A review of early blight of potato

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Early blight of potatoes, causal agent *Alternaria solani,* causes major yield losses in most potato growing areas of the world. Leaf symptoms are characteristic dark brown to black lesions with concentric rings. In this review morphological, physiological and molecular characters of the pathogen as well as the disease cycle, epidemiology and control of the disease are discussed. The early blight situation on potatoes in South Africa is also summarised.

Key words: *Alternaria solani,* disease forecasters, early blight, epidemiology, potato.

Early blight caused by *Alternaria solani* Sorauer is a major foliar diseases of potato (*Solanum tuberosum* L.). Disease symptoms are characteristic dark brown to black lesions with concentric rings, which produce a 'target spot' effect. Symptoms are initially observed on older, senescing leaves.

The causal organism of early blight was first described by Ellis & Martin (1882) as *Macrosporium solani.* The first reference to the fungus as a parasite and its association with potato leaf blight was by Galloway (1891) in Australia. In the USA, Chester (1892) noted the disease on potatoes and other cultivated plants of the Solanaceae, particularly tomato (*Lycopersicon esculentum* Mill.) and eggplant (*Solanum melongena* L.). He described the symptoms and observed that the progress of early blight was slower than that of late blight, caused by *Phytophthora infestans* (Mont.) de Bary. He also noticed that potato plants severely affected by early blight had lower yields and produced smaller tubers.

Jones (1893) was the first to suggest the name 'early' blight to distinguish the disease from 'late' blight. The name stems from the fact that early blight attacks early maturing cultivars more severely than medium or late maturing ones, whereas late blight is more severe on medium or late-maturing cultivars. A severe early blight epidemic caused shortening of the growing season due to premature death of vines, which led to smaller, immature tubers being produced. He observed that early blight was more prevalent on leaves attacked by insects and ascribed this to insect damage providing an entrance point for the fungus. He recommended the use of two to three Bordeaux sprays and later planting to control the disease.

Galloway (1893) reported that early blight was widely distributed in the USA, but less abundant in the southern and far western states. He described the development of the disease and morphology of the fungus, and also suggested the use of Bordeaux sprays as control measure.

Around this time, there was a controversy regarding the pathogen status of *M. solani.* Jones (1895) found *M. solani* to be pathogenic, entering the host directly or through insect wounds or stomata. However, an *Alternaria* species was also isolated from the lesions. Its spores resembled those of *M. solani* but were unable to produce characteristic early blight lesions in artificial inoculation studies. This species was thought to be responsible for 'tip-burn' of potato leaves. The difference between *Macrosporium* and *Alternaria* at that stage was arbitrary. (*Macrosporium* was thought to bear spores singly and *Alternaria* in chains.) Jones & Grout (1897) isolated two fungal species from potato leaves. One species was found to be the parasite causing early blight and they proposed that it be named *A. solani.* The other, a saprophyte found on dead or decaying leaves of many plant species, was identified as *Alternaria fasciculata* (Cook & Ellis) L.R. Jones & Grout (= *Alternaria alternata* (Fr: Fr.) Keissl.).(Ed.)

THE PATHOGEN

Taxonomy, morphology and cultural characteristics

A. solani is classified in the domain *Eukaryota,* kingdom *Fungi,* phylum *Deuteromycota,* class *Hyphomycetes,* order *Hyphales,* series *Porosporae.*

Colony morphology of *A. solani* varies widely, but is generally effuse, greyish brown to black, with a cotton-, felt- or velvet-like texture (Ellis & Gibson 1975). Growth is rapid on many growth media, but

special conditions are required for sporulation. Orange to dark red pigments are produced which colour the medium.

Cells of *A. solani* are multinucleate, but different organs vary in the number of nuclei. Nuclear division in hyphal cells is followed by multiple septation, which results in the division of elongated tip cells into several multinucleate cells (King & Alexander 1969). Conidiophores are dark or olivaceous brown, thick-walled, straight to flexuous, septate, arise singly or in small groups, up to 110 µm in length and 6–10 µm in diameter (Neergaard 1945; Ellis & Gibson 1975). Conidiogenesis is tretic (Neergaard 1945; Ellis & Gibson 1975).

