

**T5.7: A Review of Potato Blight -
A Disease of Global Significance**

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Abstract:

Potato late blight is a disease of potatoes wherever they are grown. The disease is caused by *Phytophthora infestans*. This pathogen attacks the leaves, stems and tubers of the potato. Under favourable conditions, it can lead to total crop destruction. Its presence was first recorded in the UK in 1845. It is a classic illustration of an alien organism attacking a crop that was uniformly susceptible and on which a major section of the population depended for its food. Resistance breeding has proved to be difficult, compounded by the reluctance of processors to use unfamiliar cultivars. Detection methods have advanced with the development of techniques such as enzyme-linked immunosorbant assay (ELISA) and polymerase chain reaction (PCR).

The repeated application of fungicides is the main method of control. However, there is pressure on growers from environmentalists and consumers to reduce fungicide applications. Much effort has been put into the development of forecasting schemes, both to inform the start of fungicide programmes and to regulate the frequency of applications according to risk. The UK population of *P. infestans* has changed due to the recent introduction of new genotypes and an A2 mating type from Mexico. The new strains are more aggressive than the strain they displaced, and the A2 mating type allows increased diversity, a means of survival in soil and the potential to initiate disease epidemics earlier in the season. After 160 years, the disease still poses a major threat to potato production worldwide.

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Introduction

Potato late blight (PLB) is caused by *Phytophthora infestans*. It is called late blight to distinguish it from early blight, which is caused by an unrelated fungus, *Alternaria solani*. Early blight, despite its name, tends to appear in more mature crops and is favoured by warmer temperatures than PLB. Early blight is not commonly present in the UK.

P. infestans was originally classified as a fungus but is now considered to be more closely allied to members of the chrysophytes, which include the diatoms and brown algae (Judelson 1997). The distinguishing features that separate it from the fungi are the composition of the cell walls, which are of cellulose and not chitin, and the motile zoospores with two flagella. However, this reclassification takes nothing away from its destructive power.

PLB is a classic aggressive disease on three fronts. It was introduced into a country from overseas. It attacked a universally susceptible crop. The crop happened to form the staple diet of the poor, and its destruction led to a major famine.

Host range

P. infestans is primarily a disease of potatoes and tomatoes. It affects a limited number of wild hosts. In addition to *Solanum* spp., it infects, among others, *Datura stramonium* (devil's trumpet), *Nicotiana glauca* (tree tobacco) and *Pharbitis purpurea* (common morning glory). *P. infestans* has been reported in the wild on *S. nigrum* (black nightshade) in Wales (Deahl et al. 2004). The pathogen was found on this common weed host after PLB was widespread in potato fields. The weed was not considered to be influential in the epidemic, but is regarded as an alternative host.

Symptoms

PLB first appears as brown spots on the leaves, at the growing point, or sometimes, on very young plants, on the stem. The brown leaf spots usually have a pale green margin, the underside of which often bears the delicate white spore-producing growth of the pathogen. In muggy weather, the leaf spots enlarge rapidly, killing the leaflets and leaf stalks but for a time leaving the main stems intact. Stem lesions may form in the absence of leaf symptoms later in the season when the canopy is closed, producing a microclimate favourable to the pathogen. This generally happens in periods of dry weather, which are not normally considered suitable for infection. In this case, the crop appears free from the disease and if an appropriate interval is not left between haulm (stems) destruction and lifting, the tubers are at risk from infection. Lesions on the stem tend to be water-soaked and black, or dark grey, in colour. Sporulation is generally sparse. PLB also affects the tubers, where it produces purple grey water-soaked areas of skin over a firm rusty brown granular rot extending into the flesh, producing a dry rot. Under damp conditions, infected tubers may rot completely due to secondary bacterial infection.

Occurrence in the UK

The first accounts of the disease appeared in the *Gardeners' Chronicle* of 16 August 1845 in a letter from Dr Bell Slater with a report from the Isle of Wight. In the issue of 13 September, an emotive report of its occurrence in Ireland began: 'We stop the Press, with very great regret, to announce that the Potato Murrain has unequivocally declared itself in Ireland. The crops around Dublin are suddenly perishing ... for where will Ireland be, in the event of a universal potato rot?' A graphic account of the disease and its effects on the Irish has been written by Large (1940). The socioeconomic impact of the disease is illustrated by the fact that in 1845 the population of the island of Ireland was about 8 million, compared with 5.6 million now. The respective figures for Great Britain were about 18 million in 1845 and 60 million in 2005. This major decline in population was due to a combination of famine and emigration and had a devastating effect on Ireland's economy, only relatively recently revived.

Historical significance

Historically, the importance of PLB is that it was the first time that a 'fungus' was associated with a disease. This was the century of theories of

spontaneous generation and of biotic causes of disease. It was also the century of Charles Darwin, Thomas Henry Huxley, Louis Pasteur and Anton de Bary, who made great advances in the classification of the plant and animal kingdoms and the scientific understanding of natural history. However, it was a Northamptonshire cleric, the Reverend Miles Berkley, a keen student of natural history, who challenged the orthodoxy and propounded the theory that the cause of PLB was a fungus (Berkeley, reprinted 1948). This could be regarded as the birth of British plant pathology. It also illustrates the importance of the amateur in scientific thinking and observation. However, it was not until 1876 that *P. infestans* was fully described as the cause of PLB by de Bary (Birch and Whisson 2001).

