Advances in Management of Late Blight of Potato

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Abstract

This chapter discusses about late blight, caused by the oomycete *Phytophthora infestans*, the main biotic threat to potato production. The pathogen evolves continuously, mainly through recombination and migration; hence, monitoring of *P. infestans* populations is critical for the development of effective management strategies. The population structure and its monitoring, symptomatology, and pathogenesis are discussed in the present chapter. No single approach is effective; hence, combination of approaches in an integrated manner is essential to combat this disease and is discussed here.

Keywords

Phytophthora infestans · Population structure · Symptoms · Host resistance · Disease forecasting · Alternative approaches · Decision support system

7.1 Introduction

Late blight, caused by the oomycete *Phytophthora infestans* (Mont.) de Bary, has historically been an important disease of potatoes and tomatoes worldwide. It continues to be the main biotic constraint of potato production and has been considered a threat to global food security (Cooke et al. 2012). Losses due to *P. infestans* have been estimated to € 12 billion per annum of which the losses in developing countries have been estimated around € 10 billion per annum (Haverkort et al. 2009). Studies conducted in the United States to estimate the impact of late

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S. Kumar Chakrabarti et al. (eds.), *Sustainable Management of Potato Pests and Diseases*, https://doi.org/10.1007/978-981-16-7695-6_7

blight on potato yield and fungicide use revealed that use of the fungicides alone cost $ 77.1 million at an average cost of around $507 per ha which do not include non-fungicide control practices (Guenthner et al. 2001). Region-wise, economic importance of late blight shows that the disease takes highest toll of potato in sub-Saharan Africa (44% crop losses) followed by Latin America (36%), Caribbean (36%), South-East Asia (35%), South-West Asia (19%), and Middle East and North Africa (9%) (CIP 1997). *Phytophthora infestans* is considered as re-emerging pathogen due to regular emergence of its novel strains with increased virulence and its appearance in new locations with surprising intensity (Fry et al. 2015). Management of this devastating pathogen is challenged by its remarkable speed of adaptation to control strategies such as genetically resistant cultivars and fungicides. In the present communication, efforts have been made to discuss about the pathogen, its population structure, symptoms, pathogenesis, and recent advances in the management of the pathogen/disease.

7.2 The Causal Organism

Oomycetes are a diverse group of organisms that morphologically resemble fungi, yet are members of the *Straminipile* (= Stramenopile), and are more closely related to organisms in aquatic environments such as brown algae and diatoms. These are the members of the Kingdom Chromista (Dick 2001; Cavalier-Smith and Chao 2006; Beakes et al. 2012) under Super Kingdom Chromalveolata (Baldauf et al. 2000; Yoon et al. 2002). *P. infestans* is a heterothallic oomycete with both sexual and asexual reproductive cycles. With few exceptions, for example, Toluca Valley, Mexico, Scandinavia, and the Netherlands (Bruberg et al. 2011; Drenth et al. 1993a, b; Fry et al. 2015; Yuen and Andersson 2013), the asexual reproductive cycle dominates resulting in the development of distinct clonal lineages. The vegetative stage of the mycelium in *P. infestans* is diploid, while in true fungi, it is haploid. However, recent studies have shown that progenies from sexual *P. infestans* populations in the modern-day lineages are diploid, but the most important pandemic clonal lineages are triploid (Li et al. 2017). The size of the *P. infestans* genome is considerably larger (240 Mb) and by far the largest and most complex genome sequenced so far in the chromalveolates and even in true fungi. A total of 17,797 protein-coding genes have been detected within the *P. infestans* genome. Overall, the genome is having an extremely high repeat content (~74%) and to have an unusual gene distribution, which is thought to contribute to *P. infestans* evolutionary potential by promoting genome plasticity, thus enhancing genetic variation of effector genes leading to host adaptation (Haas et al. 2009).

Virulence of oomycetes depends on rapidly evolving protein families including extracellular toxins, hydrolytic enzymes, and cell entering effectors that help the pathogen suppress the host plant defenses and gain nutrition from the host (Jiang and Tyler 2012). *P. infestans* secretes large numbers of effectors: apoplastic effectors that accumulate in the plant intercellular space (apoplast) and cytoplasmic effectors that are translocated directly into the plant cell by a specialized infection structure called the haustorium (Whisson et al. 2007). Apoplastic effectors include secreted