Conidia are usually pale to olivaceous-brown, produced singly or seldom in short chains, straight or slightly flexuous, obclavate to elongate, double walled with 0–8 longitudinal or oblique and 6–19 transverse septa, 75–350 µm in length and 20–30 µm in diameter in the broadest part (Ellis & Martin 1882; Rao 1964, 1969).

Beaks are about one-half to one-third the length of the conidium, filiform, straight of flexuous, septate, hyaline to pale brown and 5–9 µm in diameter (Ellis & Martin 1882; Rao 1964, 1969). Because of the variability in spore dimensions, they overlap with dimensions of other large-spored *Alternaria* species. In routine work, identification is assisted by leaf symptoms, host range and cultural characteristics. Biochemical or molecular techniques best verify the identity of the fungus.

Sporulation in pure culture

A. solani does not sporulate readily in culture if left undisturbed. Factors such as mycelial wounding, temperature and light affect the formation of spores. Various techniques to induce sporulation of the fungus have been described (Rands 1917b; McCallan & Chan, 1944 in Barksdale, 1969; Aragaki 1961; Lukens 1963, 1965; Lukens & Horsfall 1969; Rotem & Bashi 1969; Bashi & Rotem 1976). Rands (1917b) produced spores by growing *A. solani* on potato-dextrose agar (PDA) for 10 to 12 days, shredding the cultures and then allowing them to dry in the sun. According to Barksdale (1969), McCallan & Chan (1944) used a similar technique. *A. solani* was grown on PDA, the cultures scraped and then placed in moisture chambers in the sun or under UV light, with the Petri dish lids slightly ajar. Rotem & Bashi (1969) indicated that spore production is induced by inhibition of vegetative growth of the fungus. Growing the fungus on PDA, cutting the culture into 1 mm^2 blocks and placing them on a $CaCO_3$ medium in the dark at room temperature has also been found to induce spore formation (Shahin & Shepard 1979). A technique developed by Barksdale (1969) involved the growing of *A. solani* on reconstituted lima bean agar at 23 °C. During the day, cultures were placed under indirect sunlight or for 8 hours under cool-white fluorescent light. The mycelium of one-week-old cultures was scraped, the lids removed and the dishes placed upside down on a rack, 10 mm above the surface, under the same light and temperature conditions.

Although conidiophores are easily damaged by UV light (Rich & Tomlinson 1968), their formation is initiated by light. Lukens (1965) observed that blue light inhibits sporulation, but red light can reverse this inhibition. Sporulation in vitro requires a short exposure to near-UV light. This induces conidiophores to produce conidia, which then need dark conditions to complete formation (Lukens & Horsfall 1969).

Conidia do not form at temperatures above 20–23 °C (Aragaki 1961; Lukens 1963; Waggoner & Horsfall 1969, in Pscheidt 1985). An interrupted wetting period may often be more conducive to producing spores than a continuous wet period.

In vivo, sporulation of the pathogen is affected by the state of the host and tends to accelerate with an increase in necrotic tissue formation and a decrease in photosynthesis (Cohen & Rotem 1970; Bashi & Rotem 1975a). Sporulation is inhibited by sugars, which promote vegetative growth and even the production of conidiophores (Waggoner & Horsfall 1969, in Rotem 1994; Bashi & Rotem 1975a). Sporulation in the field requires at least two days. Conidiophores are produced during wet nights. Light and dryness the next day induce the production of conidia, which are then formed during the second wet night (Bashi & Rotem 1976).

Variation in culture

Variation in culture is common in *A. solani* (Barksdale 1969). When isolating single spores, it is possible to obtain a variant isolate. For this reason, Barksdale (1969) suggested that for disease screening or epidemiological studies, mass transfer of 'normal' sections of a culture would provide a more representative wild type.

The other, more time-consuming option is to use a large number of single spore isolates, each with different characteristics, as inoculum.

Molecular characterisation

Various authors have provided evidence for the existence of physiological races of *A. solani,* based on morphological, physiological and pathological differences (Bonde 1929; Henning & Alexander 1959). More recently, variation has been examined using biochemical and molecular techniques. Such genetic analyses of plant-pathogen populations are important in understanding epidemiology, host–pathogen co-evolution and resistance management (McDonald et al. 1989; Leung et al. 1993; Milgroom & Fry 1997; Aradhya et al. 2001).

