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Recent developments in managing tuber blight of potato (*Solanum tuberosum*) caused by *Phytophthora infestans*

O.M. Olanya, P.S. Ojiambo, R.O. Nyankanga, C.W. Honeycutt, and W.W. Kirk

Abstract: Tuber blight, caused by *Phytophthora infestans*, occurs wherever potato (*Solanum tuberosum*) is cultivated and accounts for significant losses under field and storage conditions. After decades of research in late blight, there has been substantial progress, but numerous challenges remain for the management of tuber blight. The tuber blight disease components comprising of infection pathways, tuber resistance, cultivar interactions, pathogen transmission, and survival on tubers are highlighted in this article. New fungicide chemistries, novel approaches, and cultural measures are presented along with the implications of pathogen diversity, cultivar differences, and edaphic factors on tuber blight incidence and control. With the application of molecular genetic tools in potato breeding to identify novel sources of resistance, effective control of the disease using host resistance is more likely. However, there are key elements of tuber infection and management that still need to be addressed. The quantitative relationships of inoculum load to tuber blight incidence and *P. infestans* interactions with edaphic factors are undetermined. Forecasting or development of predictive models for tuber blight incidence under field and storage environments will greatly improve disease management. Furthermore, quantification and characterization of the interactions between *P. infestans* and other storage pathogens (bacteria and fungi) or soil microbes, and the evaluation of biocontrol agents or alternative approaches for control of tuber blight may lead to effective management of this disease.

Key words: late blight, disease control, tuber rot.

Résumé : Le mildiou, causé par *Phytophthora infestans*, se retrouve partout où la pomme de terre (*Solanum tuberosum*) est cultivée et est responsable de pertes importantes tant au champ qu'en entrepôt. Après des décennies de recherche, les progrès sont notables, mais il reste de nombreux défis à relever quant à la gestion du mildiou. Dans cet article, les composantes de la maladie, incluant les voies d'infection, la résistance des tubercules, les interactions des cultivars, la transmission des agents pathogènes et le taux de survie des tubercules, sont mises en évidences. On y présente également de nouveaux fongicides, de nouvelles approches et méthodes culturales, parallèlement aux conséquences découlant de la diversité des agents pathogènes, des différences entre cultivars et des facteurs édaphiques quant à l'incidence du mildiou et à la lutte contre ce dernier. Grâce aux outils issus de la génétique moléculaire, utilizés dans la sélection des pommes de terre afin d'identifier de nouvelles sources de résistance, il semble de plus en plus probable que l'on trouvera des moyens efficaces de lutte contre la maladie faisant appel à la résistance de l'hôte. Toutefois, certains éléments clés relatifs à l'infection des tubercules et à sa gestion doivent être considérés. Les rapports quantitatifs relatifs à la quantité d'inoculum et l'incidence du mildiou, de même que les interactions de *P. infestans* avec les facteurs édaphiques, restent à déterminer. Les prévisions ou la conception de modèles de prévision quant à l'incidence du mildiou au champ ou en entrepôt faciliteront grandement la gestion de la maladie. De plus, la quantification et la caractérisation des interactions de *P. infestans* avec les autres agents pathogènes trouvés en entrepôt (bactéries et champignons), ou les microbes vivant dans le sol, ainsi que l'évaluation des agents de

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lutte biologique ou des approches alternatives de lutte contre le mildiou pourront aboutir à une gestion efficace de cette maladie.

Mots-clés : mildiou, lutte contre les maladies, pourriture fusarienne.

Introduction

Potato late blight, caused by *Phytophthora infestans* (Mont.) de Bary, has been a devastating disease for over 150 years. Losses due to this disease consist of yield reduction attributed to premature foliage death and tuber rots in the field and storage and excessive financial losses associated with fungicide use for disease control (Fry 2007; Guenthner et al. 2001; Wustman 2007).

Evidence over the last 30 years has indicated a major change in the population of *P. infestans* worldwide with emergence of more aggressive and fungicide-resistant strains (Fry 2007, 2008; Gavino et al. 2000). Prior to the 1980s, most of the *P. infestans* population consisted of the A1 mating type (US-1 genotype), with A1 and A2 mating types (US-8 genotype) confined exclusively to central Mexico. The recent genetic changes in *P. infestans* and corresponding increases in virulence has facilitated spread of US-8, displacing US-1 in North and South America, Europe, and other parts of the world (Fry 200[illegible]). The significance of the occurrence of both mating types in some locations is the potential ability of the pathogen to propagate through sexual recombination and produce oospores. This has necessitated the introduction of new fungicides with different chemistries in an attempt to effectively combat the disease (Andreu et al. 2006; Latorse et al. 2007; Stein and Kirk 2003). The occurrence of both mating types has also increased the survival of the pathogen and inoculum potential of oospores, which has potential epidemic implications.

Although late blight occurs mainly on potato (*Solanum tuberosum* L.) and tomato (*Solanum lycopersicum* L.), *P. infestans* infection of wild hosts belonging to the *Solanum* family, such as black nightshade (*Solanum nigrum* L.), hairy nightshade (*Solanum sarrachoides* Sendtn.), and common morning glory (*Pharbitis purpurea* (L.) Voigt), is common (Dandurand et al. 2006; Flier et al. 2003; Olanya et al. 2005, 2009). However, tuber blight occurs only on potato tubers.