Potatoes worldwide

Potatoes were introduced into Europe in the late 16th century from South America, mainly as a curiosity. It was probably in Ireland that the potato became established as a major arable crop and formed the staple diet (Burton 1989). From Europe, the potato rapidly spread around the world and, at 293 million tonnes, is only exceeded in tonnage produced by rice, maize and cereals (Anonymous 1998). PLB is found wherever potatoes are grown and, even after 160 years of investigation, still has the potential to destroy a crop completely and is a pathogen that is a major threat to global food security.

Countries producing over 10 million tonnes of potatoes annually are Belarus, China, Germany, India, Poland, Russia, Ukraine and the US. The UK produces about 6 million tonnes. The highest yields, of over 40 tonnes per hectare (t/ha), are produced by Belgium, Netherlands, Switzerland and the UK. The lowest yields, of less than 5 t/ha, are in Burundi, Cameroon, Kenya, Montserrat, Papua New Guinea, Swaziland and Rwanda (Anonymous 1998). This shows a large disparity between the efficient European countries and those that are resource-poor. Yields from some of the larger producers are also relatively low, for example, in China, which is the largest producer, at nearly 50 million tonnes, yields are only 13.7 t/ha. Average yields in Poland are 17.5 t/ha, Russia 11.4 t/ha and Ukraine 11.1 t/ha. These low yields are the result of low fertiliser inputs, a poor understanding of crop protection methods and a lack of money to pay for healthy seed. In these situations, diseases, such as PLB, play a significant role in reducing yields.

Economic significance

The losses caused by PLB are difficult to measure, except from experiments specifically designed for the purpose, as there are many other diseases, pests and abiotic factors that influence final yield. Also, it is often difficult to attribute losses in store to PLB, as soft-rotting bacteria mask the primary cause. The economic impact of PLB is not only due to the direct effect on yield but also the cost of control measures. In the UK, over 20 times as much fungicide, measured in kilograms per hectare, is applied to potatoes as to winter wheat (Garthwaite et al. 2002). It has been estimated that about \$1 billion annum is spent in the US, Europe and developing countries on fungicides to control PLB (Anonymous 2005a). Birch and Whisson (2001) quote an annual figure of \$5 billion for worldwide losses and the cost of control measures.

Lifecycle

To understand the disease, it is necessary to examine its lifecycle (Figure 1). *P. infestans* is self-sterile and exists with two mating types, A1 and A2. When these mating types interact, they produce resting spores (oospores), which are capable of long-term survival in soil. The A2 mating type was originally confined to central Mexico, believed to be the centre of evolution of the pathogen, and was only recorded in the UK in 1985 (Malcolmson 1985). Until then, the lifecycle was relatively simple. Airborne spores landed on a leaf and germinated, either directly, to produce a germ tube, or in surface moisture, via motile zoospores. Under optimum conditions, lesions became visible after about three days. Sporulation occurs about two days after lesion development. Tubers become infected when spores are washed into the soil during rain. In this phase, the pathogen could be considered an obligate parasite as it requires a living host for long-term survival. The pathogen survives from season to season through infected tubers or in solanaceous weeds. Infected shoots emerge in the spring to start the cycle off again. The arrival of the A2 type facilitated the possibility of mating, gene transfer and the production of oospores, which can overwinter in the soil. This not only provides a second means of overwintering but contaminates the soil for future plantings.

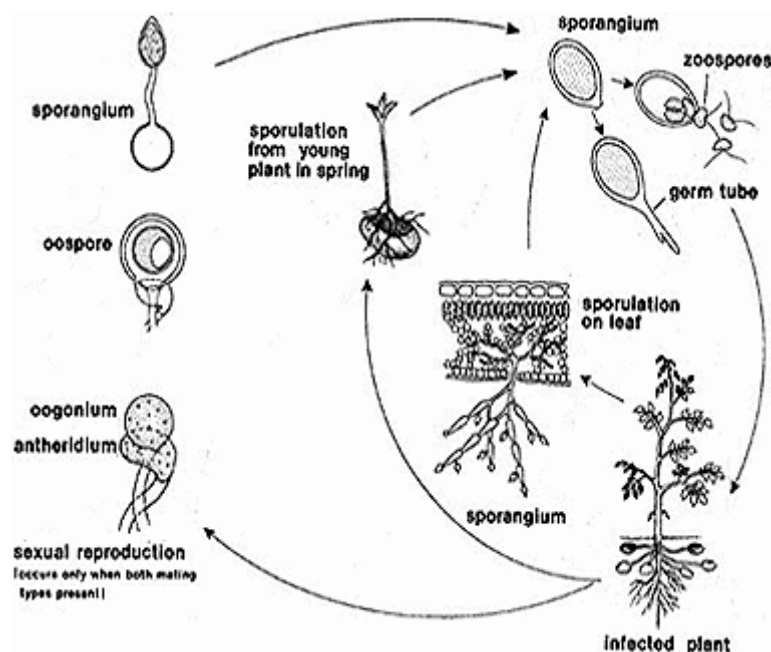


Figure 1: The lifecycle of *Phytophthora infestans* (from www.whyfiles.org/128potato_blight (accessed May 2005)).