Petrunak & Christ (1992) screened isolates of *A. solani* and *A. alternata* for isozyme activity to determine if a correlation existed between species, host or geographic origin of isolates and isozyme phenotype. A relatively high level of variation was found, when compared to results of other studies. One explanation for the variation may be natural mutation and variation in culture. Their results, however, showed no clear difference between isolates when compared according to host or locality.

Weir et al. (1998) subsequently used random amplified polymorphic DNA-PCR (RAPD-PCR) analysis to investigate genetic variation among isolates of *A. solani* and *A. alternata.* Again, extensive variation and differences between the two species were evident, confirming the results of Petrunak & Christ (1992). Host-related differentiation amongst isolates of *A. solani* from potato and tomato was, however, also found (Weir et al. 1998). Although this study did not include pathogenicity tests to determine if isolates were host-specific, the high level of genetic differentiation suggested host-specific forms of *A. solani* on potato and tomato (Weir et al. 1998; Peever et al. 1999).

A study conducted by Peever et al. (1999) on the population genetic structure of *Alternaria* species from citrus also included *A. solani* isolates. The *Alternaria* isolates were scored for variation at 16 putative RAPD loci. *A. solani* isolates were clearly distinct from the *Alternaria* isolates from citrus (genetic distance of 70 %).

Another molecular tool that is used to establish phylogenetic relationships between fungi, including members of the genus *Alternaria,* is the sequencing of ribosomal DNA (rDNA) and the analysis thereof (Pryor & Gilbertson 2000). Analysis of the relatively conserved nuclear 18S sequence has shown a close phylogenetic relationship between *Pleospora* (anamorph *Stemphylium*) and *Alternaria* (Morales et al. 1995; Berbee 1996). The more variable internal transcribed spacer (ITS) region is used in the examination of relationships between fungal taxa at or below the species level and has also been used in investigations involving host-specific toxin-producing *Alternaria* species (Kusaba & Tsuge 1995; Pryor & Gilbertson 2000).

The mitochondrial small subunit (SSU) rDNA sequence has also been used to investigate relationships among closely related fungus species (Kretzer et al. 1996). In a study using ITS and mitochondrial SSU sequences of *Alternaria* and *Ulocladium* species, Pryor & Gilbertson (2000) showed a distinct number of species-clades. The porri clade includes *A. porri* (Ellis) Cif., *A. solani, A. dauci* (J.G. Kühn) J.W. Groves & Skolko, *A. crassa* (Sacc.) Rands and *A. macrospora* Zimm. Most of the clades identified were similar to the species-groups previously established using morphological characters (Simmons 1995). In general, better taxonomic resolution of *Alternaria* was achieved with analysis of nITS than SSU sequences (Pryor & Gilbertson 2000). Analysis of ITS sequences revealed differences between some species that were not detected in analyses of SSU sequences (Pryor & Gilbertson 2000).

Toxin production

Various researchers have studied toxin production in *A. solani* (Brian et al. 1952; Stoessl 1982; Ichihara et al. 1983; Nishimura & Kohmoto 1983; Cotty & Misaghi 1984; Langsdorf et al. 1990). It has been shown that *A. solani* produces a non-host-specific toxin, alternaric acid (Brian et al. 1952). Alternaric acid is produced readily in vitro, but it is not known to what extent it is produced in the plant (Stoessl 1969, 1982). Not all strains of the fungus produce the toxin, and there is no apparent correlation between virulence of strains and toxin production. When alternaric acid is systemically introduced into a potato plant, it produces symptoms similar to those of *A. solani* (Stoessl 1982).

A. solani also produces other nonspecific toxins, such as zinniol, altersolanol and macrosporin

(Stoessl 1982; Cotty & Misaghi 1984). Ichihara et al. (1983) reported the production of phytotoxins – solanapyrones A, B and C – but other researchers could not confirme this (Rotem 1994).

A. solani produces two host-specific toxins in culture, which are able to produce early blight symptoms when applied in combination, but not individually (Matern et al. 1978). There is also a difference in toxin sensitivity between tomato and potato plants and non-host plants.

The disease

Geographic distribution

Early blight is widespread in most areas where potatoes or tomatoes are grown, but is particularly prevalent in tropical and temperate zones. The disease is a potential threat where potatoes are cultivated under irrigation or during times of heavy dew (Rotem 1994).

