Chapter 9
Fungal, Oomycete, and Plasmodiophorid Diseases of Potato

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Abstract This chapter discusses the major potato diseases worldwide: late blight, early blight, wart, and powdery scab. **Late blight**, caused by the oomycete *Phytophthora infestans*, continues to be the main biotic constraint of potato production. Annual losses have been estimated to be about €6.1 billion, with major consequences to food security, especially in developing countries. Symptoms of the disease can be seen in leaves (water-soaked light to dark brown spots), stems (brown spots), and tubers (slightly depressed areas with reddish-brown color). High humidity and mild temperatures are essential for disease development and, under optimal conditions, the disease can destroy a field in a few days. *Phytophthora infestans* evolves continuously, mainly through recombination and migration from other

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areas. Thus, monitoring of *P. infestans* populations is critical for the design of effective management strategies. Fungicides remain as the most common tactic for late blight management, but environmental considerations are increasing the pressure to use host resistance, sanitation, and other measures. New solutions being developed to manage late blight include, among others, smart phone-based decision support systems linked to portable molecular diagnostics kits that can disseminate disease information rapidly to a large number of farmers. Emerging research topics on *P. infestans* include the role of the pathogen–microbiota interaction in promotion or suppression of the disease, as well as the metabolism of *P. infestans*.

The fungus *Alternaria solani* is the main pathogen causing **early blight** on potatoes. Early blight can be found in most potato-growing countries. Typical symptoms on the leaves are dark brown to black spots with concentric rings (target spot). In susceptible potato cultivars in particular, as well as in locations (especially in warmer areas) with increased occurrence of *A. solani*, the disease can cause considerable yield losses. Integrated pest management to control early blight requires the implementation of several approaches. The disease is primarily controlled by the use of cultural practices (to reduce the soil born inoculum), less susceptible cultivars and the use of pesticides. But there is a loss in sensitivity toward two groups of fungicides described. The loss in sensitivity towards succinate dehydrogenase inhibitor (SDHI) and Quinone outside inhibitor (QoI) fungicides are caused by different point mutations. In many countries the occurrence of SDHI and QoI mutants is reported. Therefore, the control of early blight will be a considerable challenge in the future. The increasing importance of early blight in potatoes is due to a number of factors.

Synchytrium endobioticum is a soil-borne biotrophic fungus causing **potato wart** disease of cultivated potato. The fungus originates from the Andean zones of South America, from where it spread first to Europe in the late of the nineteenth century. Presently, the geographical distribution of this pathogen includes almost all European and Mediterranean Plant Protection Organization (EPPO) countries, Asia, North and South America as well as Oceania (New Zealand). The typical symptoms of cauliflower-like galls could develop on all meristematic tissues of potato except roots. *S. endobioticum* produces summer sporangia with mobile zoospores that can move in the soil. Winter (resting) sporangia are the dormant structures by which the fungus disperses to establish new infections. They can survive more than 40 years without plant hosts. The pathogen does not produce hyphae. Its long persistence in soil and the severe losses it inflicts to potato crops have prompted its inclusion into the A2 quarantine list of EPPO. Since the discovery of pathotype 2(G1) in Germany, more than 40 pathotypes were reported in Europe. In Europe, pathotypes 1(D1), 2(G1), 6(O1), and 18(T1) are the most relevant. Other pathotypes occur mainly in the rainy mountainous areas of central and eastern Europe. *S. endobioticum* is a still serious problem for crop production in countries with moderate climates. The strategies to confine the disease are strict quarantine and phytosanitary measures, and the cultivation of resistance cultivars of potato.

Spongospora subterranea causes root galling and tuber **powdery scab** leading to quality and yield losses in seed and ware crops worldwide and is also important as the natural vector of potato mop-top virus (PMTV), an economically important tuber blemish disease of potato. *S. subterranea* spreads by movement of infected

seed tubers and soil and can survive long periods in soils and some asymptomatic hosts. Powdery scab is particularly favored by cool, damp conditions and is an intractable disease. Avoidance is the best control for powdery scab, but once soil is infested with *S. subterranea*, cultural practices and chemical treatments are ineffective control methods, and host resistance appears to be the most promising mechanism for long-term management. Although sources of host resistance have been identified, they are not widely deployed in practice. *S. subterranea* is an unculturable biotroph, making research difficult. Recent progress in understanding the biology of *S. subterranea* as a result of the application of basic molecular techniques, and future opportunities to further advance knowledge of this understudied pathogen, and the virus that it vectors, are included.