A survey by Shattock et al. (1990) from 1985 to 1988 indicated that A2 mating types were widely distributed and present in about 8% of isolates examined, but there was no evidence during the four-year survey of any increase in

incidence. Pittis and Shattock (1994) reported that oospores of *P. infestans* produced *in planta* and added to soils were infective for up to 35 weeks. More recent survey data indicate that the A2 mating type is rare in Britain, comprising only 3% of 2,691 isolates tested (Day et al. 2004).

Drivers of the disease

Populations of *P. infestans* found outside North America belonged to a single clonal lineage, designated US-1. This led to a theory that the epidemics in Europe were caused by US-1, and that this strain subsequently spread to other parts of the world in trade (Fry et al. 1993). However, recent analysis of mitochondrial DNA of *P. infestans* from herbarium material suggested that a genotype different from US-1 was involved (Ristaino et al. 2001). The occurrence of resistance to the phenylamide fungicides in the early 1990s (Deahl et al. 1993) indicated that there were significant changes taking place in the population of *P. infestans*. The molecular tools then available established linkages with Mexico as a source of the new genotypes (Shattock 2002). These were designated US-6, US-7 and US-8 and soon replaced the indigenous strain, US-1. The new strains were all resistant to the phenylamides. US-8 was found to be particularly aggressive and was sometimes referred to as 'super blight'. Because of the increased aggressiveness of the new strains, there were concerns that traditional disease forecasting schemes that were established with the 'old' populations, when US-1 dominated, and based on the work of Crozier (1934), were no longer valid.

Disease tetrahedron

When considering the drivers of disease, it is appropriate to consider the 'disease tetrahedron' (Figure 2), which illustrates the interaction between host, inoculum, the environment and human activity.

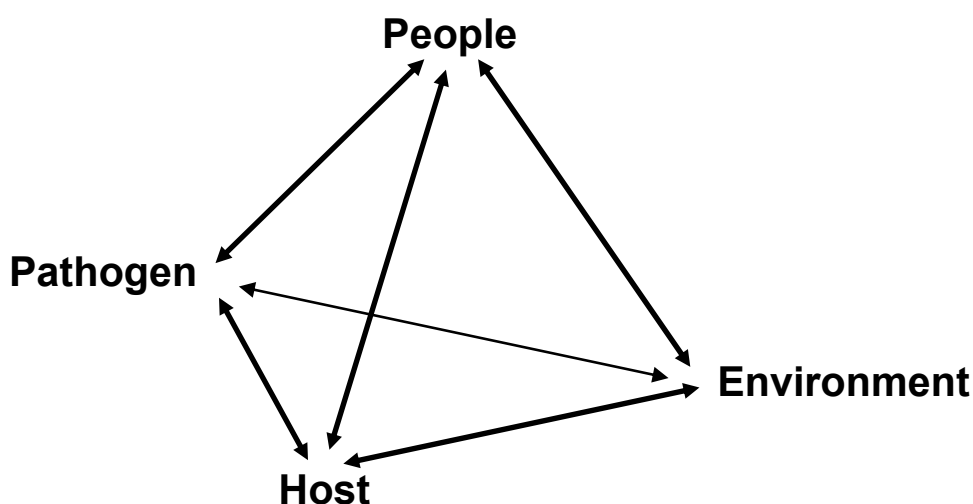


Figure 2: The 'disease tetrahedron' (after Zadoks and Schein 1979).

The relationships between the key factors that influence disease are well described by the tetrahedron. Disease will not occur: if the environment is unfavourable; if the host is absent or resistant; or if the pathogen is absent or is avirulent on the host. Overarching is the influence of human activity, which can influence all three areas. Human activity can affect the environment, not only in terms of climate but also how the crop is grown, for example, by altering the time of planting, spacing, fertiliser inputs and irrigation. The host can be affected by resistance breeding, and the pathogen by methods to exclude inoculum or by the use of fungicides to reduce its impact.

The importance of the disease tetrahedron was illustrated by the foot-and-mouth disease epidemic which affected the United Kingdom in 2001. A highly virulent virus was infecting highly susceptible hosts (sheep and cattle) under favourable environmental conditions. It was only when the human factors were addressed by the strict policing of movements of individuals and vehicles that the disease was brought under control (Anonymous 2002).

Environment

It is generally assumed that the environment is the driving force for diseases (Hardwick 2002). Smith and Hugh-Jones (1969) analysed the foot-and-mouth disease outbreak of 1967 and concluded that weather played a greater part in the spread of the disease than had previously been recognised.

The effect of climate change, however brought about, could affect the environmental part of the tetrahedron. Epidemics of PLB are dependent on warm, moist weather occurring between May and August. Predicted changes in climate in the UK to warm, drier summers in the south would suggest that PLB is likely to be less of a problem. However, the rise in temperature, concomitant with a 3% predicted increase in relative humidity in the north, could increase the risk of late blight in this region.