Early blight tuber rot may occur if tubers wounded during harvest are infected by *A. solani* spores present on or near the soil surface (Lahman et al. 1982) Tuber rot is, however, not common and has a limited distribution (Pscheidt 1985).

Economic importance

The primary damage of early blight is due to premature defoliation of the plant. Photosynthesis rates increase and respiration rates decrease in apparently healthy tissues (Rotem 1994). Physiological changes are difficult to measure and evaluation of crop loss is based on the level of disease (Rotem 1994). Early literature (Neergaard 1945) cites yield losses of 5–50 %. There is often a discrepancy between damage to foliage and yield loss, which is due to the increase in disease spread at the end of the season, when most of the yield has been produced (Rotem 1994). Control of early blight has been shown to increase yield (Harrison & Venette 1970; Potter & Hooker 1972). When tomato fruit or potato tubers become infected, the quantity and quality of marketable produce is decreased and the number of secondary pathogens increases (Pscheidt 1985).

Symptoms

Ellis & Martin (1882) were the first to describe the symptoms on dying potato leaves. Symptoms are initially observed on older, senescing leaves (Jones 1893; Rands 1917a; Pscheidt, 1985; Shuman 1995). Likewise, the most susceptible plants are those that are physiologically old, weak, malnourished and wounded by wind, sand, hail or insects (Rands 1917a; Heuberger & Dimond 1941). Characteristic symptoms are dark brown or black lesions with concentric rings on leaves, which produce a 'target spot' effect (Rands 1917a). This zonation is induced by day-night fluctuations in temperature, moisture and radiation and is often absent in greenhouse-inoculated plants. Lesions are similar on all hosts (Pscheidt 1985).

Enlarging lesions are often surrounded by a narrow chlorotic halo due to toxins produced by the pathogen, which move ahead into uninfected epidermal cells (Rands 1917a; Pscheidt 1985). Lesions are usually oval in shape, but under unfavourable conditions may remain small and angular, conforming to the interveinal spaces (Rands 1917a). Lesions enlarge, coalesce and eventually cause death of the leaf (Pscheidt 1985). Spores may be seen on older lesions when viewed under a microscope. Lesions can also develop on stems and petioles.

Infected tubers develop a dry rot, characterised by isolated, dark, irregular, sunken lesions on the surface (Pscheidt 1985). Diseased tissue under lesions is dark brown, firm and 10–12 mm deep.

Host susceptibility

Numerous studies have shown that young, immature potato tissues and plants have a transient resistance to early blight (susceptibility is age-conditioned). In tomato, *A. solani* causes a collar rot in seedlings and early blight in mature plants, whereas in potatoes, it causes early blight of mature plants, which intensifies at flowering (Moore & Thomas 1943). Susceptibility of potatoes is thus strongly correlated with cultivar maturity and early blight resistance decreases as cultivar maturity levels increase (Rands 1917a; Douglas & Pavek 1972; Pelletier & Fry 1989; Johanson & Thurston 1990; Shtienberg & Fry 1990). This type of resistance is known as temporary resistance and should be distinguished from permanent resistance which is unaffected by plant age, rate of cultivar maturity and yield (Rowell 1953).

Some studies suggest that early blight susceptibility may not be totally due to cultivar maturity and is heritable, subject to directional selection (Leclerg 1946; Douglas & Pavek 1972; Abel-

Rahman 1977; Brandolini 1992; Gopal 1998). Breeding and genetic identification of resistance in potato are difficult. Sources of resistance to *A. solani* among lines of *S. tuberosum* subsp. *tuberosum* are very low or absent (Rotem 1994). However, levels of resistance have been found in wild diploid potatoes (Thompson & Mendoza 1984; Herriot et al. 1986; Mendoza 1989). Breeding these plants with tetraploid species has resulted in a few early-maturing but resistant varieties (Thompson & Mendoza 1984; Mendoza 1989).

Reports exist for varietal susceptibility levels in South Africa (Visser 1999), America (Herriot et al. 1986, 1990; Johanson & Thurston 1990; Christ 1991), India (Gopal 1998), Israel (Caligari & Nachmias 1988) and Peru (Brandolini 1992).