hydrolytic enzymes such as proteases, lipases, and glycosylases that probably degrade plant tissue, enzyme inhibitors to protect against host defense enzymes, and necrotizing toxins such as the Nep1-like proteins (NLPs) and PcF-like small cysteine-rich proteins (SCRs). At least 563 RxLR genes have been predicted in the *P. infestans* genome. RxLR effectors act as activators of plant immunity, resulting in effector triggered immunity (ETI) (Oh et al. 2009; Wang et al. 2017), while the apoplastic effectors act as activators of the PAMP-triggered immunity (PTI) (Domazakis et al. 2017). All oomycete avirulence genes (encoding products recognized by plant hosts and resulting in host immunity) discovered so far encode RxLR effectors that define a domain required for delivery inside plant cells, followed by diverse, rapidly evolving carboxy-terminal effector domains (Jiang et al. 2008). CRN cytoplasmic effectors were originally identified from *P. infestans* transcripts encoding putative secreted peptides that elicit necrosis *in planta*, a characteristic of plant innate immunity (Torto et al. 2003). Analysis of the *P. infestans* genome sequence revealed an enormous family of 196 CRN genes of unexpected complexity and diversity. Like RXLRs, CRNs are modular proteins and are defined by a highly conserved N-terminal ~50-amino-acid LFLAK domain and an adjacent diversified DWL domain. The effector genes locate mostly in the gene sparse regions of the genome that are rich in repetitive sequences and are rapidly evolving, probably enabling the evolutionary arms race between *P. infestans* and the host plant (Haas et al. 2009; Dong et al. 2015).

7.3 Population Structure of *P. Infestans*

It is imperative to understand the diversity of the pathogen to devise efficient management strategies. Knowledge on the pathogen population structure and its relation to phenotypic characteristics, such as fungicide sensitivity or aggressiveness, is important to develop effective management strategies for the disease (Saville et al. 2015). *Phytophthora infestans* is highly variable and has undergone a drastic change in structure over the period of time. Pathological specializations (races) within potato isolates were reported by Schick (1932) after almost 7 years of introduction of resistant hybrids/cultivars having R genes. However, universal appearance of races did not occur until resistance genes from *Solanum demissum* were transferred to commercial potato, *S. tuberosum*. Since then, the racial complexity has reached its zenith in different countries/regions (Guo et al. 2009; Li et al. 2009; Runno-Paurson et al. 2009; Arora et al. 2014). Up to 1984, only one mating type (A1) was known to occur throughout the world, except Mexico (Tooley et al. 1985). However, there had been worldwide migration as a result of which A2 mating type was introduced other parts of the world. First report of A2 mating type outside Mexico was from Switzerland (Hohl and Iselin 1984). Subsequently, A2 mating type was detected in USSR during the 1990s (Vorobev et al. 1991); the United States (Deahl et al. 1991); Belarus (Ivanyuk and Konstantinovich 1992); the Netherlands (Drenth et al. 1993a, b); India (Singh et al. 1994); Pakistan (Ahmed and Mirza 1995); Northern Ireland (Cooke et al. 1995);

Canada (Chycoski and Punja 1996); France (Gilet 1996); China (Zhiming et al. 1996); Hungary (Bakonyi and Ersek 1997); Italy (Cristinzio and Testa 1997); Ecuador (Oyarzun et al. 1997); Indonesia (Nishimura et al. 1999); Myanmar (Myint 2002); Colombia (Vargas et al. 2009); Sri Lanka (Kelaniyangoda 2011); Tunisia (Harbaoui et al. 2014); Scandinavia and Estonia in 1987 (Vorobyeva et al. 1991); Bolivia, Argentina, Uruguay, and Brazil (Plata 1998; Deahl et al. 2003; Forbes et al. 1998; Casa-Coila et al. 2017); and Algeria (Rekad et al. 2017). Though existence of both mating types has opened up the possibility of sexual reproduction, no evidence of frequent sexual reproduction has been found, suggesting that the sexual populations are ephemeral (Fry et al. 2015). Nevertheless, there are reports (e.g., the Nordic countries) which indicated the frequent occurrence of sexual reproduction in the field and survival of oospores that led to earlier onset of epidemics (Widmark et al. 2007; Schepers 2019). *P. infestans* is generally heterothallic requiring two different mating types for sexual reproduction. The presence of both mating types in central Mexico and in the Nordic countries of Europe and the Netherlands has led to sexual reproduction and high genetic diversity (Drenth et al. 1993a, b; Sjoholm et al. 2013; Wang et al. 2017). However, there are reports of occurrence of homothallic isolates which are self-fertile and constitute a new threat to potato and tomato crops because of their increased genotypic variability, better fitness, and greater aggressiveness (Zhu et al. 2016; Tian et al. 2016; Casa-Coila et al. 2017).

There are platforms. Viz., EuroBlight (http://euroblight.net/), USABlight (http://www.usablight.org/), Tizon Latino (https://tizonlatino.github.io/), AsiaBlight (https://www.asiablight.org), and AfricaBlight, which are carrying out monitoring of *P. infestans* populations across the globe. The findings have revealed that *P. infestans* populations are constantly evolving, and novel, usually more aggressive, genotypes appear periodically replacing the previously dominating genotypes (Schepers 2017). New genotypes can emerge through divergence from other genotypes, through recombination, or migration from other areas (Knaus et al. 2016). The main mode of reproduction of *P. infestans* is asexual, and variable numbers of clonal lineages exist in different countries and regions. Several studies have confirmed that appearance of new genotypes can often be attributed to migration (Fry et al. 2015; Knaus et al. 2016; Saville et al. 2016).