9.1 Late Blight

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9.1.1 Causal Organism

Late blight is caused by *Phytophthora infestans* Mont de Bary. It was previously classified as a fungus due to the superficial resemblance to filamentous fungi but is now classified as oomycete in the kingdom of stramenopiles (Kamoun et al. 2014). The vegetative stage of the mycelium in *P. infestans* is diploid, while in true fungi it is haploid. However, recent research has shown that in the modern-day lineages the progenies from sexual *P. infestans* populations are diploid, but the most important pandemic clonal lineages are triploid (Li et al. 2017). Virulence of oomycetes depends on large, 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). Elicitins are an example of structurally conserved extracellular proteins of *P. infestans* that have a function in the sequestration of sterols from the host plant, but can also act as pathogen-associated molecular patterns (PAMPs), and as such can activate PAMP triggered immunity (PTI) (Du et al. 2015). *P. infestans* secretes large numbers of effectors: apoplastic effectors, such as EPIC1, interact with the host cell wall, host proteases and other defense-related molecules in the host extracellular space, while cytoplasmic effectors, the RxLR proteins, and CRNs (crinkling and necrosis-induced proteins) function inside the plant cells (reviewed in Whisson et al. 2016). RxLR effectors act as activators of plant immunity, resulting in effector triggered immunity (ETI) (Oh et al. 2009; Wang et al. 2017a), while the apoplastic effectors, similarly to elicitins, act as activators of the PTI (Domazakis et al. 2017). Recent research has also shown that some *P. infestans* effectors can target host proteins

whose activity enhances susceptibility possibly through the inhibition of positive regulators of immunity or promote the activity of susceptibility (S), that in turn can negatively regulate immunity (Boevink et al. 2016). 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).

9.1.2 Symptoms

The asexual, aerially dispersed sporangia (Fig. 9.1) are responsible for most of the devastating epidemics on potato. When the sporangia lands on a plant surface it can germinate directly or first form zoospores, which encyst, germinate, and penetrate the host tissue (reviewed by Fry et al. 2015). This stage of infection is unnoticeable to the naked eye, but inside the plant cell a repertoire of molecular interactions takes place. After penetration and adhesion, the pathogen forms haustoria inside the plant cells, from where it secretes effector proteins (reviewed in Nowicki et al. 2012; Whisson et al. 2016; Wang et al. 2017a). At this biotrophic stage *P. infestans* requires living cells to obtain nutrients.

The first visible symptoms appear within 2–3 days when the pathogen switches to the necrotrophic stage. In leaves, lesions are light to dark brown in color, water-soaked, irregularly shaped, sometimes surrounded by a yellow halo and not limited by leaf veins. Symptoms typically begin to develop where water accumulates near the leaf edges or tips (Fig. 9.2) and in stems (Fig. 9.3) near petioles. Affected tubers show irregular, slightly depressed areas with brown color. In a cross-section, finger-like extensions can be seen from the external surface to the tuber medulla (Fig. 9.4) (Perez and Forbes 2010).

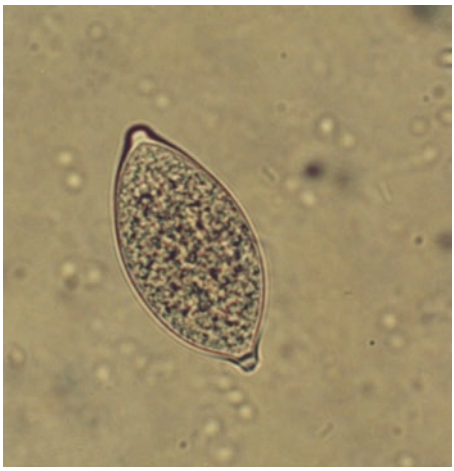

Fig. 9.1 Lemon-shaped sporangium of *Phytophthora infestans*

Fig. 9.2 Symptoms of late blight on leaves

Fig. 9.3 Symptoms of late blight on stems

Fig. 9.4 External and internal symptoms of late blight in tubers

The dying, necrotized cells serve as nutrient for the pathogen and under high humidity, a white mildew growth is formed on the underside of the leaves, which represents the sporangiophores and sporangia that emerge through the stomata (Nowicki et al. 2012). During spore formation and germination, large numbers of genes involved in pathogenesis, calcium signaling, and metabolism are upregulated or transcribed in waves (Ah-Fong et al. 2017), whereas genes of the fatty acid biosynthesis pathway are downregulated (Rodenburg et al. 2018). Identification of the proteins and enzymes essential for pathogen growth and development, and linked to the different symptomatic reactions in the plant, can lead to the discovery of potential targets for crop protection chemicals.