Pathogen

It is generally considered that inoculum is always available to infect the crop. The major source of inoculum is from infected haulm that develops on piles of discarded tubers (dumps) created during the harvesting and storage operations. These should be covered with black polythene sheeting or treated with a herbicide before the season begins. If they are not, the tubers sprout and the haulm develops, generally earlier than the surrounding crops due to the warmer and more humid microclimate in the dumps. If blighted tubers are present, the pathogen sporulates on the haulm, providing a source of early inoculum, which, under favourable conditions, will spread to neighbouring crops. Within the growing crop, the main source of inoculum is from the planting of infected tubers and occasionally from groundkeepers. Once a crop becomes infected, spores are released and are capable of travelling large distances in air currents and rain-generated aerosols to infect neighbouring and distant crops, where they can initiate local and national epidemics. It is possible that blight appeared in the Isle of Wight from spores blown across from northern France, as did the outbreak of foot-and-mouth disease in 1981 (Bourke and Lamb 1993).

Host

The host affects the speed of development of the disease through its resistance and the nature of that resistance, whether it is absent, partial or total. The habit of the host, for example, the structure of the crop canopy, influences the way disease moves about the plant, while the architecture of the plant influences the microclimate and also the amount of damage caused by wind. This is important where damage or wounding can aid pathogen entry.

More effort has been put into breeding for resistance against *P. infestans* than any other potato disease. However, resistance breeding has been only partially successful. Sources of resistance genes, R genes, have come primarily from *Solanum demissum*. However, early failures were due to over-reliance on R genes, which were readily overcome by new races of the pathogen. Since the 1970s most breeding programmes have been directed towards horizontal resistance (Landeo et al. 1995). Eastern Europe is an area of interest in resistance breeding. Recently, cultivars have been imported from Hungary which show promising resistance to PLB. Tests are now under way to identify cultivars that will be commercially acceptable (Anonymous 2005b).

Zimnoch-Guzowska and Tatarowska (2004) report on seven areas that have caused problems. They are (i) the identification and utilisation of new sources of resistance and insufficient agronomic value of the resistance sources used; (ii) combination of earliness with PLB resistance; (iii) complexity of genetic determination of PLB resistance; (iv) combination of foliage and tuber resistance; (v) screening method applied for resistance evaluation; (vi) cost of selection for resistance and (vii) lack of molecular markers applicable to select for PLB resistance.

Sources of the disease

Potatoes originate from the Andean region of South America. It has generally been assumed that the disease did too (Bourke and Lamb 1993). There was much trade between Peru, the US and Europe, with fleets of cargo boats that were engaged in the guano boom in the mid-19th century. Bourke and Lamb (1993) considered that the most probable entry point of potato late blight into Europe was a cargo of potatoes officially imported into Belgium to boost productivity of a national crop, which was degenerating from virus and other diseases. This has been the pattern of spread of the disease round the world – by the importation of apparently sound but subclinically diseased tubers.

It is now accepted that the centre of genetic diversity for *P. infestans* is in the Toluca Valley in central Mexico (Shattock 2002). In 1976, there was a major drought in Europe, which resulted in a severe shortage of potatoes. A quarantine-breaking shipment of potatoes was imported from Mexico, which introduced new genotypes of the A1 mating type and also, more devastatingly, the hitherto absent A2 mating type.

Pathways of spread

Currently in the UK, the primary pathway of local spread is from infected tubers that have survived the winter in the field or in piles of discarded tubers. Recently, the British Potato Council, in a 'Fight against blight' campaign, has highlighted the risks from such dumps. Data from 2003 and 2004 seasons showed a reduction in outbreaks that were considered to be dump-related from 17% to 2% (Anonymous 2005c). Long-range spread is via seed or ware tubers that have subclinical levels of infection and that have been transported transnationally. Moreover, as has been described above, this route has been the source of the introduction into Europe of new genotypes of the pathogen that have replaced the native population (Fry et al. 1993). Long-range spread of spores is also possible and distances of several hundred miles have been suggested (Van der Zaag 1956).

Risk

Now, 160 years after its introduction into Europe, this disease still poses a major risk. It is currently suppressed by the use of chemicals in the form of repeated fungicide application. Sometimes as many as 18 applications are made in a single season (Hardwick and Turner 1996).

The risk from the disease is twofold. The first is premature defoliation, which restricts yield. The second is from infected tubers that initiate secondary bacterial soft rots when harvested tubers are stored. The former tends to result in lower yields, especially when PLB strikes early in the season not long after crop emergence, while the latter can result in total crop loss from the loading of infected tubers into poorly managed stores.

From about the end of May, the crop is exposed to inoculum. The growth of foliage is rapid in weather conditions that also favour the spread of the pathogen. Once the crop becomes infected, it is very difficult to contain the disease, even with fungicide use. Under favourable conditions, only about two to three weeks may elapse before the disease reaches a level where it is prudent to destroy the haulm (Large 1959). Intervals of hot, dry weather can halt progress of the pathogen and, therefore, delay the epidemic. It is recommended that harvest shouldn't begin until at least two weeks after the death of the haulm in order to prevent any blighted haulm coming into contact with the tubers and infecting them. The disease does not generally spread in store. However, the presence of the disease means the tubers are susceptible to moisture loss, which, if not dealt with by ventilation and covering, can lead to secondary bacterial rotting and major storage losses.