Multiple clonal lineages have been found in the United States since the 1990s, revealing the history of the displacement of lineages over time (Fry and Goodwin 1997; Hu et al. 2012a, b). Genetic analysis using simple sequence repeats (SSRs) of *P. infestans* from herbarium samples from the nineteenth century historic outbreaks revealed the presence of a single dominant clonal lineages FAM-1 that caused disease in both the continents, i.e., the United States and Europe (Saville et al. 2016), suggesting the migration of the pathogen from a similar point of origin (Yoshida et al. 2013). With the emergence of the US-1 lineage during the 1930s in the United States, the historic FAM-1 lineage subsequently declined (Saville et al. 2016). The dominance of US-1 clonal lineage lasted in the United States until the 1980s, when new lineages of the pathogen emerged that were insensitive to mefenoxam (Goodwin et al. 1996). The new genotypes were US-6 (A1 mating

type), US-7 (A2 mating type), US-8 (A2 mating type), and US-11, emerged out of Mexico, majority as a result of sexual recombination and some as clonal derivatives of earlier lineages (Goodwin et al. 1998). US-11, which is thought to be the progeny of US-6 and US-7 lineages (Gavino et al. 2000), still occurs in the fields of the Pacific Northwest and Florida.

Although US-1 was a dominant lineage in the United States for 60 years, it declined in the mid-1990s, probably because of its sensitivity to the fungicide mefenoxam. Majority of US lineages, with the exception of US-6, were detected in the 1990s in the United States, and many were resistant to mefenoxam (Saville and Ristaino 2019). The US-8 clonal lineage was responsible for the first pandemic during the 1990s in the United States (Fry and Goodwin 1997; Johnson et al. 1997). The second pandemic was in 2009 due to wide spread of US-22 clonal lineage with infected tomato seedlings throughout northeastern USA (Fry et al. 2013). The population of *P. infestans* in the United States continues to be dominated by relatively few clonal lineages (Hu et al. 2012a, b; Fry et al. 2013). The most recent dominant strains are US-8, US-11, US-22, US-23, and US-24 (Fry et al. 2015). Generally, lineages differ in terms of their response to mefenoxam, and pathogenicity and common lineages in the United States during 1990 to 2009 were largely resistant to mefenoxam (Fry et al. 2015), and growers were not using this molecule to manage late blight. However, the dominant lineage (US-22) in 2009 was sensitive to mefenoxam, and some dominant lineages since 2009 have also been sensitive to mefenoxam (Hu et al. 2012a, b; Saville et al. 2015). Further, lineages in the United States differ in terms of their pathogenicity. US-11 and US-23 are very good pathogens of both tomatoes and potatoes, whereas US-8 and US-24 are not good pathogens of tomatoes. The US-23 lineage has dominated the *P. infestans* population in the United States since 2012 by replacing the previously dominant lineages, including US-8 and US-22. The possible reasons for dominance could be its aggressiveness on both foliage and tubers (Danies et al. 2013) and its pathogenicity on both potatoes and tomatoes (Danies et al. 2013). Studies on genetic structure and sub-clonal variation of extant and recent US lineages revealed that many clonal lineages in the United States have come from Mexico via introduction, but US-23 (from Bolivia and Brazil) and US-1 (from Peru) lineages were introduced from other sources (Saville and Ristaino 2019). However, a survey for the presence of RXLR effector *PiAVR2* revealed the presence of lineages that carried either *PiAVR2*, its resistance-breaking variant *PiAVR2-like*, or both, suggesting lineages have experienced different levels of selection to the *R2* gene in potato, thereby indicating that populations of *P. infestans* in the US are the result of introductions from both South America and Mexico (Saville and Ristaino 2019).

The South and Central America can harbor divergent genotypes as these regions are rich in solanaceous species biodiversity and are centers of origin of the economically important crops that are potential alternative hosts of *P. infestans*. No sexual reproduction of *P. infestans* has been reported in South America; hence populations maintain strictly clonal structures, and A1 mating type is mostly dominant (Acuna et al. 2012; Cardenas et al. 2011). In Mexico, recombination is frequent and the population is extremely divergent (Wang et al. 2017), and it is also considered the

origin of the newly emerged genotypes in the United States (Goss et al. 2014; Saville et al. 2016).