Rotem (1994) noted that the effect of age on susceptibility is modified by prevailing temperature, which suggests that susceptibility is governed by physiological, rather than chronological, age. Other growth or stress factors that also affect susceptibility include vigour of plant growth, soil moisture and nutrition (Rowell 1953; MacKenzie 1981; Kumar et al. 1983). However, the physiological causes are not well described (Rotem 1994). Increased sensitivity of older leaves to toxins and enzymes due to changes in the membrane composition does not explain the susceptibility of tomato seedlings to the disease.

Reduction in ratio of tuber yield to foliage is associated with a reduction in lesion size and early blight incidence (Rotem & Feldman 1965). The susceptibility of tomatoes to early blight is also governed by the ratio of fruit to foliage, rather than the absolute level of yield (Rotem 1994). It has been suggested that susceptibility increases in plants deficient in sugars, but that this is due to increased sporulation, rather than increased infection (Horsfall & Dimond 1957).

Host nutrition

High nitrogen levels, together with low phosphorus and medium to high potassium levels, decreases host susceptibility (Barclay et al. 1973; Soltanpour & Harrison 1974; MacKenzie 1981; Kumar et al. 1983). This could be explained as follows: High N levels may prolong vegetative growth and delay ripening, low levels of P lead to reduced fruiting and low K levels cause reduced tuber formation. Calcium deficiencies may also reduce tuber formation. MacKenzie (1981) showed that the apparent infection rate and final amount of early blight decreased with increasing rates of nitrogen fertilisation, but unfortunately this also caused a drop in specific gravity of tubers and reduced chip quality.

Host range

The most important hosts of *A. solani* are tomato, potato and eggplant (Pscheidt 1985). Other hosts include horse nettle (*Solanum carolinensis* L.), chili (*Capsicum frutescens* L.) (Gupta et al. 1980), black nightshade (*Solanum nigrum* L.) and non-solanaceous hosts such as wild cabbage (*Brassica oleracea* L.), cucumber (*Cucumis sativus* L.) and zinnia (*Zinnia elegans* Jacq.) (Rands 1917a; Neergaard 1945).

Disease cycle

Overwintering and survival

A. solani is a polycyclic pathogen as many cycles of infection are possible during a season (Shuman 1995). Primary infections on new plantings of potatoes or tomatoes are caused by overwintering inoculum (Pscheidt 1985). The pathogen overwinters as mycelium or conidia in plant debris, soil, infected tubers or on other host plants of the same family (Pelletier 1988; Shuman 1995). Chlamydospores have also been reported as a source of overwintering inoculum for early blight, allowing the pathogen to survive cold temperatures in or on the soil (Basu 1971; Patterson 1991). The inoculum remains infective in debris in uncultivated soil for 5–8 months. The dark pigmentation of the hyphae increases their resistance to lysis (Lockwood 1960). Spores survive most frequently in infected debris and seed. Primarily meteorological, edaphic and biotic factors determine survival in debris and seed. The fungus survives best in dry, fallow fields (Rotem 1994).

Dispersal

The primary inoculum produces conidia in the spring, which are then splash- or wind-dispersed to the lower leaves of the plant where they germinate and infect (Rotem 1994). Wind, rain and insects are the principal methods of dissemination of *A. solani.* For example, the Colorado potato beetle (*Leptinotarsa decemlineata* Say) spreads the fungus when it feeds on leaves of infected plants (Rands 1917a).

Rotem (1964), using spore traps, found that

peak spore dispersal preceded the hottest and driest hour of the day by two hours, and the time of maximum wind velocity by four hours. A rapid rate of dispersal does not set in until infection has reached the stage at which whole leaves dry up and plants begin to die. The curve of wind velocity resembles that of spore dispersal. However, there appears to be no significant correlation between spore dispersal and other climatological factors (Rotem 1964). Rotem (1964) also postulated that there is a reservoir of spores that needs to be dispersed during the day, which is why the concentration of spores increases proportionally to disease incidence.

Infection

Spore germination is facilitated by free moisture, but can be induced by relative humidities close to saturation. Reports on the germination of spores give varying values for minimum, optimum and maximum temperatures. Bashi & Rotem (1974) noted that spores could germinate at 20 °C after only a two-hour wetting period. Elongation of the germ-tube requires a longer wetting period.