Potato tubers in the field become infected by sporangia washed off or dislodged from diseased foliage. Unlike the foliage phase of late blight, the disease cycle on tubers has not been extensively investigated (Fig. 1). Precipitation and (or) irrigation are important for splash dispersal of inoculum from infected foliage and stems into the soil by increasing soil moisture levels. The level of disease severity on foliage, late blight management, susceptibility of tubers, environmental factors (soil temperatures, moisture or rainfall, soil structures), soil microbes, and cultural practices (hilling and tuber set) have been shown to impact tuber infection and development (Fig. 1). The relationships of inoculum levels to incidence of tuber blight have not been adequately documented. However, partial control of foliage infection prolongs duration of sporangia production, which may increase tuber blight (Naerstad et al. 2007a). Although temperature may be important for sporangia germination, zoospore release, and subsequent infection of tubers, below-g[illegible]d microflora on the tuber surface may also impact levels of tuber infection.

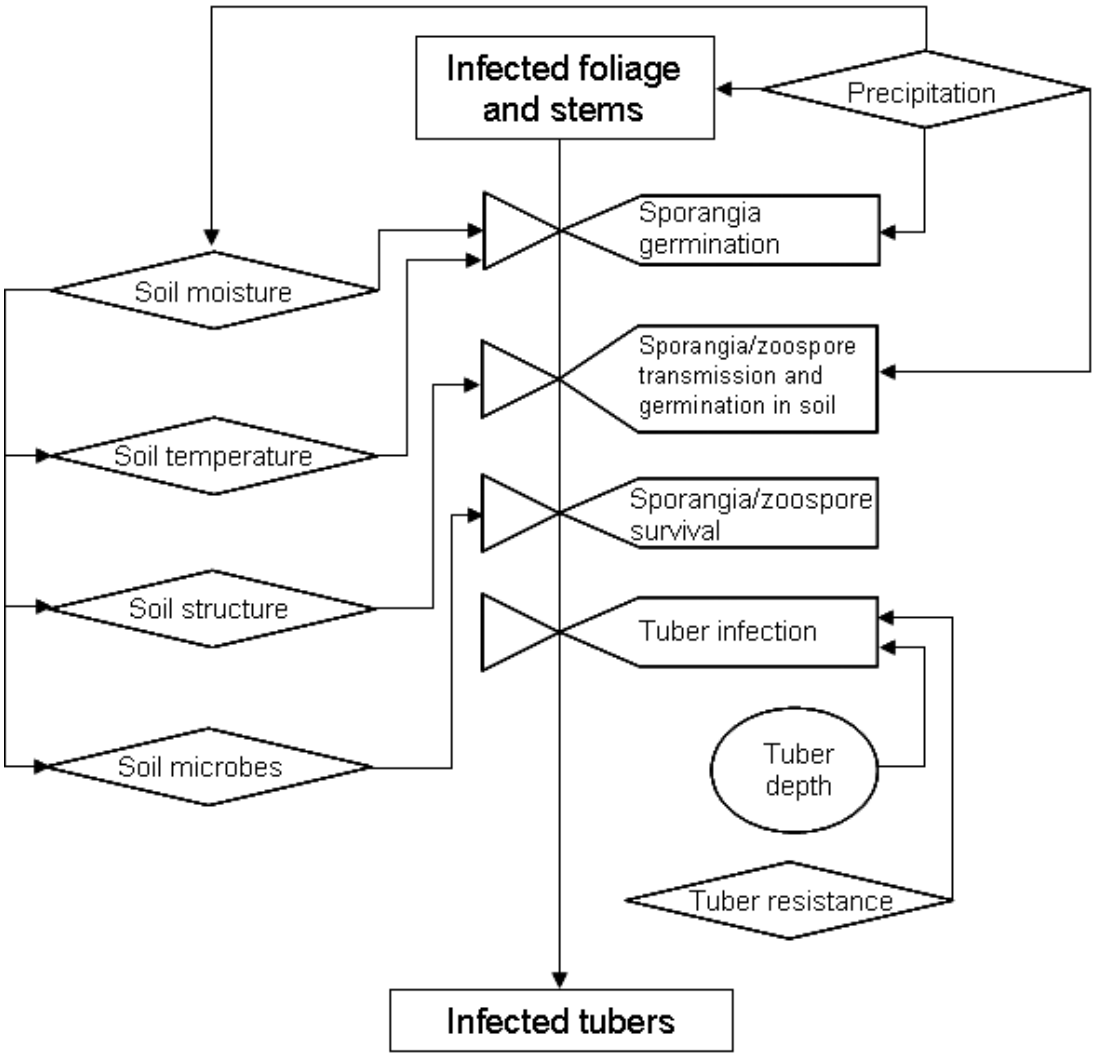
Reports on intensity of tuber blight infection are abundant in the literature, but there are no consistent data on levels of infection in tubers. The incidence of tuber blight varies greatly among cultivars, seasons, and growing locations or pathogen genotypes. In temperate latitudes where the A2 mating type is dominant, heavy losses attributed to tuber blight and its interactions with storage rot pathogens, such as the soft-rot bacteria (*Pectobacterium carotovorum* (Jones) Waldee emend. Gardan et al., synonym *Erwinia carotovora* (Jones) Bergey et al.) and leek (*Pythium ultimum* Trow.), have been reported (Wustman 2007). In tropical regions where multiple cropping cycles and only rudimentary storage options sometimes exist, incidence of tuber blight has been sporadic (Nyankanga et al. 2007; Olanya et al. 2006; Oyarzon et al. 2005). In this review, we discuss recent advances in managing tuber blight caused by *P. infestans*. Detection, infection, and development of *P. infestans* on tubers, characterization of tuber blight resistance, transmission and survival of *P. infestans* on blighted tubers, control of tuber blight (chemical, cultural, and host resistance) and challenges in the management of tuber blight are discussed.