EuroBlight is continuously investigating the evolution of potato late blight pathogen in the Europe. A complex population structure is observed in Europe with population dominance (70%) by a few widely disseminated clonal lineages. The clonal lineage 13_A2 was first detected in 2004 in the Netherlands and Germany which has now emerged in regions beyond Europe (Cooke et al. 2012). Some clones are widespread and have been present for more than a decade, but recently the frequency occurrence of three clones (EU_37_A2, EU_36_A2, and EU_41_A2) have increased from 10% (2016) to 40% (2019) by displacing the established clones (EU_13_A2, EU_6_A1, and EU_1_A1) from 60 to 40% of the population. Besides, 20–30% of the sampled European population is genetically diverse and consistent with local, ephemeral oospore-derived sexual populations. The frequency occurrence of the clonal lineage EU_13_A2 (blue-13) and EU_1_A1 has dropped to 9.3% and 0.4%, respectively, whereas the frequency of EU_6_A1 increased to 20.4% due to severe outbreaks in parts of Britain. A progressive displacement of these three lineages is occurring (Cooke et al. 2019). Clone EU_36_A2, which was first sampled at a low frequency in Germany and the Netherlands in 2014, has spread rapidly in Europe to the frequency of 26% in 2019. Clone EU_41_A2, first recorded in Denmark in 2013, has now spread to neighboring states, and its frequency has also increased from 4.6 to 5.7% of the European population in 2019 (Schepers 2019).

In eastern Africa, the first late blight epidemic occurred in Kenya in 1941 and the pathogen was thought to be introduced via potato seed tubers from the United Kingdom. After 1 year of the epidemic, the disease was also noticed in Uganda, Democratic Republic of Congo, and Tanzania (Natraas 1944). The US-1 was probably dominant in Europe at the time of the introduction of *P. infestans* in eastern Africa and is assumed to be the only lineage introduced into the region. In eastern Africa, only the A1 mating type has been detected so far, thereby signifying the persistence of a clonal population (Njorog et al. 2016). The US-1 had been the only lineage reported in the eastern African region, apart from RW-1 and RW-2 genotypes in Rwanda in the mid-1980s (Forbes et al. 1998; Goodwin et al. 1994). However, these two genotypes (RW-1 and RW-2) were not detected in a later study in 2007 that reported all isolates from Rwanda to be US-1 (Pule et al. 2013). They further reported that US-1 was still the only lineage in central and eastern Africa apart from Kenya, where US-1 and a new lineage KE-1 were found. The new genotype KE-1 was first reported from Kenya in 2007 and later from Uganda in 2011 and found to be the only lineage on potato in Kenya (Njoroge et al. 2016). The recent population of *P. infestans* infecting potato in the eastern African region is dominated by KE-1 lineage, which had similar SSR fingerprints to that of EU_2_A1 (Njoroge et al. 2019). They further found decline in US-1 lineage but still present on potato in Uganda, Rwanda, Burundi, and Tanzania. Besides, a tomato-adapted US-1 sub-population is also still present in all the countries. Two new European lineages (EU_33_A2 and EU_13_A2) have been emerged recently in Nigeria and Senegal on potato and are a cause for concern for potato production in sub-Saharan Africa (Schepers 2019).

Although there have been a number of publications on the late blight population structure of *P. infestans* in Asian countries, a very few have used markers for comparative analysis (Forbes 2015). The Indian population of *P. infestans* has been characterized for phenotypic and genotypic characters (Chimote et al. 2010; Sharma et al. 2016, 2017) and ploidy status (Sharma et al. 2018). The findings of these studies have shown that population is possessing complex virulence genes, resistance to metalaxyl, Ia mtDNA haplotype, and varied allele size for SSR markers. There are records of at least four migrations of *P. infestans* into India over the past 100 years. The oldest samples of *P. infestans* collected from Bagalpur (Bihar) in 1913 by J.F. Dastur were the Ia mtDNA haplotype (Ristaino and Hu 2009), and the US-1 clonal lineage (Ib mt DNA haplotype) was present in India by the 1960s (Ristaino and Hu 2009). The occurrence of the A2 mating type in the 1990s in the northern hills provided the additional evidence of migration from an outside source (Singh et al. 1994), and more recently, the European 13_A2 genotype was intercepted in southern India (Chowdappa et al. 2013, 2015). Dey et al. (2018) found that mutations have generated substantial sub-clonal variation in EU_13_A2 genotype, having 19 out of 24 unique variants not yet reported elsewhere globally. Nevertheless, the Asian population of *P. infestans* has also been genotyped using markers, and findings revealed the widespread occurrence of aggressive genotype 13_A2 in many parts of Asia as reported from China (Li et al. 2013), India (Chowdappa et al. 2013, 2015; Dey et al. 2018), Bangladesh (Kessel et al. 2017), and Pakistan (Raza et al. 2020). In Vietnam, the *P. infestans* population is still the "old" US-1 (Le et al. 2008). The *P. infestans* population in northwestern China is genetically distant from European lineages, including the recently identified 13_A2 lineage (Tian et al. 2016), though its presence (13_A2) was reported in Sichuan and Yunnan provinces, south western China (Li et al. 2013). Four clonal lineages, viz., KR_1_A1, KR_2_A2, SIB-1, and US-11, have been reported from South Korea. KR_2_A2 was confined to Gyeongnam Province, whereas SIB-1 was dominant until 2013 and thereafter its frequency declined gradually. US-11 was first found in 2014, and its frequency has increased to become co-dominant with KR_1_A1. The EU_13_A2 genotype was not found in South Korea (Choi et al. 2020). The Indonesian population is dominated by EU_2_A1 (60%), EU_4_A1, and EU_13_A2 (1.5%) (Dangi et al. 2021).