High level of moisture is essential for the lesion development, and under optimal conditions the disease can advance very fast and destroy the plant in matter of days (Perez and Forbes 2010).

9.1.3 Impact

The potential economic and social impact of potato late blight is best illustrated by the well-publicized role it played in the Irish Famine in the middle of the nineteenth century. Because of the famine, millions of Irish died or emigrated (Bourke 1993). Other devastating late blight outbreaks have been reported around the world, causing food insecurity, hunger (International Potato Center 2007), and oftentimes crippling the local potato industry. Haverkort et al. (2009) estimated that the global costs and losses due to late blight may take 16% of all global potato production. At 100 €/t the world potato production represents a value of €38 billion today. The 16% loss then represents an annual financial loss of €6.1 billion per annum today, considering that the increase in global potato production in the last decade has mainly been in developing countries, which suffer low yields and the vast majority of these estimated losses to late blight compared to developed countries.

9.1.4 Resistance to Late Blight

After the discovery of the Mexican wild species *Solanum demissum* as an excellent source of resistance, eleven major genes were introduced in cultivated tetraploid potato breeding lines (Black et al. 1953; Malcolmson and Black 1966). Although some of these genes can be considered defeated, others, for example R8, are still effective against current pathogen populations (Vossen et al. 2016). Over 50 R genes have been identified from wild Solanum species as detailed by Rodewald and Trognitz (2013), and the research field remains active with a growing list of genes available for potato breeding programs (Jo et al. 2015; Vossen et al. 2016; Witek et al. 2016; Yang et al. 2017). However, due to crossing barriers and linkage drag, there are only few successful cases where R genes have been introduced into

improved tetraploid breeding lines by classical breeding (Bethke et al. 2017). Introduction of a single R gene from wild germplasm is a lengthy procedure as demonstrated by the examples of commercial varieties Bionica and Toluca that contain Rpi-blb2 originating from *S. bulbocastanum*, and were released almost 50 years after the first crosses were made (Haverkort et al. 2016). Genetic engineering bears promise and varieties containing stacked or single R genes are in the process of being released in the markets that accept this technology (Haverkort et al. 2016; Schiek et al. 2016; Pacifico and Paris 2016; Ghislain et al. 2018). As opposed to the major R genes, quantitative resistance was generally expected to be governed by many minor genes. However, recently it was shown that R genes can also have quantitative effects. The potato cultivar Sarpo Mira contains at least four R genes that confer complete resistance against incompatible isolates and a quantitative R gene, Rpi-Smira1 that confers broad-spectrum field resistance (Rietman et al. 2012). A biparental cross using a haploidized resistant clone from the CIP B3 population was used to locate a strong QTL in chromosome 9 (Li et al. 2012a). Subsequent association mapping confirmed the importance of the same genome region for late blight resistance in the tetraploid B3 breeding population (Lindqvist-Kreuze et al. 2014), and recently the R8 gene was identified in the QTL by dRenseq (Jiang et al. 2018).

Identification of new resistance sources and functional resistance or susceptibility genes has been recently greatly accelerated by modern techniques, such as effectoromics and resistance gene enrichment sequencing technologies. To date, all effector proteins identified that are recognized by the plant resistance (R) proteins belong to the RXLR category. Therefore, RXLR effectors cloned into expression vectors have been used to successfully identify functional new R genes from potato germplasm using agroinfection (Vleeshouwers et al. 2011; Vleeshouwers and Oliver 2014). Apoplastic effectors are recognized by pathogen recognition receptors (PRR) and can be used in a similar manner to identify resistance germplasm (Domazakis et al. 2017). In the resistant germplasm, the NB-LRR (nucleotide binding-site leucine-rich repeat) resistance genes can be rapidly identified and cloned using gene-targeted, resistance gene enrichment and sequencing method (Jupe et al. 2013; Witek et al. 2016).

Durability of quantitative resistance will, however, continue to depend on the size of the cultivation area of a variety as well as the dynamics of the pathogen population.