Tuber blight detection and identification

Although identification of *P. infestans* can readily be accomplished from observations of sporulating lesions on diseased leaves, detection of late blight on tubers is difficult because of the presence of latent infections or soil residue that often mask symptoms (Hardwick 2006). Late blight detection on tubers, particularly those destined for storage, generally relies on visual and macroscopic observations of symptoms after cleaning (D[illegible]v 2004). Incubation of tubers or a baiting assay at 18[illegible]C is often used in laboratory or growth-chamber environments to induce symptom development for visual inspection. This technique can readily evaluate the presence of disease qualitatively but may not be well suited for pathogen detection in large quantities because of the subjective nature of visual assessments. Similarly, misidentification may also occur because symptoms of a disease such as pink rot (*Phytophthora erythroseptica* Pethybr.) may easily be confused with tuber blight, particularly during initial stages of symptom development.

Molecular techniques have been developed to detect and identify potato tuber blight and other potato diseases with higher frequency (Lees et al. 2005). Because visual disease inspections reveal only established infections and do not account for latent (nonsymptomatic) infections (Lees et al. 2005), use of molecular methods has been necessitated by

Fig. 1. Schematic pathway depicting infection of potato tubers by *Phytophthora infestans*. Precipitation or irrigation water may dislodge and deposit sporangia on tubers in soil. Soil moisture, soil temperature, soil structure, or soil microbes may influence survival or viability of sporangia in soil or on tubers. Sporangia, zoospore, or oospore germinate, depending on suitability of environmental conditions, and infect tubers in soil. The depth of tuber set in soil and tuber resistance (cultivar susceptibility) may also impact infection of tubers and blight development on tubers.



the need to detect low levels of pathogen and accurately quantify infection levels in tubers. Species-specific and sensitive polymerase chain reaction (PCR) assays have been used to detect *P. infestans* in leaves, stems, tubers, and soil (Hussain et al. 2005). In this approach, a *P. infestans* species-specific primer pair (INF FW2 and INF REV) was designed by comparing the aligned sequence of ribosomal DNA internal transcribed spacer regions of most known *Phytophthora* species. Specificity of the primers was demonstrated against DNA from nine other *Phytophthora* species and seven potato-blemish diseases (Hussain et al. 2005). In a nested PCR assay, using Peronosporales-specific primers, sensitivity of the assay was detected for as low as 5 fg DNA of *P. infestans* and as few as two sporangia or four zoospores were detected. Pathogen contamination of tubers and the relationships of inoculum load to blight development have been readily assessed based on the above assay. Sporangia survival, oospore inoculum, and the effects of control methods on *P. infestans* populations have been investigated using species-specific PCR primers (Hussain et al. 2005). Microsatellite markers, amplified fragment length polymorphisms, and random amplified polymorphic DNA have also been used to characterize *P. infestans* isolates or pathogen populations (Mahuku et al. 2000), but they have not been extensively employed for specifically detecting tuber blight or as diagnostic assays.

The advantage of molecular diagnostics is based on its ability to detect latent infection by *P. infestans* as opposed to well-established infections and the sensitivity of the assay. Very limited quantities of pathogen asexual (sporangia and mycelium) or sexual (oospores) structures are required for detection of *P. infestans* in plant tissues or tubers; however, if the pathogen is present in low frequency, then a large amount of plant samples should be assayed. Similarly, the specificity of the assay allows for the specific detection of *P. infestans* relative to other *Phytophthora* species or potato pathogens. The assay, which also requires relatively limited

amounts of tissue sam[illegible], is rapid and less labor intensive. The internal transcribed spacer regions of the rDNA are more highly conserved than other regions of the repeat and occur in multiple copies, leading to improved sensitivity (Wangsomboondee and Ristiano 2002; Judelson and Tooley 2000).

Regardless of whether visual inspection or molecular-based assays are used, seedlots, or tubers destined for storage present sampling problems because of the large number or volume of tubers. Accurate sampling strategies and optimum sample sizes are required for efficient pathogen detection. Molecular diagnostics cannot distinguish among the different *P. infestans* inoculum types that may be present in potato tubers, foliage, or soil. Because molecular techniques are based on pathogen DNA, it is also difficult to discern if pathogen propagules are viable or nonviable. Another drawback of the PCR assays for detection of *P. infestans* in tubers, leaf tissues, or soil under field conditions may also be attributed to presence of PCR inhibitors in degraded leaf tissues, tubers, or soil (Judelson and Tooley 2000; Lees et al. 2005). On the other hand, visual examination or symptom-based identification is time consuming, expensive, and not very practical (Hussain et al. 2005).

Enzyme-linked immunosorbent assay kits or portable PCR equipment may be used for tuber blight detection at various potato handling or loading sites if qualitative analysis is required. However, there are many challenges for detecting *P. infestans* on tubers. Techniques that detect volatile compounds released from tubers infected by various pathogens (Lui et al. 2005) or rotting tubers would greatly aid in early detection of tuber rot and its associated pathogens (Hardwick 2006).