7.4 Symptoms

P. infestans adopts a two-step infection style typical of hemibiotrophs. Infection generally starts when sporangia lands on a plant surface and release zoospores that encyst, germinate, and penetrate the host tissue or sporangia directly germinate and initiate the infection. Germ tubes form an appressorium and then a penetration peg, which pierces the cuticle and penetrates an epidermal cell to form an infection vesicle. Branching hyphae with narrow, digit-like haustoria expand from the site of penetration to neighboring cells through the intercellular space. At this biotrophic stage, *P. infestans* requires living cells to obtain nutrients. However, this stage of

Fig. 7.1 Late blight symptoms (**a**) Foliar blight on upper surface (**b**) On lower surface (**c**) On stem (**d**) On tubers

infection remains unnoticed to the naked eye, but at cellular level a repertoire of molecular interactions takes place. The first visible symptoms appear within 2–3 days when the pathogen switches to the necrotrophic stage. Later on, the mycelium develops sporangiophores that emerge through the stomata to produce numerous asexual spores that initiate new infections (Judelson and Blanco 2005). In leaves, water-soaked irregular pale green lesions mostly near tip and margins that enlarge into brown to purplish black necrotic spots appear. A white mildew, which consists of sporangiophores and spores of the pathogen, can be seen on the lower surface of the infected leaves especially around the edges of the necrotic lesions under high humidity (Nowicki et al. 2012). On stems and petioles, light to dark brown lesions encircle the stems; as a result, the affected stems and petioles become weak at such points and may collapse. Affected tubers show irregular reddish brown to purplish areas which extend into internal tissues of the tubers (Fig. 7.1).

Pathogenesis involves the secretion of proteins and other molecules by *P. infestans* that participate in helping the pathogen attach to plant surface, breaking

down physical barriers to infection and influence the host physiology by suppressing or inducing host-defense responses (Huitema et al. 2004). Gene expression profiling during asexual development of *P. infestans* revealed highly dynamic transcriptome. Differentially expressed genes encode potential cellular regulators, especially protein kinases; metabolic enzymes involved in glycolysis, gluconeogenesis, or the biosynthesis of amino acids or lipids; regulators of DNA synthesis; structural proteins; and pathogenicity factors like cell-wall degrading enzymes, RXLR effectors and enzymes protecting against plant defense responses (Tani et al. 2004; Judelson et al. 2008). A MADS-box protein (*PiMADS*) is required for sporulation of *P. infestans* but not for hyphal growth or host colonization as both mRNA and protein levels decline upon spore germination (Leesutthiphonchal and Judelson 2018). *P. infestans* possesses a large repertoire of phospholipase D (PLD) proteins which are essentially required for the promotion of virulence, possibly by executing membrane modifications to support the growth of *P. infestans* in the host (Meijer et al. 2019). Identification of these factors involved in pathogen growth and development and in pathogenesis would be of help in designing management strategies.

7.5 Management of the Disease

Management of this devastating pathogen is challenged by its remarkable speed of adaptation, with respect to emergence of virulence towards resistant cultivars and to fungicide resistance (Haas et al. 2009). One of the prerequisites for durable management of late blight is up-to-date knowledge on characteristics of local *P. infestans* population and its dynamics. Since the pathogen population is continually evolving, the emerging clonal lineages with new traits highlights the need to tailor management to the local pathogen population. No single approach is effective; hence, combination of approaches in an integrated manner is essential to combat this devastating disease.

7.5.1 Cultural Practices

These are an important part of an integrated disease management program as they reduce the incidence and severity of the disease epidemic thereby reducing yield losses and lowering the requirements of fungicides (Mizubuti and Forbes 2002). Reduction of primary source of inoculum is the first step, and this can be achieved by eliminating volunteers and cull piles, waste heaps, infected tubers, use of certified seed and resistant varieties, balanced fertilization, adequate space between rows and plants, rotation with non-host crops, adequate hilling, harvest in dry conditions, and when the tubers are mature (Garrett and Dendy 2001; Perez and Forbes 2010). Onset of epidemic can be delayed by 3–6 weeks if all primary infection from early potato is eliminated (Forrer et al. 2000). Covering of dumps with black plastic sheet throughout the season is an important step in reducing the primary inoculum as it prevents re-growth and the proliferation of spores on the piles thereby reducing the risk to