The displacement of the indigenous A1 mating type in Europe by more aggressive genotypes, and the arrival of the A2 mating type, have fundamentally changed the levels of risk. We now have a more aggressive pathogen that is capable of sexual reproduction and may therefore be persistent in the soil between crops.

The UK potato industry is generally very conservative in its choice of cultivars. Much of the crop is grown under contract requiring specific cultivars to be grown, for example, for processing for crisps, frozen chips (French fries) and fresh for the fish and chip trade. Maris Piper, used primarily for general ware, pre-pack and processing (French fries) occupies 21% of the potato area of Great Britain. Eight cultivars occupy 46% of the area grown. Of these cultivars, only one, Pentland Dell, has a resistance rating above 4 for foliage bight (on a scale of 1–9, where 1 is susceptible and 9 is highly resistant), and it occupies only 4% of the national area of potato crops. Cultivars with a resistance rating of below 5 are considered to be at significant risk from PLB. One instance of the industry's conservatism was Macdonald's requirement for the cultivar Russet Burbank. This cultivar was bred in the US in 1914 and is highly susceptible to PLB. Planting PLB-susceptible cultivars not only increases the risk of crops becoming infected but also provides a major potential source of inoculum and puts other potato crops at risk.

Detection

Field identification can be difficult if the characteristic white halo of sporophores is absent. Symptoms can be confused with wind damage, fertiliser scorch, herbicide damage and lesions caused by *Botrytis cinerea*.

Traditionally, the detection of *P. infestans* has been by incubation in moist chambers, scraping off the resulting spores and identifying them under a light microscope. The spores are very characteristic, in being translucent and lemon-shaped – elliptical with a papilla at each end – and c. 30 x 20 µm in size.

Identification of the pathogen in tubers is more difficult. Lesions are visible on clean tubers with white skins, but are more difficult to detect on russeted dark-skinned cultivars. On the surface of the tuber the presence of the disease is apparent as dark purple water-soaked lesions. Scraping away the skin to a depth of 2–3mm reveals a rusty-brown granular dry rot. This can sometimes be confused with internal rust spot, a physiological blemish disorder that does not spread but affects the marketability of the crop.

The advent of molecular techniques now permits identification directly from lesions in the field. Some molecular tests may not be species-specific. But with potato foliage, species identification is not required, as lesions are not caused by other *Phytophthora* species or closely related pathogens. This is not the case with tubers, where there are two other rots, one called pink rot, caused by *P. erythroseptica*, and the second, watery wound rot, caused by *Pythium ultimum*, which could make non-specific test results invalid. Amplification of DNA using PCR can improve specificity (Judelson and Tooley 2000; Wangsomboondee and Ristaino 2002). Molecular methods using Taqman PCR and lateral flow devices can both speed up the process of identification and improve the sensitivity of detection. Mitochondrial markers and nuclear markers have been used to examine diversity in populations (Shattock 2002).

Fungicide resistance is detected by floating leaf disks on dilutions of systemic fungicide or spraying the fungicide onto the leaf disks, inoculating the disk with a zoospore suspension and monitoring lesion development. Detection of resistance in pathogen populations is also now possible using molecular methods (Ishii et al. 2001).

Critical infection points and control

Understanding the aetiology and epidemiology of PLB is critical to its control. PLB can obviously be controlled by not growing potatoes at all, and in some high-risk areas this may be the only practical solution. In which case, other appropriate food crops have to be considered. In areas where the pathogen, or some key strains, are not currently present, it may be possible to exclude them by normal phytosanitary and quarantine methods. However, for the UK, this is no longer an option because the pathogen and its varying pathotypes and strains have already been found.

Control of inoculum is largely dependent on the frequent application of fungicides, generally on a 7–10-day cycle, from when the haulm meets along the rows and certainly before canopy closure. The use of resistant cultivars is limited because there are few, if any, that are commercially acceptable for growing on a wide scale.

Limited manipulation of the environment and agronomy can be important measures for organic producers where the use of fungicides is not an option. They tend to have small plantings in areas where few potatoes are grown commercially. Early plantings and early cultivars tend to be less affected by blight as the crop can reach maturity before blight reaches the crop. The potato plant tends to vary in susceptibility during the growing season as the carbon ratio varies (Granger and Rutherford 1963). Reduced nitrogen inputs, so that the haulm is not so vigorous and lush, and increased plant spacing to alter the microclimate to restrict periods when conditions are favourable for infection, will reduce the risk but are not necessarily practical in the UK, where areas for production are limited. Methods appropriate for organic producers may also work in less developed countries (Agu 2004, Nyankanga et al. 2004).

There is increasing pressure on growers to reduce the number of fungicide applications to the crop, not only from environmentalists but also from consumers who wish to purchase pesticide-free produce. Forecasting schemes have been developed in an attempt to predict the onset of blight epidemics, but with limited success (Beaumont 1947; Hansen et al. 1995; Hims et al. 1995; Krause et al. 1975; Schrödter and Ulrich 1967; Smith 1956; Sparks 1984). The commonly used forecasting scheme in the UK, the Smith Period (Smith 1956), uses criteria of two consecutive days, ending at 09.00, when the minimum air temperature does not fall below 10°C and the relative humidity is at least 90% for 11 hours or more on each day.