Infection and development of tuber blight

Infection of potato tubers by *P. infestans* may be initiated by zoospores, sporangia, or oospores washed from plant foliage and deposited in soil (Fry 2008). Soilborne oospores of *P. infestans* are rare in most potato production areas but have been shown to occur in some instances, for example, in the Toluca Valley of Mexico (Fernández-Pavía et al. 2004). Many of the sporangia produced in the lower half of the leaf canopy and deposited or washed onto the soil surface may be degraded or rendered nonviable; however, depending on environmental conditions, some sporangia or zoospores can infect tubers in soil (Mayton et al. 2007). Sporangia produced in the upper half of the leaf canopy can also be aerially dispersed and may initiate foliar infection at some distance away from the initial infection foci (Aylor et al. 2001). Again, inoculum produced on diseased foliage may be washed by rain or irrigation and deposited on the surface of the potato hill for subsequent tuber infection (Porter et al. 2005).

Tuber infection may also be enhanced through abrasion during harvest or incidental wounding if diseased tubers or sporangia come into contact with nonwounded tubers (Porter et al. 2005; Van den Bosch and Kiessel 2007). Similarly, tuber abrasion from harvest or wounds created by insects such as potato tuber moth (*Phthorimaea operculella* Zeller) or infection by bacterial soft rot *Pectobacterium carotovorum* may induce wounds and also serve as additional infection points for tuber blight (Nyankanga et al. 2007). Under natural conditions, tubers become infected by *P. infestans* through wounds, buds, lenticels, and intact skin (Darsow 2004), although many barriers exist in tubers that can prevent development of tuber infection (Glass et al. 2001). Wounds are considered most susceptible, buds and lenticels are moderately susceptible, and intact skin is the least susceptible to infection by *P. infestans* (Darsow 2004; Nyankanga et al. 2008). Harvesting tubers prior to natural haulm senescence often leads to greater tuber rot, suggesting that tuber resistance increases as tubers mature (Miller et al. 2002). The periderm, which consists of several layers of phellem cells can provide a good barrier to infection of tubers by *P. infestans*. Physiological properties of tubers related to disease susceptibility, such as suberin formation and integrity of cortical tissues, are dynamic during growth and storage periods. Suberin formation, which occurs after wounding and at tuber maturity, usually increases in storage. Once infection occurs, various symptoms and manifestations of light brown, speckled, mottled, or brown lesions can be observed along the tuber surface (Fry 2008). The cellular reaction of potato tubers following infection varies with cultivar susceptibility, and considerable research is still lacking in this area.

The relationship between foliar blight level and tuber blight incidence has been shown to be variable. Some studies have reported low tuber blight incidence at harvest concurrent with an apparent absence of foliar blight (Nyankanga et al. 2008). In other studies, low incidence of tuber blight was found after high levels of foliar blight (Olanya et al. 2006). Similarly, research reports have indicated high levels of tuber infection even in fields where numerous applications of protectant and curative fungicides were applied (Nyankanga et al. 2007). This variability has been attributed to the differential effects of soil temperature, moisture, cultivar resistance, architectural characteristics, levels of foliage blight, inoculum for tuber infection, and management practices (Nyankanga et al. 2007).

Characterization of tuber blight resistance in potato cultivars

Eleven *R* genes from *Solanum demissum* Lindl. have been introduced into the cultivated potato to confer resistance to foliage blight. Five of these, *R1*, *R2*, *R3*, *R6*, and *R7*, have been mapped and shown to confer race-specific resistance (Li et al. 1998). Unlike foliage resistance, the genetics of tuber blight resistance have not been extensively studied. Further, the expression of resistance for tuber or foliage late blight depends on the *R* genes within the study population. Park et al. (2005) found that, in their RH4X-103 population, *Rpi-abpt* and *R3a* genes were foliage specific, whereas *R1* or *R1-like* genes acted both in tuber and foliage. In addition, resistance expression depends to a large extent on testing conditions and, therefore, is difficult to evaluate (Świeżyński and Zimnoch-Guzowska 2001). The susceptibility of tubers to blight infection is dependent on tuber ontogeny. Increase in tuber resistance occurs as tubers mature, with the most stable expression of tuber resistance observed between 16 and 28 weeks after planting (Lebecka et al. 2006).

Late blight resistance in tubers can be attributed to three major components (Pathak and Clarke 1987). The first defense barrier consists of several layers of phellem cells, known as the periderm. When intact, the periderm provides an absolute barrier to infection. Tuber infection can only take place through buds, lenticles, or cracks and wounds of the periderm. The second defense barrier is located in the peripheral layers of the cortex. The outer cortical cell layers may retard the growth of lesions and can completely block hyphal growth. The third barrier is located in the storage tissues of the tuber known as the medulla. This resistance component is characterized by reduced hyphal growth and sporulation of *P. infestans*. Although potato cultivars with resistance gene *R1* confer a strong hypersensitive reaction in the cortical region when inoculated with incompatible isolates, other *R* genes do not seem to inhibit the growth of *P. infestans* after tuber infection (Flier et al. 2001). It has been documented that many more cells are involved in the hypersensitive-like reaction in the cortical region, compared with the hypersensitive response in the foliage (Pathak and Clarke 1987). In their study, Flier et al. (2001) concluded that it is very unlikely that the existing gene-for-gene pathosystem in potato is responsible for the observed differential interaction in tuber infection. They observed the presence of cultivar and isolate-cluster discrepancies for the three components of tuber blight resistance (attributed to periderm, cortex, and medulla) suggesting that these three resistance components might be under different genetic control. Therefore, the defense responses in the tuber could be under the control of several putative quantitative trait loci (QTLs).