nearby crops (Cooke et al. 2012). Infection usually starts early in fields which are not subjected to crop rotations. A sound crop rotation for 3–4 years is an effective way of reducing the risk of soil-borne inoculum as oospores can remain infectious up to 48 months in soil (Turkensteen et al. 2000; Bodker et al. 2006; Hannukkala et al. 2007). Choice of suitable cultivars, well-aerated fields, pre-sprouting of tubers, early planting, use of resistant varieties, and mixtures of potato varieties (resistant and susceptible) are some of the measures against foliar blight (Meinck and Kolbe 1999; Garrett and Mundt 2000; Pilet et al. 2006). Strip cropping of potatoes significantly reduced late blight severity in organic production when planted perpendicular to the wind neighbored by grass clover (Bounes and Finckh 2008). Avoiding excess nitrogen and use of moderate nitrogen fertilization is often recommended as a cultural practice to delay the development of late blight, whereas higher dose of phosphorus and potassium has been found to give a higher yield in a late blight year (Roy et al. 2001). High ridging is often used to reduce tuber contamination by blight. Another approach to reduce tuber blight is to destroy the canopy when blight reaches to 75% severity. Elimination of infected foliage reduces the likelihood of tuber infection. Intercropping with garlic has been found effective against potato late blight under Ethiopian condition (Kassa and Sommartya 2006).

7.5.2 Host Resistance

Host resistance is the most preferred environment and economic option globally for the management of late blight. With the use of host resistance, fungicide load can be reduced either by lowering the fungicide dose or increasing the application intervals (Kirk et al. 2005; Cooke et al. 2012; Haverkort et al. 2016). Durable resistant cultivars with multiple resistant genes are needed today, which can be developed by a blend of conventional and molecular approaches. So far resistant genes from the wild species *Solanum demissum* and *S. stoloniferum* and the cultivated *S. tuberosum* subsp. *andigena* and *S. phureja* have been utilized into common potato in different parts of the world (Bradshaw et al. 2006). Thus, it warrants the breeders to search for new sources of resistance in wild gene pools and their faster deployment into cultivars through modern techniques. Late blight resistance genes/QTLs and molecular markers for late blight resistance genes/QTLs in potato have been reviewed by Tiwari et al. (2013). Genetic engineering may also provide options for generating resistant cultivars. A resistance gene effective against most known strains of blight has been identified from a wild relative of the potato, *Solanum bulbocastanum*, and introduced by genetic engineering into cultivated varieties of potato (Song et al. 2003; Van der Vossen et al. 2003). Introgression of RB gene in Indian popular potato cultivars has demonstrated variable level of late blight resistance and generation of valuable genetic material for resistance breeding (Shandil et al. 2017).

7.5.3 Use of Fungicides

The chemical-based management still continues to be the most common method to supplement host resistance and to manage the late blight. Recent changes in the population structure of the pathogen have led to the advent of new genotypes that are more aggressive and resistant to previously effective fungicides (Fry et al. 2015). Sixteen classes of fungicides with different modes of action are available for the control of oomycete plant pathogens (FRAC n.d.). The three most important single-site compounds are phenylamides, quinone outside inhibitors, and carboxylic acid amides (Gisi and Sierotzki 2014). Products containing mefenoxam or metalaxyl (a.i. mefenoxam) have been the most widely used fungicides for control of *P. infestans.* However, more recent dominant lineages are largely sensitive to mefenoxam (Matson et al. 2015; Saville et al. 2015). The build-up of resistance to single-site oomycides has accelerated the research for anti-oomycete compounds with new modes of action. The development strategy for creating new fungicides consists of fungicides that are (1) effective at an extremely low dosages, (2) readily degradable and less residual in the environment, and (3) selective toxic agrochemicals (Umetsu and Shirai 2020). Many fungicides possessing various novel modes of action have been launched or are under development. Two such novel compounds are ametoctradin (Quinone QoSI inhibition of the respiratory chain) binding to the mitochondrial bc1 Complex III (Fehr et al. 2015) and oxathiapiprolin (inhibitor of oxysterol-binding protein) (Sweigard et al. 2014). Oxathiapiprolin binds in the oxysterol-binding protein (OSBP) domain of oomycetes and inhibits zoospore and sporangial germination, stops mycelia growth in the host plants before visible symptoms occur, and inhibits further lesion growth and spore production and viability. It belongs to the FRAC U49 group of fungicides (Cohen 2015).