Germ-tubes form appressoria, and penetrate the epidermis directly or through wounds or stomata. According to Rotem (1994), Waggoner & Horsfall (1969) showed that, with a favourable inoculum dose and wetting period, the minimum temperature for infection can be as low as 10 °C, the maximum >35 °C, and the optimum between 20 °C and 30 °C. Incubation periods (time from infection to symptom development) vary greatly, depending on age and susceptibility of plants (Rowell 1953 in Rotem 1994).

Epidemics increase in severity after sandstorms, due to increased wounding of the epidermis (Rotem & Reichert 1964). The primary infections become necrotic with chlorotic halos. Mycelium from necrotic lesions produces conidia that infect healthy leaves and begin secondary infections (Shuman 1995).

Tubers are infected through wounds, as the conidia are unable to infect directly through intact periderm (Folsom & Bonde 1925; Venette & Harrison 1973). Wound healing, by suberisation and the development of wound periderm, reduces infection markedly.

Sporulation

On potato plants, sporulation occurs at temperatures of between 5 and 30 °C, with the optimum around 20 °C (Pscheidt 1985). The heaviest sporulation occurs after heavy rain or dew. Large numbers of spores are produced during alternating wet and dry periods (Rands 1917a; Bashi & Rotem 1975b). Spore production is initiated by daylight, but spores accumulate over a 7–14-day period and are then dispersed during the day (Bashi & Rotem 1975b).

Epidemics

Moisture plays a major role in the development of early blight. Studies have shown that free water is critical for disease development (Rands 1917a; Rotem & Reichert 1964) and that duration of leaf wetness can account for up to almost 90 % of variability in disease development and severity (Guthrie 1958; Holley et al. 1985). Increased leaf maturity, heavy fruit load, crowded plants, above-average rainfall or dew and shading also enhance early blight development (Horsfall & Heuberger 1942).

A. solani reacts differently to weather conditions, depending on the circumstances (Waggoner & Horsfall 1969 in Rotem 1994). In certain cases, weather factors may act indirectly by influencing the susceptibility of the host (Rotem 1994). Cooler temperatures may, for instance, retard the growth of the plant (Kreutzer & Durrell 1933 in Rotem 1994), while short photoperiods are associated with a decrease in sugar content in leaves (Bambawale & Bedi, 1982).

Epidemics do not generally occur until late in the season, when the plants are most susceptible. However, disease progress curves differ depending on location and prevailing weather conditions.

Control

Early blight can be controlled by efficient use of cultural practices, such as a 3–5-year crop rotation with non-host crops, site selection, sanitation of fields, providing proper plant nutrition, avoiding water stress and planting disease-free seed (Madden et al. 1978).

Generally, the best crops for rotation are forage crops and grains, including maize (*Zea mays* L.). A high frequency of potato or tomato cropping in one field, as well as consecutive plantings of potatoes or tomatoes, are associated with an earlier appearance of initial early blight lesions (Shtienberg & Fry 1990).

Planting cultivars that are less susceptible to early blight may also reduce disease severity.

However, Shtienberg & Fry (1990) showed that host resistance has no significant effect on the initial appearance of early blight.

Tuber infection can be decreased by allowing tubers to mature before harvesting, avoiding excessive wounding at harvest and providing storage conditions conducive to wound healing (Venette & Harrison 1973; Workman et al. 1983).

These various cultural practices can reduce the severity of early blight, but under situations of sufficient inoculum and environmental conditions favourable for disease, complete control will not be achieved.

The most effective control method is a protectant fungicide spray programme used from early in the growing season to vine kill (Jones 1912; Harrison et al. 1965a,b; Harrison & Venette 1970; Douglas & Groskopp 1974). In Colorado field trials, yields in plots treated with fungicide were approximately 20–40 % higher than in untreated plots (Harrison & Venette 1970), while in Minnesota chemical control of early blight resulted in yield increases of up to 90 % compared to unsprayed controls (Teng & Bissonnette 1985). Proper timing of initial and subsequent fungicide applications can reduce the overall number of sprays with no significant loss in yield. The most important consideration in the use of fungicides to control early blight is coverage. With aerial application of fungicides it is important to ensure that the lower, senescing leaves (where most of the early blight lesions occur) receive fungicide to prevent spread of the disease.

