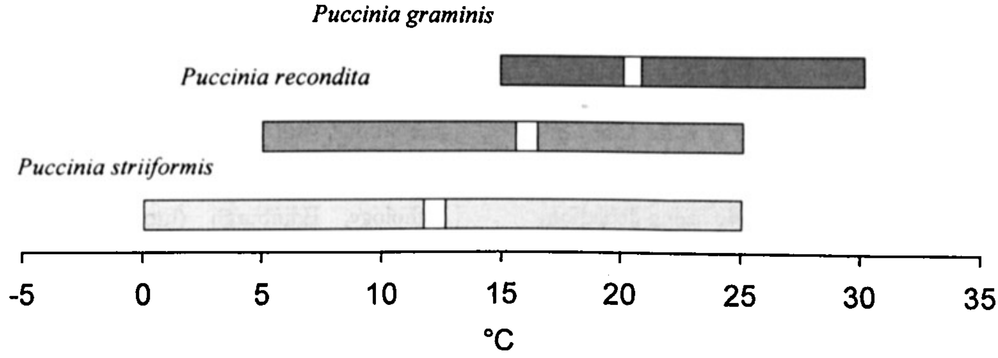

Fig. 3 Temperature ranges for spore germination of three cereal rusts (optimum is the clear zone)

Conclusions

Weather plays a vital role in plant disease development, affecting both the dispersal and infection processes. The severity of disease fluctuates annually depending on seasonal changes in weather. Plant pathologists try to determine which combination of climatic conditions affect specific pathogens in order to understand the disease risk so that farmers and growers can be advised on the appropriate precautions to take to control diseases. This may involve the optimum timing of fungicide applications or manipulation of the cultivation of the crop to avoid, or reduce, disease risk.

Climate change is likely to bring about changes in the geographical range of current crops and plants, and with it the spectrum of diseases affecting them. There are likely to be introductions of new plant species, and plants hitherto confined to glasshouses may be grown in the open. This will bring new diseases and challenges for their control.

It is rather disconcerting that over 150 years have passed since potato late blight was found in the UK and, despite intensive research and a plethora of chemicals that have been developed for its control, the disease is still causing major economic losses. This is primarily because we still have no control over the weather!

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The science of weather: Cumulus clouds

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Clouds like those in Fig. 1 are very commonly observed. They are called 'cumulus' because they have a heaped-up form (from the Latin 'cumulus', meaning 'heap'). When their vertical extent is small, they are called 'cumulus humilis' or 'fair-weather cumulus'. When their vertical extent is such that cloud width appears to be about the same as cloud height, they are called 'cumulus mediocris'. When they are taller than that, they are called 'cumulus congestus'. The tops of cumulus clouds often resemble cauliflowers. The towering clouds that produce showers of rain, snow and/or hail are called 'cumulonimbus' (from the Latin 'nimbus', meaning 'rainy cloud'). The tops of these clouds are often anvil-shaped, as shown in Fig. 2.

Cumulus clouds are manifestations of convection. They form when bubbles of buoyant air rise. These bubbles, called 'thermals', form over 'thermal sources' such as concrete, bare sand and rocky hillsides, which become warmer on sunny days than surrounding areas of grass or forest. As the bubbles rise, the air inside them cools because of adiabatic expansion. The rate of cooling is 9.8 degC per kilometre of ascent. If bubbles rise far enough, the air inside them cools sufficiently for saturation to occur, at a height called the 'condensation level'. Above this level (*i.e.* inside the cloud), the latent heat that is released when condensation occurs reduces the rate of cooling to about 6 degC per kilometre.

The height at which condensation occurs depends upon the temperature and dew point of the surface air. If this air is moist, the condensation level is low. In the British Isles in winter, it is typically at a height of about 600 m. On a summer afternoon, it is typically at a height of 1200 m or more. Over deserts, surface air is often so dry that saturation does not occur at all, even when convection is vigorous enough to lift air to substantial heights.