9.1.5 Phytophthora infestans *Populations*

Knowledge on the local pathogen population structure is important for the design of impactful disease management actions (Fry et al. 2015). In recent years, the initiatives EuroBlight (http://euroblight.net/), USABlight (http://www.usablight.org/), and TizonLatino (https://tizonlatino.github.io/) have been carrying out is monitor-

ing of *P. infestans* populations. This work has confirmed that *P. infestans* populations are constantly evolving and novel usually more aggressive genotypes appear periodically replacing the previously dominating genotypes. 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). Until recently the mating type A1 was dominating worldwide, except in the presumed center of origin, Mexico, where both mating types were found in similar frequencies (Goodwin et al. 1992). This situation has changed dramatically, and A2 has now been reported in Scandinavia and Estonia (Hermansen et al. 2000; Runno-Paurson et al. 2016; Montes et al. 2016), Central Europe (Flier et al. 2007; Li et al. 2012b; Mariette et al. 2016), China (Zhu et al. 2015), Bolivia, Argentina, Uruguay and Brazil (Plata 1998; Deahl et al. 2003; Forbes et al. 1998; Casa-Coila et al. 2017), the USA (Rojas and Kirk 2016), Tunisia (Harbaoui et al. 2014), Algeria (Rekad et al. 2017), India (Chowdappa et al. 2015), and Canada (Danies et al. 2014). However, even though both mating types are present in most cases, no evidence of frequent sexual reproduction has been found, suggesting that the sexual populations are ephemeral (Fry et al. 2015). There are notable exceptions however, such as the Nordic countries, where it was shown that the sexual reproduction in the field is frequent and the oospores surviving in the field over winter in plant debris has led to earlier onset of epidemics (e.g., Widmark et al. 2007). The diversity of *P. infestans* in South and Central America is a particularly interesting question, because these regions are extremely rich in biodiversity of Solanaceous species that are potential alternative hosts of this pathogen and thus can harbor divergent genotypes. Furthermore, the centers of origin of the economically most important hosts, potato and tomato, are there. Interestingly, in South America, no sexual reproduction of *P. infestans* has been reported, and populations maintain strictly clonal structures. In Colombia, Chile, Ecuador, and Peru the A1 mating type has been found mostly (Acuna et al. 2012; Perez et al. 2001; Forbes et al. 1997; Cardenas et al. 2011). In Mexico, in contrast, recombination is frequent and the population is extremely divergent with subdivisions associated with geographic regions (Wang et al. 2017b). Mexico was also shown to be the origin of the current genotypes found in South America and continues to play an important role as the source population of the newly emerged aggressive genotypes in the USA (Saville et al. 2016; Goss et al. 2014). Although *P. infestans* is generally heterothallic requiring two different mating types to form sexual oospores (Fig. 9.5), some isolates are homothallic. Recent studies have shown that these self-fertile isolates are found more frequently, constituting a new threat to potato and tomato crops because of their increased genotypic variability, better fitness, and greater aggressiveness (Zhu et al. 2016; Casa-Coila et al. 2017).

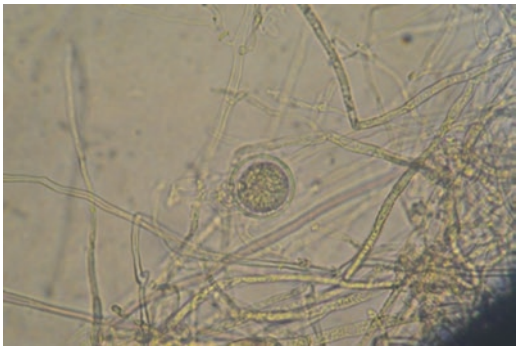

Fig. 9.5 Oospore of *Phytophthora infestans*

9.1.6 Management

Late blight of potatoes can be suppressed by a combination of approaches. As a polycyclic disease that explodes under favorable conditions, integrated strategies are crucial. These include sanitation measures that eliminate or reduce initial sources of the disease (e.g. infected seed, cull piles, infected neighboring plots, and volunteers), prophylactic fungicide sprays before the appearance of symptoms, curative fungicide sprays and use of resistant cultivars to reduce the rate of disease development, use of early-maturing cultivars to reduce the duration of the epidemic, or planting the crop in seasons or areas where the environment is not favorable for the pathogen.