Forecasting of early blight

Early blight is a model disease for the development of forecasters and simulators. Although it is difficult to predict the first arrival of *A. solani* in potato fields, this is not important as the disease develops slowly while the plants are young.

Almost a century ago, Rands (1917a) suggested delaying fungicidal sprays until the plants were 15–20 cm high. Later, suggestions were made to initiate spraying only when plants become susceptible (at flowering) or, when the first symptoms are observed. Conventional spraying regimes are based on these observations.

Two models were subsequently developed to initiate fungicide sprays. The first model initiated sprays when secondary sporulation by *A. solani* is detected by a rise in spores trapped by weathervane spore traps (Harrison et al. 1965a,b). When Pscheidt & Stevenson (1986) evaluated this model, they found higher disease levels compared to other calendar-based spray regimes. Furthermore, dramatic increases in spore concentrations were not detected annually despite high disease severities. The second model developed used daily temperature data in a growing degree-day (GDD) model to forecast development of the first early blight lesions. The model calculates accumulation of day-degrees above 7.2 °C from the date of planting to predict the beginning of secondary spread of inoculum (Franc et al. 1988). Sands et al. (1979) used daily temperature data to calculate physiological days (p-days), which aided in the prediction of potato yields. These p-days might be used in the same way as GDD to help time the first fungicide spray of the season.

Madden et al. (1978) developed the FAST (Forecaster of *Alternaria solani* on Tomato) predictive system for initiating and timing fungicide sprays on tomato in Pennsylvania. The FAST system uses leaf wetness, air temperature, relative humidity and rainfall to calculate daily severity, and rating values that quantitatively represent conditions favourable for development of early blight (Madden et al. 1978; Pscheidt & Stevenson 1986). The FAST forecaster consists of two empirical models, compiled from numerous other works. Environmental parameter combinations are used to depict the relationship between *A. solani* and its microenvironment, and to determine periods when environmental conditions are favourable for early blight disease development (Madden et al. 1978). The first model uses daily severity (S) values that are determined by the combination of hours of leaf wetness and mean air temperature during the wetness period. The second model derives daily severity-ratings (R) from measurements of mean air temperature for the past five days, hours of relative humidity (RH) greater than 90 % for the past five days and total rainfall for the past seven days (Madden et al. 1978; Pscheidt & Stevenson 1986). The programme analyses the daily environmental data and keeps a record of (i) total of S values (TS) since the beginning of the growing season, (ii) total S values for the past seven days (cumulative severity value, CS) and (iii) five-day cumulative rating value (CR), the total of the R values for the past five days. When TS reaches a critical level of 35 and plants have been kept in the field for at least five weeks, the first early blight spray is recommended (Madden et al. 1978; Pscheidt & Stevenson 1986). Madden et al. (1978) showed

that spray recommendations based on environmental data and scheduled by the forecasting programme may control early blight as effectively and with fewer spray applications than the commercial schedules.

Shtienberg et al. (1989) developed a simulation model based on patterns of host development, resistance of the cultivars planted and efficacy of the fungicide used. Their observations showed that the economic benefits of fungicide sprays are low in the beginning and end of the season and high in the middle (Shtienberg & Fry 1990).

Early blight of potatoes in South Africa

History

The first recorded observation of early blight in South Africa was in KwaZulu-Natal by Fuller (1900) (in Nevill 1985). Further descriptions of the disease in South Africa included those of Fisher (1911), Doidge (1915), Jack (1913, 1916), Hector (1918), Wager (1931, 1945) and Gorter (1954). Between 1980 and 1984, a study was conducted to determine the epidemiology and control of potato early blight in the mist belt of the high-rainfall sourveld areas in KwaZulu-Natal (Nevill 1985). Since then, however, very few similar studies have been done in South Africa.

Distribution and importance

Doidge (1950) described *A. solani* as common and widespread on tomato in South Africa, but only listed the localities from which the fungus was recorded on potato in the country. However, in a subsequent publication, Doidge et al. (1953) indicated that potato early blight was extremely prevalent in all provinces and a limiting factor in production in late summer. The prevalence of the disease was confirmed by Trench et al. (1992), Denner & Theron (1999) and Crous et al. (2000). Early blight is currently one of three diseases taken into account when selecting new potato varieties in South Africa, the other two being late blight caused by *P. infestans* and common scab caused by *Streptomyces scabies* (Nortje et al. 2000). Heavy infection early in the growing season can cause yield losses of 20–50 % (Trench et al. 1992; Denner & Theron 1999).