7.5.4 Alternatives to Fungicides

Various chemicals other than fungicides have also been found effective against late blight; for example, ammonium molybdate, cupric sulfate, and potassium metabisulfite have been reported to partially inhibit the growth and spore germination of *P. infestans*, whereas ferric chloride, ferrous ammonium sulfate, and $ZnSO_4$ completely inhibited growth and spore germination (Bhat et al. 2006). The foliar application of $ZnSO_4$ and $CuSO_4$ (0.2%) micronutrients in combination with host resistance delayed the onset of late blight by 12 days and subsequently reduced disease severity with higher yield (Basu et al. 2003). Phosphites (Phi), derived from phosphorous acid, are fungitoxic chemicals that can be combined with different elements such as calcium, copper, manganese, magnesium, potassium, or zinc and are classified by the US Environmental Protection Agency (US-EPA) as biopesticides, specifically biochemical pesticides (http://www.epa.gov/pesticides/biopesticides/). Thus, they have low environmental impact (Guest and Grant 1991). Besides their fungistatic or fungicidal activity (Fenn and Coffey 1984; Lobato et al.

2008), Phi stimulate defense mechanisms in plants against diseases (Daniel and Guest 2006; Andreu et al. 2006; Lobato et al. 2011) and promote growth (Thao and Yamakawa 2009). Because of these attributes, the horticultural industry widely uses Phi for oomycete control (Pilbeam 2003). Cicore et al. (2012) evaluated the effect of calcium phosphite (Phica) and potassium phosphite (PhiK) on late blight control and found that PhiK had significantly lower damage and higher yields than PhiCa and untreated control. Sub-phytotoxic dose of boron with reduced rate of propineb + iprovalidicab has been found more effective than treated with fungicides alone (Frenkel et al. 2010). Similarly, application of potassium phosphate in combination with reduced doses of fungicides provided the same level of protection as full dose of fungicides. Thus, combined treatments could help to reduce the quantity of traditional fungicides and may also decrease the selection pressure for fungicide resistance development in the pathogen. β-aminobutyric acid (BABA) has been known as an inducer of disease resistance. Plant activators, viz., BABA and phosphoric acid, have been evaluated against late blight with combination of fungicides or alone (Tsai et al. 2009). A 20–25% reduction of the fungicide dose in combination with BABA gave the same result on late blight development as full dose of Shirlan alone in field condition, while reduced dose of Shirlan alone sometimes resulted in less effective protection. The partially resistant cultivars Ovatio and Superb reacted to lower concentrations of BABA where no effect was found in susceptible cv. Bintje (Liljeroth et al. 2010). The expression of the defense-related genes and *P. infestans* effector proteins β-1,3 glucanase, PR-1 protein, phytophthora inhibitor, protease inhibitor, xyloglucanase, thaumatin protein, steroid binding proteins, proline, endochitinase, and cyclophilin genes was upregulated with the SAR activator treatment compared to unsprayed (CPRI 2014). Better results than with copper were achieved with Phosfik® (Ph), a phosphonate-based product. Two to three applications with 2–3 L/ha of Ph would be feasible to not exceed a minimal risk level (MLR) of 20 mg/kg of phosphorous acid as proposed by the European Food Safety Authority (Forrer et al. 2017). Due to an excellent environmental profile and a complex mode of action counteracting *Phytophthora infestans* resistance, phosphonate-based products would be most suitable for sustainable late blight management in integrated disease management programs.

7.5.5 Biocontrol

New strategies to manage plant diseases without harming the environment are urgently needed. Biocontrol agents and bio-pesticides could be a safe option to the use of synthetic fungicides. Some workers have reported the use of *Trichoderma* isolates (Yao et al. 2016), *Chaetomium globosum* (Shanthiyaa et al. 2013), *Trichoderma viride*, and *Penicillium viridicatum* (Gupta 2016) and species of *Bacillus*, *Pseudomonas*, *Rahnella*, and *Serratia* (Daayf et al. 2003) as biocontrol agents in the management of late blight disease in potato. The bio-based products, viz., neem-based products and bio-agents (*T. viride* and *P. fluorescens*), have shown some efficacy against late blight under field conditions (Lal et al. 2021). The

biocontrol agents in general have been found to be very effective under laboratory and glasshouse conditions but less effective under field conditions (Arora 2000). However, an integrated use of biocontrol agents along with fungicides could help to reduce the quantity of fungicides used in the management of late blight (Lal et al. 2017). Biosurfactants produced by microbes can be used as alternatives to chemical surfactants because of their low toxicity, high specificity, and biodegradability (Lima et al. 2011). Significant reduction in late blight development was observed when plants were treated with biosurfactant—*Pseudomonas koreensis* 2.74—and also, biosurfactants have the potential to induce resistance in potato to late blight (Bengtsson et al. 2015). The biosurfactant produced by *Pseudomonas aeruginosa* has shown high efficacy against *P. infestans* under in vitro and glass house conditions (Tomar et al. 2013, 2014). The rhamnolipid-based formulation prepared from *P. aeruginosa* biosurfactant was found effective against late blight when evaluated through detached leaf (Tomar et al. 2019) and could be used in field spray as green chemicals.