Advances in computer technology have led to an increased interest in extending linkage analysis and QTL mapping methods from diploid to polyploidy plant species. In potato, *R* genes and QTLs have been localized as new sources for foliage resistance against *P. infestans* (Bradshaw et al. 2004; Kuhl et al. 2001; van der Vossen et al. 2004). Park et al. (2005) analyzed tuber resistance in three mapping populations carrying *R* genes or a major QTL for foliar resistance to late blight. In one mapping population, tuber blight resistance was inherited independently of foliar blight. In this population, two *R* genes (*R3a* and *Rpi-abpt*) functioned as foliage-specific *R* genes, whereas the *R1* (or *R1-like*) gene acts on both the foliage and tuber. In their remaining two populations, tuber and foliar blight resistance were significantly correlated, suggesting that both traits are conferred by the same gene (possibly *R3b*) and QTL, respectively. One of the *R* genes reported by Park et al. (2005), *Rber*, located on chromosome 10 that carries a major QTL has also been reported by Simko et al. (2006). The *Rber* gene is derived from *Solanum berthaultii* Hawkes (Ewing et al. 2000), whereas the QTL originated from *Solanum phureja* Juz & Bukasov or *Solanum stenotomum* Juz & Bukasov. In a separate study, Simko et al. (2006) reported that four chromosomal regions were significantly associated with tuber resistance to *P. infestans*. The largest effect was detected near the marker locus *PSC* located on chromosome 10. This locus explained about 63% of the total phenotypic variation of the trait. The other three resistance-related loci were mapped on chromosomes 8, 6, and 2. None of the four tuber-resistance loci coincided with the foliage-resistance loci detected in this same family. Tuber blight resistance QTLs on chromosomes 2, 8, and 10 were distinct from the maturity QTLs and have an additive effect on tuber resistance. Bradshaw et al. (2006) reported a QTL on chromosome 4 that confers resistance in both foliage and tubers, which was selected in breeding for quantitative resistance.

Transmission of *P. infestans* and survival potential on blighted tubers

The removal and deposition of *P. infestans* sporangia from foliage and stems on tubers in soils often initiates the tuber infection process. Once tubers are infected, transmission of *P. infestans* from diseased tubers to nondiseased tubers is one of the pathways for late blight spread (Kirk et al. 1999; Lambert et al. 1998). Tuber to tuber infection by contact is crucial for localized transmission within the same plant, among neighboring plants, or among tubers in storage environments (Fairclough et al. 1993). The distance that *P. infestans* can grow from infected tubers to healthy tubers in soil is approximately 1.3 cm within the same plant, depending on temperature and moisture conditions (Fairclough et al. 1993). At 75% field capacity, the passive spread of the pathogen from diseased or inoculated tubers to healthy tubers in neighboring plants has been recorded over a distance of 60 cm, even resulting in infection of daughter tubers from seed (Fairclough et al. 1993). Transmission of *P. infestans* from infected tubers to healthy tubers during storage or seed handling, particularly where seed cutting is practiced, may also pose considerable risk for tuber blight spread (Lambert et al. 1998). Once tubers are infected, the rate of spread or development of blight in tissues of potato tubers depends largely on storage temperature (Kirk et al. 2001). Long-distance spread of late blight through latently infected seed tubers across regional, national, or international boundaries has been suspected, but no concrete evidence is available.

The survival of *P. infestans* in infected tubers either in storage or in the field until the following season has been well documented (Gigot et al. 2009). It has been hypothesized that survival potential depends on pathogen aggressiveness. Although this hypothesis has not been thoroughly examined in potato growing regions in the tropics or North America, it has been investigated in Europe. For example, the trade-offs between aggressiveness and overwinter survival of *P. infestans* on tubers have been investigated and documented. Aggressive strains that are favored during epidemics in a cropping cycle exhaust their nutrient supply too early to bridge seasons, resulting in limited survival compared with less-aggressive strains (Montarry et al. 2007). In any case, overwinter survival of *P. infestans* on tubers or cull piles was demonstrated to vary with pathogen population. The thermal properties of the cull piles or temperature regimes during that period also affect survival potential of the pathogen.

The survival potential of *P. infestans* is also aggravated by the fact that the pathogen may infect and possibly survive on alternate hosts or volunteer plants (Flier et al. 2003) depending on the environmental conditions during the off season. In situations where poor-quality seed is used, tubers

may easily be degraded and deposit pathogen structures in the soil. Reports from studies on oospore formation in potato tubers indicate that these fungal propagules may easily be deposited in soil (Levin et al. 2001). Although only a limited number of infected tubers have been shown to have oospores (Levin et al. 2001), the potential for pathogen survival is exacerbated, given the fact that soilborne oospores have been found in certain potato production regions, such as the Toluca Valley of Mexico (Fernández-Pavía et al. 2004).