Varietal resistance

Some potato cultivars planted locally are less susceptible than others, but none has total resistance. The four main cultivars, BP1, Buffelspoort, Up-to-date and Vanderplank, are all susceptible (Visser 1999; Nortje et al. 2000). Indeed, Vanderplank appears to be the most susceptible of all the commercial cultivars. Relatively high levels of resistance exist in the cultivars Mnandi and Ropedi.

Control

Despite the paucity of experimental work on the control of early blight in South Africa, guidelines have been proposed for the control of the disease (Trench et al. 1992; McLeod 1997). These include the following:

(i) Kill off the foliage at least 2–3 weeks before harvesting to prevent tuber infection.
(ii) Overhead irrigation promotes development of early blight by increasing the leaf wetness period. If only overhead irrigation is available, it should not be applied at night as this practice will increase spread of, and infection by, the pathogen.
(iii) Promote plant health and growth through balanced fertilisation.
(iv) Use tolerant cultivars if possible.
(v) Restrict volunteer plants and cull piles to reduce the number of fungal spores in the area.
(vi) Rotate with non-host crops.
(vii) Use certified seed.
(viii) Avoid harvesting too early or under wet conditions and do not bruise the tubers during harvesting and sorting.

Although certified seed and tuber sanitation are recommended as control measures, tuber infection by *A. solani* is not common in South Africa (McLeod 1997). Indeed, early blight is one of the very few diseases not specified in the South African Seed Potato Certification Scheme (Republic of South Africa 1998).

Like elsewhere, potato growers in South Africa rely mostly on the use of fungicides to control early blight. Almost as many fungicide formulations are registered for the control of early blight as for all other potato diseases together (Nel et al. 1999). The fungicides include chlorothalonil, difenoconazole, flusilazole + mancozeb, folpet, iprodione/mancozeb, mancozeb, mancozeb + procymidone, maneb/zinc oxide, metiram, propineb, tebuconazole, and various copper and fentin formulations. Regardless of the manufacturers' specifications, it is recommended that contact fungicides (e.g. chlorothalonil, mancozeb, maneb, copper and fentin formulations) be applied regularly in the

early stages of the disease to prevent infection. From early flowering onwards, 3–4 sprays of a systemic fungicide (e.g. difenoconazole, flusilazole, tebuconazole) should be applied. If symptoms appear before flowering, a systemic fungicide must be applied immediately (McLeod 1997).

Conclusion

Although the *A. solani* pathosystem on potato and tomato has been researched extensively, various aspects remain that need to be investigated. Major gaps in our knowledge of the epidemiology and economic impact of the disease still exist in South Africa.

The identification of *Alternaria* species based only on morphological characters is inadequate and more emphasis should be placed on the development of biochemical, serological and molecular methods. The use of additional molecular markers and larger population sizes should be paramount in any future study to confirm the existence of physiological races of *A. solani* (Weir et al. 1998). The biochemical aspects of sporulation still require attention, in order to explain the physiological processes during infection (Rotem 1994).

Another area of research that requires more extensive studies is the predisposition of the host to infection. Knowledge is also lacking about the physiology of resistance, for example the effects of senescence, stress and susceptibility as well as the role of toxins in pathogenesis (Nishimura & Kohmoto 1983; Rotem 1994). This information can be utilised in developing more effective control and breeding strategies.

Although overwintering and transmission of primary inoculum to host plants have been studied, little information concerning the biochemical modes of survival is available. This information can provide a sounder basis for control, focused on eradication of primary inoculum (Basu 1971; Pscheidt 1985; Pelletier 1988; Patterson 1991; Rotem 1994; Shuman 1995).

Although the control of early blight using disease-forecasting models has been extensively studied and is currently used in many countries, this is not the case in South Africa. The focus at present in South Africa is to develop a forecaster that schedules fungicidal sprays for both early and late blight, based on weather conditions and cultivars used in South Africa.

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