The application of chemical fungicides continues to be the most common strategy for late blight control, making late blight one of the top drivers for pesticide use in the world. The demand for weekly applications generates a billion-dollar business globally every year (Haverkort et al. 2009). To optimize the use of fungicides, it is important to know the efficacy and type of activity of the active ingredients. The frequency and timing of fungicide applications may depend on the foliar resistance of the cultivar, fungicide characteristics, rate of growth of new foliage, weather conditions, irrigation, and incidence of blight in the region (Cooke et al. 2011). The range of fungicide types available to farmers vary depending on the numbers of products registered and commercialized in their area. In Europe the number of registered products is being reduced due to health and environmental concerns and in some European countries farmers have access to less than ten fungicide products for late blight control (http://www.endure-network.eu). In other countries the process of registration of pesticides is less restricted. For example, in Ecuador, hundreds of fungicide products are registered for late blight control based on more than 30 active ingredients of which most contain old generic substances like mancozeb, cymoxanil, and carbendazim (http://www.agrocalidad.gob.ec), yet in other countries only a couple or so products are readily available to farmers due to trade limitations.

The most efficient and arguably the most elegant strategy to control late blight is the use of host resistance. Today, it is well known that with the use of genetic resistance late blight can be controlled with less fungicide either by lowering the fungicide

dose or using longer application intervals (Kirk et al. 2005; Nærstad et al. 2007; Cooke et al. 2011; Liljeroth et al. 2016; Haverkort et al. 2016). The use of resistant varieties could sharply reduce losses from late blight, especially in developing countries, where disease is less well managed for many reasons (e.g. high disease pressure, problems of access to fungicides, and inadequate farmer knowledge of disease dynamics). Nevertheless, the use of resistant varieties continues to be an uncommon disease management approach as susceptible varieties are promoted and required by many wholesalers and processing industries, leaving farmers with little option but to grow susceptible varieties (Forbes 2012). Integrating genetic resistance and chemical control helps in reducing the use of fungicides, decreases production costs, and reduces damage to human health and the environment (Perez and Forbes 2010; Cooke et al. 2011). One way of achieving better design and integration of management elements is through the use of simulation models (reviewed by Forbes et al. 2008) and especially decision support systems (DSS). These typically integrate and organize all available information on the life cycle of *P. infestans*, monitoring of inoculum, weather (historical and forecast), plant growth, fungicide characteristics, cultivar resistance, and thereby predict disease pressure and action thresholds that can guide decision-making. Based on the information provided by the DSS farmers can make informed disease management decisions. DSS can deliver general or very site-specific information to the users via extension officers, telephone, SMS, e-mail, and websites (Cooke et al. 2011). In the case of smallholders in developing countries, basic information to understand the disease is critical to improve management (Nelson et al. 2001; Andrade-Piedra et al. 2009; Ortiz et al. 2019).

Cultural control involves all the activities carried out during agronomic management which alter the microclimate, host condition, and pathogen behavior in such a way that they avoid or reduce pathogen activity (survival, dispersal, and reproduction) (Garrett and Dendy 2001). Among them are the elimination of volunteers and cull piles and associated debris, use of clean seed potatoes preferably certified seed, use of resistant varieties, adequate space between rows and plants, rotation with other crops not susceptible to late blight, adequate hilling, harvest in dry conditions and when the tubes are mature (Garrett and Dendy 2001; Perez and Forbes 2010). Mixtures of potato varieties (resistant and susceptible) offer partial improvement on disease suppression (Phillips et al. 2005; Pilet et al. 2006). Under temperate climate conditions and where tuber infections are a concern, potato vines are typically killed by applying chemical desiccants 2–3 weeks before harvest (Perez and Forbes 2010). Biological control consists of reducing disease through the interaction of one or more live organisms with the disease-causing pathogen or use of extract of plants. Some findings report the use of *Trichoderm*a isolates (Yao et al. 2016), *Chaetomium globosum* (Shanthiyaa et al. 2013), *Trichoderma viride* and *Pennicillium viridicatum* (Gupta 2016) and bacteria from the genera *Bacillus, Pseudomonas*, *Rahnella,* and *Serratia* (Daayf et al. 2003) as biocontrol agents in the management of late blight disease in potato. Garlic has, for example, been suggested as a potential intercropping plant for the management of potato late blight disease under Ethiopian condition (Kassa and Sommartya 2006). Still, few biological control measures are used by nonorganic growers due to low efficacy and farmers' lack of knowledge about these options and access to the most efficient products.