Control of tuber blight by chemicals and cultural practices

Fungicide application and planting of resistant cultivars to control foliage blight and foliage desiccation before harvest are the most common methods for reducing tuber infection (Cooke and Little 2001; Daayf and Platt 2002). However, adequate control of foliar blight does not necessarily lead to effective control of tuber blight (Nyankanga et al. 2007). As a result, there have been efforts to directly control tuber blight with fungicides. Foliage application of contact, systemic, and curative fungicidal compounds have been shown to be effective in minimizing foliar blight and destruction of sporangia and, therefore, indirectly reducing the levels of tuber infection by *P. infestans* (Latorse et al. 2007; Stein and Kirk 2003). Application of contact fungicides, such as fluazinam (0.4 L·ha^{-1} with a dose of 200g a.i./ha) and mancozeb (2.4 kg·ha^{-1}), prevents tuber infection by inhibiting sporangia production and indirect germination of sporangia (Nærstad et al. 2007a; Latorse et al. 2007). Fungicidal compounds with translaminar or systemic activity such as propamocarb + chlorothalonil (Tattoo C),[2] dimethomorph + mancozeb (Acrobat), and cymoxanil + mancozeb have been shown to reduce the formation of sporangia on foliage or have sporicidal effects, thereby minimizing available inoculum for potential infection of tubers (Latorse et al. 2007).

Different trials have shown that phosphonites or phosphates can suppress tuber and foliar late blight when applied at intervals of 7–14 days (Cooke and Little 2001; Lobato et al. 2008; Mayton et al. 2007; Miller et al. 2006). When applied to foliage, phosphonates such as aluminium tris *O*-ethyl phosphanate (3.6 kg a.i.·ha^{-1}), dipotasium phosphonate (2.1 kg a.i.·ha^{-1}), and potassium phosphate (2.1 kg a.i.·ha^{-1}) can move systemically to the roots and developing tubers and reduce tuber infection (Mayton et al. 2007). The mode of action of the phosphonate fungicides is thought to be both direct and indirect. Direct effects include inhibition of mycelial growth, reduction or alteration of membrane metabolism, and phosphorylation reactions in the pathogen, as well as suppression of sporulation and germination. In the above, fungicidal compounds are applied preharvest, and their effectiveness in minimizing tuber blight is based on limitation of sporulation of *P. infestans*, inhibiting viability, or killing sporangia and zoospores before they can infect tubers. The concentration of compounds, timing of application relative to rainfall, and role of rainfall and irrigation in the redistribution or dilution of fungicides may affect the effectiveness of compounds applied during crop growth in reducing the incidence of blighted tubers.

Indirect effects of chemicals such as phosphonates have been reported to be due to the activation of plant defense mechanisms (Andreu et al. 2006). For example, foliar application of aminobutyric acid at 40 mmol·L^{-1} (equivalent of 4 kg·ha^{-1}) at 3 mL·plant–1 at 15, 35, 55, and 75 days and fosetyl-aluminium at 3 mL·plant–1 (3 kg a.i.·ha^{-1}), applied at the same time to foliage has been shown to increase the resistance of potato foliage through induction of systemic acquired resistance (SAR) to late blight (Andreu et al. 2006). Similarly, a significant increase in tuber resistance to late blight was detected on 'Shepody' and 'Kennebec' potatoes (Andreu et al. 2006). This approach highlights the potential use of SAR as a technology for integrated control of foliar and tuber blight by detailing application time, persistence of resistance induced and.molecules associated with SAR in *P. infestans* pathosystem. A drawback to this methodology is that evidence for enhancement of defense response was evaluated in postharvest tuber samples harvested from plants in which foliage was treated with the above chemicals. Therefore, the question arises as to whether resistance to foliage and tuber infection would be displayed if plants were challenged or subjected to late blight infections during periods of potato growth.

Disinfectants such as chlorine dioxide (2–122 mg·kg^{-1}) and mixtures of hydrogen peroxide and peroxyacetic acid are often used for postharvest management of tuber diseases in the potato industry. These have been evaluated for control of tuber blight and potato diseases, such as soft rot (*Pectobacterium carotovora*), dry rot (*Fusarium* spp.), and silver scurf (*Helminthosporium solani* Durieu & Mont.), which often interacts with *P. infestans* in storage (Olsen et al. 2003). The application of chlorine dioxide on tubers destined for storage at the rate of 400 mg·kg^{-1}, followed by a secondary application of 200 mg·kg^{-1} with additional daily application of activated sodium chlorite at 50 mg·kg^{-1} resulted in significant reduction in coverage of late blight lesions on tubers of potatoes (Olsen et al. 2003). In postharvest application studies, zoxamide at the rates of 0, 2, 9, and 17 g a.i.·t^{-1} of tubers, significantly reduced incidence and severity of late blight and pink rot when applied immediately after inoculation with the pathogen (Miller et al. 2006). Foliar applications of zoxamide have exhibited both foliar and tuber protection against *P. infestans* in potatoes (Miller et al. 2006; Olsen et al. 2003). Zoxamide functions by arresting nuclear division and destroying the microtubule cytoskeleton of oomycete pathogens (Young and Slawecki 2001). Hydrogen peroxide and peroxyacetic acid mixtures provided inconsistent control of potato diseases when applied to prevent disease development at harvest (Kimes 2002). For example, peroxyacetic acid applied at 9 g a.i.·t^{-1} was less effective in controlling disease development, whereas calciun and potassium phosphite salts applied to seed tubers at 1% (*v*/*v*; equivalent to 3 L·ha^{-1} of commercial product) were effective at reducing late blight development