The multiplicity of schemes for forecasting blight, even within single countries, and the continued development of further schemes, indicate that forecasting for this disease is complex. It is highly unlikely that a standard scheme will be applicable to all countries, or even all regions within a country. The increasing sophistication of the meteorological data-capture equipment may permit more precise schemes. However, Royle and Shaw (1988) suggested that, with an annual substantial build-up of inoculum with a disease such as potato blight, a 'relatively complex tactical spray programme based on forecasting is unlikely to be worthwhile because it will so rarely differ from routine spraying.' This is true in part, but there are still savings to be made from defining the start of the programme in situations where inoculum is limiting. All blight forecasting schemes assume the presence of inoculum. This is unlikely, when a range of forecasting schemes can trigger applications based on weather well ahead of the first occurrences of symptoms in the crop (Taylor et al. 2003). Using forecasts can also reduce the number of sprays applied, because even modern fungicides can reduce yields in the absence of PLB (Taylor et al. 2000).

Forecasting schemes, when successful, should give the farmer the information necessary to provide a cost-effective means of protecting crops from the ravages of PLB. However, there are a number of factors that impinge on the success of forecasting schemes.

The forecasting models all seek to interpret the biology of the pathogen in the context of conditions that affect its development, survival and ability to infect and colonise the relevant host. There is an infinite variety of possible combinations. Fortunately, the advent of powerful computers has solved many of the technical constraints. However, biological processes are in a constant state of flux, particularly with the introduction of new strains and mating types and the consequent potential for more rapid changes in aggressiveness and temperature adaptability, and it is unlikely that all eventualities will be covered by even the most complex of models. That is an important constraint. If the models are too complex, they may be impractical, particularly if they are dependent on information being supplied by farmers or their consultants.

Most of the models impose a cut-off to some of the measurements, which is necessary to trigger a response. This is not necessarily arbitrary and may be derived experimentally. But it is likely to be a mean from a range of results. In biology, there is no reason why 10°C and not 9.9°C should be critical, or 90% relative humidity and not 89.5%, 11 hours and not 10 hours 59 minutes as in the Smith Period (Smith 1956). And, though it is now possible to collect data by the minute rather than every 6 hours from the synoptic network, this gives a false sense of precision. Bourke (1953) suggested that, 'Even the finely evolved model can scarcely avoid the defects of over-simplification and over-rigidity – over-simplification because the complexity of the phenomena involved cannot be precisely reflected in any easily handled formula; over-rigidity because, even if the criteria are to be in a form capable of objective application by a number of workers, they must introduce abrupt, and to a certain extent, arbitrary discontinuities which appear in nature only as gradual transitions. This does not mean that the evolution of a good working model would not be of considerable value ...' The inclusion of husbandry factors, by

weighting each factor empirically, gives some confidence that schemes might work, as it involves the crop and the field history. Computer models such as NEGFY (Hansen et al. 2000), which can be modified to take account of local factors, are an improvement but still include a level of subjectivity.

The siting of weather stations is important with a disease like PLB, where precision is important. Where in the field should the equipment be sited to give the most reliable reading if at one end there is an exposed hill and the other a wooded area by a lake or river, providing a completely different microclimate? Some schemes can be impractically precise. Large (1956) suggested that it is the intelligent interpretation of a network of stations that is important. A network has the advantage that failure, or inaccurate readings, at one or two sites will not fail to trigger a general warning (Smith 1956). This implies that forecasts should be general and not specific.

Some on-farm systems are linked on a network basis (e.g. PLANT-Plus, Raatjes et al. 2004), supplementing the regional weather forecasts. It is the scale of the network required that is worthy of investigation, particularly in areas like the UK with its varied topography and weather systems when compared to countries like the Netherlands, with a more uniform weather system. Bourke (1953) was sceptical and commented, 'However, it is unfortunately true that even an expensive network of special weather observations from every potato field in the country would not fully cover the local variations in susceptibility to blight.' However, forecasting is ultimately the only method by which pesticide use can be confidently reduced.

Farmers are coming under increasing pressure from their customers, particularly the large supermarket chains, to justify pesticide use. Forecasts provide that justification. However, the main constraint on using such systems is farmers' concerns about the reliability of forecasting systems, for the reasons given above. There are also practical difficulties to overcome. Scheduling is one, particularly when repeat spraying is required. The ability to spray crops in all fields at risk is a major logistical constraint and it is often easier, once started, to maintain a 7- or 10-day schedule rather than one varied according to risk. The increasing use of contractors for spray operation also means that they have to be reserved in advance and this imposes limitations on the ability to 'fine-tune' the spray programme. Where spray intervals cannot be altered for practical reasons, it may be possible to reduce dose according to forecast risk, as suggested by Nielsen (2004). Spraying on a large scale also poses logistical problems in terms of sprayer capacity, adequate stock of fungicides and competing demands for the treatment of other crops (Hinds 2000).