9.1.7 Looking Forward

Phytophthora infestans has proven capable of overcoming fungicides and resistant varieties through decades. Potato late blight, thus, continues to be the main potato constraint worldwide despite huge investments in its management. The growing intercontinental trade in potato is also increasing the risk of worldwide dissemination of dominant *P. infestans* strains. The threat from the disease will without doubt continue in the future. Fortunately, encouraging solutions to improve its management are arising from new advances in molecular, sensor, computational, and smartphone technologies. Efficient inoculum monitoring tools are becoming more accessible that can indicate whether *P. infestans* is in an area for the guidance of the initial fungicide spray and *P. infestans* population's movements (Fall et al. 2015). These could even be connected to molecular diagnostics that can predict phenotypic traits of the pathogen population, such as fungicide sensitivity and R-gene interaction, and perhaps even predictions of aggressiveness and fitness in a matter of hours. Spore traps could eventually be mounted on tractors or drones allowing for real-time spatial monitoring (Fall et al. 2015; Fry 2016). New high-throughput methods for monitoring and assessing plant populations are being developed, such as remote sensing, image processing, and web-based farmer-extension service networks. Such indirect disease detection approaches are improving disease surveillance significantly with high potential in remote and low-input production systems. Smartphone-based extension systems linked to rapid and portable molecular diagnostics kits (e.g. Loop-mediated isothermal amplification—LAMP—and lateral flow immunodiagnostics tools) can support immediate dissemination of disease information to a large number of farmers. If made widely available such smartphone tools, handheld portable diagnostics kits and novel weather sensors could lead to precision management and significant impacts on production capacity. Farmer-extension service networks based on internet connectivity are already important options that improve disease surveillance and supplying fast access to appropriate and up-to-date knowledge on pathogen distribution and management (Fry 2016). Capacity building (for farmers and extension services) to improve disease management skills, coupled to early-maturing and resistant cultivars, novel diagnostic tools, improved DSSs, and low-toxicity fungicides have the potential to reduce crop loss, management costs, and environmental impact, and even more so as biological options become accessible to farmers.

Current research opportunities in late blight management focus on *P. infestans* studies, fungicide testing, dissemination of resistant cultivars and validation of DSSs and training. In addition to continued research and extension efforts, alliances with the agro-chemical industry seem to be necessary to fully achieve integrated pest management strategies (Pacilly et al. 2016). This is being promoted through local and regional research networks that via collaboration strengthen institutional capacity in research and extension related to late blight (see http://euroblight.net/). Emerging research topics on *P. infestans* include the role of the pathogen–microbiota interaction in promotion or suppression of the disease

(Larousse and Galiana 2017) as well as the metabolism of *P. infestans* (Judelson 2017), its effector repertoire on the plant, and, *in fine*, how it promotes or suppresses the disease. At the same time, *P. infestans* as an infectious entity is no longer only considered at the species or lineage level but also at the level of a resident microbiota or part thereof (Larousse and Galiana 2017).

9.2 Early Blight

Hans Hausladen and Birgit Adolf

9.2.1 Symptoms

Foliar symptoms of early blight (EB) are dark brown to black necrosis. First foliar symptoms become visible on the lowest and therefore oldest leaves just a few weeks after emergence. Initially, dark brown dot-like blotches appear, a few millimeters in diameter. The necrotic area gradually increases and the leaf symptoms grow to take up the whole of the green leaf tissue (Fig. 9.6). Often, the lesions are restricted by leaf veins and take on an angular shape. The size of the necrosis can vary in width, from a few millimeters to 2 cm. Within larger lesions, a series of dark concentric rings are visible. This target pattern is typical of EB symptoms. Subsequently, the necrotic leaf tissue is often surrounded by a chlorotic border caused by fungal mycotoxins, which turn the leaf tissue yellow. The chlorosis can extend to the whole infected leaf. During EB progression the infected areas enlarge, and the whole leaf becomes necrotic and falls off. In Europe, a heavy increase of EB infestation occurs from mid-July onwards, especially during hot and dry weather or when the potato crop is under stress, and on physiologically older plants. EB then starts spreading from the lower leaves to the middle and finally upper leaf levels.

Alternaria conidia which are washed of the leaves can also infect tubers. The symptoms of EB on tubers are dark, slightly sunken lesions (Fig. 9.7). The dry or

Fig. 9.6 Initial (left) and advanced (right) symptoms of potato early blight in foliage

Fig. 9.7 Symptoms of potato early blight in tubers

hard rot of tubers causes storage losses, reduces quality of table potatoes, and reduces germination capacity of seed potatoes.

It isn't possible to distinguish between the different *Alternaria* spp. causing EB based on the symptoms even though sometimes symptoms referred to as brown leaf spot (small, irregular to circular, dark brown spots ranging in size from a pinpoint to 4 mm) are attributed to *A. alternata* (Fairchild et al. 2013).