Plant-associated bacteria contribute to their host's health in diverse ways, among which the emission of disease inhibiting volatile organic compounds (VOCs) is one option. Volatile organic compounds (VOCs) produced by the plant microbiota have been demonstrated to elicit plant defenses and inhibit the growth and development of numerous plant pathogens. The inhibitory impact of volatiles emitted by *Pseudomonas* species against late blight has been shown by impeding mycelial growth and sporangia germination of *P. infestans* (Bailly and Weisskopf 2017). The VOCs containing sulfur compound S-methyl methane thiosulfonate (MMTS) had shown high *in planta* protective potential against late blight without phytotoxic effects. Short exposure times were sufficient to protect plants against infection. This protective activity of MMTS is not mediated by the plant immune system but is due to its anti-oomycete activity (Chinchilla et al. 2019). This provides new perspectives for plant protection by opening new research avenues on the role of VOCs in the interaction between plants and their microbiome and thus could help select for efficient biocontrol strategies and lead to a greener chemical disease management in the field.

In organic potato production, the only synthetic direct control measure allowed is the use of copper-based products despite its persistence in soil and toxicity to soil organisms (Buenemann et al. 2006). Based on such reports about the toxicity of copper, the EU proposed a ban of copper fungicides as early as 2002, though it was not imposed as of now, but this would have threatened the feasibility of organic potato production. This initiative led to intensified research for new approaches to reduce the risk of late blight attacks and for natural products to replace or reduce the use of copper (Leifert and Wilcockson 2005). Three promising botanicals, including bark of buckthorn (*Frangula alnus*, FA), roots of medicinal rhubarb (*Rheum palmatum*), and galls of the nutgall tree (*Galla chinensis*), have been reported effective under field conditions and could replace copper reaching a level close to that of 2–3 kg copper per hectare and year (Forrer et al. 2017).

7.5.6 Late Blight Forecasting

Currently, late blight management has been heavily based on numerous fungicide applications due to introduction of new, more aggressive genotypes of the pathogen (Schepers 2017, 2019). However, this strategy faces increasing concerns due to societal pressure for reducing pesticide use on crops and acreage of organically grown food crops. Innovative and effective control measures are needed if fungicide use is to be reduced or, as in the case of organic production, eliminated. One way of achieving this goal is through the use of forecasting models and decision support systems (DSSs). Forecasting allows a better control of a disease and a more efficient use of fungicides by making informed disease management decisions. Various late blight forecasting models and DSSs have been developed across the globe for the management of late blight in different agro-ecologies (Table 7.1). The DSS-based strategy can deliver general or site-specific information to the stakeholders via print and electronic media (Cooke et al. 2012) enabling them to take firm decisions on the management of late blight thereby resulting in economic gains and environment protection (Sekhon et al. 2017; Liu et al. 2017; Sharma 2019).

Table 7.1 List of forecasting models and decision support systems used for forecasting and management of late blight (Source: Singh and Sharma 2013; Schepers 2019)

Decision support system	Country	Decision support system	Country
BliteCast, SimCast, BlightPro	USA	Guentz-Divoux	Belgium, France
Blight-watch, plant plus, BlightCAST	England, Wales, Scotland	Estonian crop research institute	Estonia
Plant plus	Latvia	Mileos	France
Phytophthora model Weihenstephan, ISIP, Phytoprog, SimPhyt, ProPlant, ProGeb	Germany	Prophy, plant plus, Akkerwels (WUR model)	Netherlands
Met. Service based on Irish rules (Bourke)	Ireland	VIPS (Naerstad model)	Norway
Plant-plus, VNIIF blight, Agrodozor	Russia	Plant plus, blight management (DK), VIPS (no)	Sweden
Bio-PhytoPRE, PhyoPRE, PhytoPRE +2000	Switzerland	WISDOM, web-blight, NegFry	Denmark
IPI, MIP	Italy	Blight watch	UK
China-blight	China	Indo-Blightcast, Jhulsacast	India

7.6 Looking Forward

Phytophthora infestans is capable of overcoming host resistance and fungicides; hence, late blight would continue to be the main constraint in potato cultivation throughout the world. Nevertheless, the advances in molecular, sensor, computational, and electronics technologies would provide stable solutions for its management. New high-throughput methods (remote sensing, image processing, UAV, etc.) would be of significance in disease detection and surveillance. Robust, quick, and onsite detection methods are needed for early diagnosis of the pathogen and monitoring of population structure. Research is warranted on development of new oomycides having efficacy at very low dosages, highly degradable, and with novel mode of action. Besides, there is need to identify new molecules of biological origin that can be used under organic production. Smartphone-based systems can be of help in monitoring, forecasting/DSSs, and dissemination of the disease information to the stakeholders. Emerging research topics on *P. infestans* include genome editing for genetic improvement of plant disease resistance and the role of the pathogen–microbiota interaction in promotion or suppression of the disease.

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