Improvements in the detection of low levels of inoculum to provide warning of impending attack are possible using flow cytometry (Griffith et al. 2002). This, when combined with improved air samplers, such as those used by the military to detect pathogens in biological warfare, can be used to distinguish *P. infestans* spores from the other air spora. Early detection of PLB in the field by field spectral reflectance based on identifying plant stress (Wojtowicz and Piekarczyk 2001) is possible. While this may be appropriate for surveys, by

the time the stress has been detected, the PLB is likely to be well established and it is probably too late to be able bring the disease under effective control.

Current understanding

Potatoes are a major world crop. PLB is a significant pathogen the importance of which has not diminished in the 160 years since its discovery. If anything, the risk from the disease has increased because of the introduction of new strains and the A2 mating type into Europe. This provides a route to increased diversity in the population through sexual activity and an addition survival mechanism. The impact is yet to be realised in the UK but it is having an effect on continental Europe (Flier et al. 2004).

Lessons to be learned are to maintain phytosanitary inspection and warning systems capable of early detection of species that may be indigenous and of new strains that may replace indigenous ones to detrimental effect. Quarantine is still a major tool for screening material imported into the UK. It is also recognised that it is not only seed potatoes that are a source but ware tubers too. Both the A2 and new pathotypes originated from imports of ware during potato shortages.

Molecular methods now provide the opportunity for the more rapid detection of pathogens and, as their sensitivity increases, of detecting them in bulk samples.

The story of PLB is also a warning to maintain diversity, not only in terms of cultivar resistance but also of crop. The famine in Ireland was avoidable, for cereals and meat and dairy products were still being exported. It was the poverty of the cottiers that meant they had no money to buy food, which resulted in starvation and mass emigration. This was not helped by the indifference of the British Government at the time, or by absentee landlords immune to their tenants' plight.

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Summary views: Forward look to 2015 and 2030

Potato late blight

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Advances in biology, in particular, tend to follow from inventions and developments in other sciences. For example, it was not possible to know about the 'micro fungi' until advances in physics and improvements in optics led to the development of the compound light microscope. The advent of the electron microscope allowed the viewing of virus particles for the first time and also the fine structure of host–pathogen interactions. Advances in immunological and molecular detection methods, including ELISA, PCR, restriction fragment length polymorphism (RFLP) and microsatellites have all been used to improve significantly the specificity and speed of identification. Lateral flow devices, used in pregnancy testing, have been adapted to recognise plant pathogens. They are used widely by the UK Plant Health and Seeds Inspectorate for rapid diagnosis of diseases such as sudden oak death, which is caused by another *Phytophthora* species, *P. ramorum*. Advances in electronics and computing have made possible some of the miniaturisations that allow PCR equipment to be made portable, and allow meteorological data to be captured locally by relatively inexpensive portable weather stations. So, when considering developments likely to affect our understanding of PLB in 10–25 years, it is important to be aware of developments taking place in other areas of science and technology.

1 Drivers

The drivers of PLB originate on two fronts, the environment and the requirement for food.

Changes to the environment through the human activity that is leading to climate change may affect areas where potatoes can be successfully grown. The widely predicted trend to more extreme weather events will affect the ability to plant and harvest the crop. High rainfall at planting and harvest will have a major impact because waterlogged soils will not allow the use of the heavy equipment required for these operations. Warmer weather in the early summer is likely to advance the time of first outbreaks of PLB, leading to early crop defoliation and reduced yields. Significant shifts in the earliest reported dates of the disease would be an indicator of changes taking place, either in climate or by adaptations in the pathogen.

Statistics from the Food and Agriculture Organisation (FAO) indicate that potato production is expanding rapidly in developing countries. In the past decade, production has increased at an annual rate of 4.5%, exceeding those for maize, wheat and rice, which have slowed. Potatoes are becoming an increasing source of food, employment and income in Asia, Africa and Latin America. As the area expands, so does the potential for an increase in

disease, as the sources and supply of inoculum increase. The levels of disease reported from countries and regions could indicate that crops are expanding at a rate that is not sustainable due to the absence of a concomitant increase in control measures.

2 Sources

There is a history of new genotypes of PLB arriving in the UK from trade during times of shortages. As demands for convenience foods increase, there is a requirement for continuity of supply to meet the needs of the processing industry. Potatoes for processing currently account for about half of UK production. To reduce the impact of soil-borne pests and diseases, it is advisable not to crop with potatoes more frequently than one in four years. This has resulted in a decrease in land available for potato production. Companies are sourcing supplies from continental Europe or in some cases growing them there. This trend is likely to increase. It means an increased risk of bringing in PLB on ware tubers. The development of molecular methods using portable PCR machines would be a valuable aid to the detection of this threat.

3 Pathways

Hitherto, the pathways of entry for PLB have been restricted to importation via subclinical infected seed and ware tubers, and possible from airborne spores from the near continent under favourable wind conditions. Waste from the processing industry should be taken care of under normal arrangements for dealing with waste. Household peelings which are inadequately composted would pose a risk. However, the presence of oospores has opened up a second channel of entry in that soil from land which has previously cropped potatoes and where A1 and A2 mating strains have been found could be contaminated with oospores. Data on the PLB situation in exporting countries needs to be under constant review.

4 Linkages

Historically, PLB has demonstrated its potential impact where the crop dominates the consumption of a sector of the population. Lessons to be learned from that experience are that diversification is vital, not only in terms of crop type but of deploying cultivars with a range of disease resistances.