[2] Mention of trade names or commercial products in this publication is solely for the purpose of providing specific information and does not imply recommendation for endorsement by the U.S. Department of Agriculture, North Carolina State University, University of Nairobi, or Michigan State University.

at all time intervals up to 6 h postinoculation. Zoxamide appeared to have good postharvest disease control if applied soon after inoculation (Miller et al. 2006) but was less effective as the interval between inoculation and treatment increased. The study concluded that phosphites are highly effective postharvest management tools for controlling late blight and pink rot. Other studies have similarly found phosphites to be effective against *P. infestans* when applied postharvest (Lobato et al. 2008). In the study by Lobato et al. (2008), calcium and potassium phosphite were applied to cut seed pieces at 1% (*v*/*v*, equivalent to 3 L·ha^{-1}) followed by inoculation of seed pieces ('Shepody' and 'Kennebec' potatoes) with zoospore suspension of *P. infestans* isolates (A2 mating type) and then planted. Control of late blight based on emergence of seed pieces was significantly higher with potassium than with calcium phosphite treatments (Lobato et al. 2008). The effectiveness of potassium and calcium phosphites is not known if treatments are applied after inoculations with *P. infestans*. Other salt compounds, such as sodium metabisulfite, sodium hypochlorite, copper sulphate pentahydrate, and acetic acid, that were applied to tubers in storage at concentrations of 0.002, 0.02, and 0.2 mol·L^{-1} were effective in inhibition of late blight and pink rot when tubers were inoculated with sporangia or zoospores at 0, 1, 2, 4, and 6 h before or after treatment (Mills et al. 2005). These salt compounds also effectively inhibition of mycelial growth, sporulation, and spore germination of numerous potato pathogens (Mills et al. 2004). Postharvest applications of disinfectants appear to offer practical solutions for management of tuber blight and other storage diseases because of the low concentrations and the relative ease of compound application in huge storage bins. Differences in concentrations of active ingredients (formulations) and lack of optimization of application strategy are limiting factors to the compounds' efficacy for tuber blight control (Miller et al. 2006; Olsen et al. 2003). Although this approach may be partially effective in preventing diseases on the surfaces of tubers, the extent of their effectiveness on established infections in tubers is not known. Additionally, phytotoxicity of the postharvest compounds to potato tubers and human toxicity or antigenicity may also affect the preference for their utilization.

Other agronomic and cultural practices may also be used to reduce incidence of tuber blight caused by *P. infestans*. Shallow tubers may be more prone to infection than deeper set tubers (Nyankanga et al. 2008). In this regard, potato hills and mulches are used as a cultural practice that can protect tubers from blight by filtering spores out of the soil water suspension and by reducing direct contact of inoculum with tubers (Glass et al. 2001; Nyankanga et al. 2008). However, with the rise of more aggressive strains, the effectiveness of this practice is variable. Field experiments involving US-8 strains showed that hilling provided partial protection of tubers, but its effectiveness was limited under favorable conditions for foliar late blight development. Even though large hills had a proportionally lower tuber blight incidence than medium-sized hills, the difference between hill sizes was not significant (Glass et al. 2001; Nyankanga et al. 2008). The effectiveness of mulches has been variable. Black polyethylene film and copper hydroxide treated agricultural textile reduced the incidence of tuber blight, whereas polyurethane spray foam (Glass et al. 2001) and straw hay mulch (Nyankanga et al. 2008) were ineffective for controlling tuber blight. These results highlight the difficulty of direct suppression of tuber blight in a field with barriers once the foliar stage of the disease has been allowed to develop.

Haulm destruction prior to potato harvest has been shown to decrease the risk of tuber infection. Mechanical top removal or desiccation of potato haulms and foliage 2–3 weeks prior to harvesting tubers is recommended to eliminate the foliage source of tuber blight inoculum (Miller et al. 2002; Naerstad et al. 2007b). Water management has also been investigated for reducing foliar and tuber blight, particularly in production regions requiring irrigation. Management of irrigation frequency and duration may be adjusted to minimize the microclimate conditions that are conducive for late blight (Johnson et al. 2003; Olanya et al. 2007). In comparison with other options, cultural practices offer a simple approach for management of tuber blight that are easy to implement and less costly. A major disadvantage of this approach is that effectiveness is dependent on the time and duration that cultural measures are implemented relative to availability of *P. infestans* inoculum. Data on tuber blight development relative to inoculum load and cultural measures are required to assess the effectiveness of this approach. Moreover, environmental conditions (rainfall, irrigation water, soil texture and type, and temperatures) may enhance or limit the effectiveness of cultural options. It appears likely that the most effective management of tuber blight will require a spectrum of controls that includes use of disease-resistant cultivars and cultural practices.

Future challenges in managing tuber blight

Currently, the most reliable method for controlling foliage and tuber blight is an integrated approach that utilizes an array of tactics including the planting of less susceptible cultivars and application of fungicides. Presently, there are no commercial potato cultivars that are resistant to tuber or foliage blight. It would be notable if a single *R* gene were completely durable and could be bred or engineered into cultivars. Potato growers worldwide would prefer such a simple, highly effective management strategy in comparison with the complex array of management strategies currently required (Fry 2008). However, because potatoes are tetraploid and highly heterozygous, significant improvements are slow even with advances in genetic engineering.