9.2.2 Causal Organism

The main causal agent of early blight on solanaceous crops is generally considered to be *Alternaria solani* Sorauer (Gannibal et al. 2014). However, there are reports of other large-spored *Alternaria* spp. involved in EB of potato. Rodrigues et al. (2010) found that *A. grandis* Simmons, but not *A. solani*, was the causal agent of EB affecting potato plants in several growing regions in Brazil, and Duarte et al. (2014) confirmed that this species can cause EB on potato in field trials using artificial inoculation with *A. grandis*. *Alternaria protenta* has been detected as the causal *Alternaria* spp. for EB in Algeria (Ayad et al. 2017) and was, together with *A. grandis* and *A. solani*, found to be part of the complex of *Alternaria* spp. detected on EB lesions in Belgium (Landschoot et al. 2017a). Of the small-spored *Alternaria* spp., *A. alternata* occurs on EB lesions on a regular basis, but is considered as a secondary invader (Leiminger et al. 2014; Stammler et al. 2014; Rotem 1994).

Alternaria solani overwinters as mycelium, chlamydiospores or conidia in the soil and infested plant debris (see disease cycle in Fig. 9.8). In spring, the primary infection occurs through inoculum (conidia) carried to the lower leaves by rain splashes. The pathogen is able to penetrate the leaf tissue directly through the intact epidermis or through stomata and wounds. First symptoms of EB on leaves become visible 2–4 weeks after the emergence of the potato crop. Initially, the older leaves closer to the ground are infested. The fungus is restricted to the lower leaf level for several weeks and seems initially to be of no concern. Conidia formation occurs on

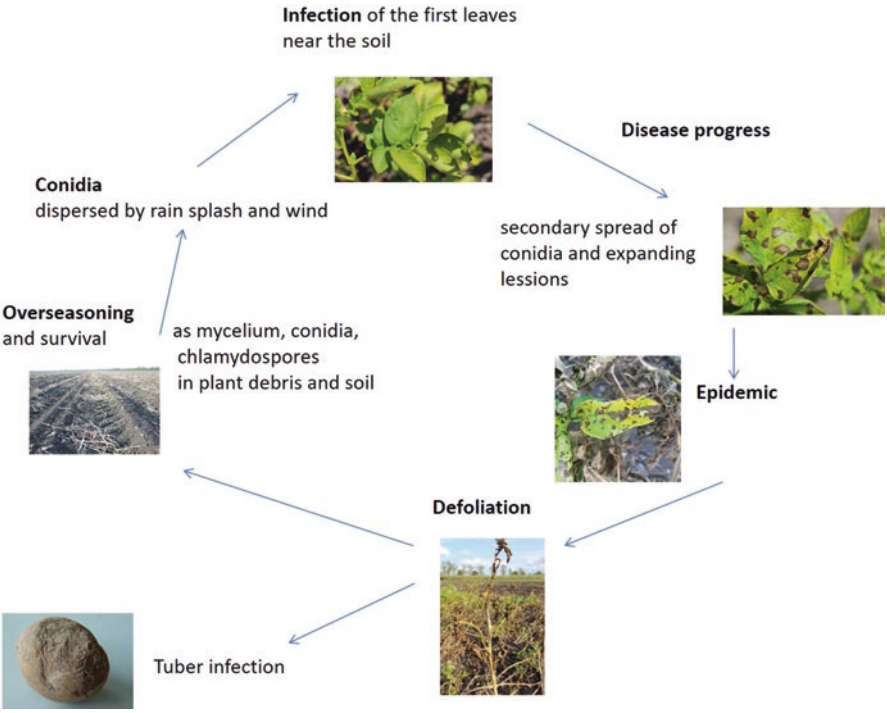

Fig. 9.8 Disease cycle of potato early blight

the necrotic leaf tissue at temperatures between 5 and 30 °C (optimum 20 °C). This secondary inoculum is disseminated by wind and causes infections of the surrounding leaves and stems. The latency period averages 3–7 days. During favorable infection conditions, and once the plant has got to a certain age, *A. solani* very rapidly colonizes the middle and upper leaf layers. In the field, a cascade-like progression of the fungus from the lower, via the middle, to the upper leaves is visible. Heavily infested leaves fall off and remain as inoculum source on and in the soil.

The disease progression of EB depends on weather parameters, plant age/crop growth stage, maturity group, susceptibility of the cultivar and inoculum concentration, the latter being influenced by short crop rotations.

Favorable weather conditions for infection with *A. solani* are temperatures above 22 °C and leaf wetness for more than 8 h. Furthermore, alternating wet and dry periods favor disease development.