Fungicides currently play a vital role in PLB control. Repeat applications of the same active ingredient have implications for the development of resistance. Resistance developed to the phenylamide group of fungicides shortly after their introduction. They are still used to a limited extent in the UK and it is their continued presence that may have kept the population of A2s at low levels, as all A2s detected in the UK were sensitive to them. Pressure to reduce dependence on the use of fungicides currently endangers our ability to control the disease.

PLB is a limiting factor in the production of potatoes in less-developed countries with poor infrastructure and restricted financial resources to pay for

crop protection measures, including the necessary advice on their use. This is a major constraint on an expansion in potato production.

5 Early warnings of high risk

Key indicators of high risk from PLB would be:

- increased development of fungicide resistance, putting control measures at risk,
- increases in the national areas planted to disease-susceptible cultivars, due to commercial pressure to produce cultivars required by the processing industry
- changes in weather patterns to those favouring an early start of the epidemic
- changes in aggressiveness in the pathogen population
- an increase in detecting A2 mating types and finding oospores in soil.

All these aspects can be monitored effectively given the appropriate resources.

6 Uncertainty in identification and detection

There is little uncertainty in identification of the pathogen at species level. It can be readily identified from sporulating lesions. In the past, the identification of strains has relied on the use of differential cultivars and is now achieved by molecular methods.

Early cultivars of potatoes are generally lifted and marketed straight from the farm. Second earlies and main crop are generally harvested and put into store for periods of several months to ensure a continuity of supply until new supplies are available in the following season. Detection of PLB on tubers going into store is difficult, because they are generally covered in soil particles which obscure the symptoms. Methods that will aid the detection of subclinical levels of PLB in tubers going into store would be of immense value, as PLB is one of the major triggers of soft rotting of the tubers, which can lead to major losses. This is of particular importance in regions where storage conditions and management techniques are not optimal. Portable PCR equipment that can be taken on site for qualitative analysis of bulk samples, and the use of electronic 'sniffers', which can detect the release of specific volatile compounds generated in the interaction between the pathogen and the tissues it is destroying, would be valuable. The early detection of PLB can lead to decisions being taken on unloading the store and marketing the produce before there is a major deterioration in quality.

7 Challenges confronting efficient and effective detection and identification

The most important challenge confronting the efficient and effective detection and identification of PLB in the future is to reverse the decline in the number

of scientists in the UK with a depth of knowledge and understanding of the disease. There has been little financial support from the industry until relatively recently, when the environmental issue of pesticide use has become a significant issue, together with recent seasons where PLB has been more widespread. However, there is a perception that the disease can easily be controlled by fungicides and there is little need to study it in depth. The attitude has been that if fungicides dominate control, research can be funded by the pesticide industry. Defra currently does not fund any work on PLB. It is left to the industry, though the British Potato Council, which is currently funding three projects. Some work is funded in Northern Ireland and Scotland. There are only about three scientists in the UK working full-time on the disease and a similar number part-time. Institutes where PLB work is ongoing are ADAS; the Central Science Laboratory; the Department of Agriculture and Rural Development, Northern Ireland; SAC; and the Scottish Crops Research Institute.

The need to maintain expertise in PLB research seems not to figure in succession planning and it is probably true that there are more scientists in retirement with an understanding of PLB than are currently working. The absence of a significant advisory and extension service means that scientists are not receiving intelligence on problems occurring in the industry and are unable to monitor changes that can affect the identification and detection of the disease.

8 Addressing the challenges

The identification and detection of a pathogen should only be considered as a means to an end. The end is effective and economic control of PLB to secure a continued, adequate supply of a major internationally important staple food. So the challenge to implementers and policy makers is to develop a strategy to tackle the disease in a holistic manner.

Support to scientists is required to allow them to develop a detailed understanding of the disease, not interrupted by discontinuities in funding. If funding issues are resolved, scientists can more readily address the challenges of developing the programmes of research. These involve setting in place monitoring systems to provide evidence-based data to detect changes in aggressiveness, fungicide resistance and climate-change impacts.

The role of oospores in the initiation of PLB epidemic in the UK is unknown. Methodologies for detection in soil are required, as are data on their survival, inoculum potential and cultivar responses to infection. Methods of detecting new and potent strains from overseas in bulk shipment are required and also detection, particularly, of oospores and how they are dealt with during the processing of imported ware and seed tubers. Sound storage is predicated on the loading of disease-free tubers and so the ability to detect infected tubers entering store is an important area. Most current forecasting schemes are based on an assumption that inoculum is not limiting. This is demonstrably untrue. Methods to quantify spores of *P. infestans* in the air spora before they can infect crops could assist in improving the accurate timing of fungicides for PLB control.

It is over 160 years since PLB was first recorded in the UK. While it was the first pathogen to be associated with a disease, its identity has changed and is continuing to change, it is still defying detection and continues to defy attempts to bring it under control. It has been an enigma, it is currently an enigma and is likely to continue to be an enigma well into the future and most probably beyond 2030.

All the reports and papers produced within the Foresight project 'Infectious Diseases: preparing for the future,' may be downloaded from the Foresight website (www.foresight.gov.uk). Requests for hard copies may also be made through this website.

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