As plant breeders and plant molecular biologists now focus intensively on developing resistant plants and discovering mechanisms to engineer resistance (Smart and Fry 2001), progress is likely, in hopes that consumer acceptance will also follow. The obvious approach to manage tuber and foliar late blight is to combine the various types of tuber and foliar resistance already identified. With increased understanding of the molecular aspects of the *P. infestans* – potato interaction (Birch and Whisson 2001) and progress in genetic engineering techniques, a breakthrough in resistance breeding may be expected (Melchers and Stuiver 2000). For example, overexpression of tobacco (*Nicotiana tabacum* L.) osmotin, a PR-5 protein, in potato has been

shown to significantly delay the appearance of lesions caused by *P. infestans* (Zhu et al. 1996), and the constitutive expression of a H_2O_2-generating enzyme, glucose oxidase, has been shown to provide resistance to *P. infestans* (Wu et al. 1995). Future research will need to focus on the successful integration of these transgenic strategies into breeding programs to develop durable resistance to tuber blight in new commercial cultivars, in combination with a selective use of fungicides.

The introduction of the A2 mating type of *P. infestans* into the United States in the latter part of the 20th century has dramatically increased the complexity of managing this oomycete because migrating strains were resistant to metalaxyl ~~compound~~, which had been particularly effective at suppressing both foliage and tuber blight. The sexual reproductive potential of *P. infestans* has greatly increased, and with pathogen diversity, these new strains have resulted in greater pathogenic fitness than the previously dominant A1 mating type strains (Smart and Fry 2001). In turn, this has posed a series of challenges in managing the disease on foliage and tubers.

In locations where sexual reproduction occurs, it is possible for *P. infestans* to survive as oospores in field soil or in infected tubers (Levin et al. 2001; Fernández-Pavía et al. 2004). Even though *P. infestans* survival from one season to the next in Canada and the United States is still via infected potato tubers that overwinter~~ed~~ in fields, storage bins, and cull piles in the temperate zones, increased pathogen diversity, virulence, and sexual reproductive potential makes it difficult to manage tuber blight. In the United States, Canada, and many other potato growing regions of the world, reports of oospore occurrence in field soil have not yet been documented.

In part, the lack of widely adapted potato cultivars that are resistant to tuber or foliage blight has justified the continued use of fungicides in potato production, and the availability of effective fungicides has enabled the continued use of cultivars susceptible to the disease. However, concern about the sustainability of potato production (environment, health risks associated with pesticide use, and increasing cost of pesticide applications) have stimulated efforts to reduce the fungicide inputs in potato production systems. Although forecasting and decision-support systems are routinely used for timely application of fungicides to reduce pesticide input and manage foliar blight, a paucity of information exists with regard to the tuber blight pathosystem. In part, this is due to the numerous edaphic factors (atmospheric and soil variables) that influence tuber blight incidence and severity, making it difficult to predict tuber blight occurrence. Attempts to model or predict tuber blight have resulted in inconsistent results and a lack of good model performance (Nyankanga et al. 2007). It is possible to develop a more comprehensive model of tuber infection pathways based on Fig. 1. Researchers in the Netherlands have developed a tuber blight prediction model (Hadders 2007). The model is not commercially available to growers but is mainly used by consultants to analyze the cropping season of potato fields that have shown signs of tuber infection. Based on this model, it is also possible to identify when protection failed; however, precise quantification of eventual tuber infection in storage has proven difficult.

Future research should identify, assess, and quantify the precise variables that influence the dynamics of tuber infection (inoculum load and types, soil temperature, soil moisture, soil microbes, tuber ontogeny, and chemical and cultural practices) under field and storage conditions and use the information in predicting tuber blight infection in the field and in storage environments under various disease management scenarios. Research is needed to understand the interactions of *P. infestans* spore types with soil microbes (pathogens and saprophytes) and how they impact tuber blight development under field conditions and in storage. The development and application of biorationals (plant extracts) and biocontrol (microbial antagonists and biopesticides) thath are antagonistic, inhibitory, or suppressive to spore types (mycelia, sporangia, zoospores, and oospores) of *P. infestans* and to tuber blight infection and disease development should be examined. Resistant chemical inducers such as chitosan, phosphonate, and other compounds already mentioned that could induce resistance in potato plants or tubers to infection by *P. infestans* should be explored. Early detection of tuber blight in field and storage can be a critical component of controlling tuber blight. Already, volatile metabolic profiling of compounds based on gas chromatography and mass spectrophotometry are being used to discriminate volatiles emitted from potato tubers inoculated with dry and soft rot pathogens (Lui et al. 2005). Such a technique could be examined and applied to early detection of late blight in tubers. Development of dipstick serological methods for antibody screening of potato tubers infected by *P. infestans* will be particularly useful for random testing of individual suspect tubers in field and storage sites. This can address the sampling problem by increasing the number of samples that can be assayed for *P. infestans*, thereby minimizing variances due to limited sample size. High throughput techniques, such as microarra[illegible]r detection of multiple pathogens causing tuber rots or scar markers for specific detection of *P. infestans* on tubers, would definitely enhance epidemiological research and management of tuber blight in field and storage environments. Therefore, quantification and elucidation of the above research questions and factors are required for better understanding and managing potato tuber blight.

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