An important factor for disease progression is the crop growth stage. In addition to the cascade-like spread within plants upwards from older to younger leaves, older plants are significantly more susceptible due to the earlier planting emergence time.

Tuber infestation is caused by conidia that are washed into the soil. The fungus can enter the tuber tissue through lenticels or mechanical injuries of the skin during harvest.

EB can also occur on other host plants apart from potatoes. It has been observed on hosts such as tomato (*Solanum lycopersicum* L.), eggplants (*S. melongena* L.), hairy nightshade (*S. sarrachoides* Sendt), black nightshade (*S. nigrum* L), horse nettle (*S. carolinense* L.), pepper (*Capsicum* spp.), and non-solanaceous weeds (Jones et al. 1993; Pscheidt 1985; Hausladen and Aselmeyer 2017).

Differentiation of *Alternaria* species within the large-spored or the small-spored group based on morphological traits is time-consuming and requires experience. The molecular approach to delineate large-spored isolates fast and precise is a multi-locus analysis based on different partial gene regions, like the internal transcribed spacer regions 1 and 2 and intervening 5.8S nrDNA (ITS), glyceraldehyde-3-phosphate dehydrogenase (GAPDH), RNA polymerase second largest subunit (RPB2), translation elongation factor 1-alpha (TEF1), the Alternaria major allergen gene (Alt a 1) (Woudenberg et al. 2014). Based on the RPB2 and calmodulin gene sequence *A. solani* can be distinguished from A. grandis and A. protenta (Landschoot et al. 2017a). For the small-spored species the glyceraldehyde-3-phosphate dehydrogenase (gapdh), RNA polymerase second largest subunit (rpb2), translation elongation factor 1-alpha (tef1), Alternaria major allergen gene (Alt a 1), endopolygalacturonase (endoPG), an anonymous gene region (OPA10-2), and the histone H3 gene can be used for differentiation (Woudenberg et al. 2015; Landschoot et al. 2017a).

9.2.3 Impact

After potato late blight (*Phytophthora infestans*), EB represents one of the most important fungal diseases of potato today. It can be found in most potato-growing countries (Woudenberg et al. 2014), but *A. solani* is described as a very important pathogen especially in warmer areas due to the requirement for higher temperatures. In susceptible potato cultivars in particular, as well as in locations with increased occurrence of *A. solani*, the disease can cause considerable yield losses. The potential EB-induced damage for different countries is shown in Table 9.1.

The necrosis of leaf tissue and considerable defoliation caused by *Alternaria* infestation reduces the assimilation area of the potato plant, and therefore has a

Table 9.1 Yield loss due to early blight in different regions

Country	Yield loss
Australia	>20% (Horsfield et al. 2010)
Brazil	up to 58% (Campo Arana et al. 2007)
Germany	2– >40% (Leiminger and Hausladen 2014)
Poland	6–34% (Kapsa 2004)
Israel	up to 24% (Shtienberg et al. 1996)
United States	18–39% (Harrison and Venette 1970)
South Africa	20–50% (Van der Waals et al. 2001)

negative effect on tuber size and starch content. *Alternaria solani* is also able to infect tubers. The subsequent hard rot of the tubers leads to reduced quality for marketing as table and processing potatoes. In Europe, the occurrence of tuber infections is known only in exceptional years.

9.2.4 Pathogen Populations

The genetic diversity among *A. solani* isolates is relatively high for an asexually reproducing fungus. Furthermore, when analyzed by virulence assays (VC), random amplified microsatellites (RAMS), random amplified polymorphic DNA (RAPD), and AFLP marker techniques, there is no clear clustering of isolates according to geographical origin, year, or even deriving from the same field (Van der Waals et al. 2004; Leiminger et al. 2013; Odilbekov et al. 2016). Polymorphism exists even within the usually strongly conserved mitochondrial DNA. Concerning the cytb gene, Leiminger et al. (2014) detected two genotypes in German *A. solani* populations which differed in their intron–exon structure. The occurrence of the two genotypes was confirmed for *A. solani* populations in the USA (Bauske et al. 2018), Belgium (Landschoot et al. 2017b), and Sweden (Odilbekov et al. 2016).

The registration of *Alternaria*-specific fungicides with single site modes of action for potato changed the population structure of *A. solani* in many countries due to the occurrence of point mutations, leading to reduced sensitivity of mutated isolates. For Quinone outside inhibitor fungicides (QoIs) like Azoxystrobin, the change of amino acid phenylalanine to leucine at position 129 in the cytb gene (F129L) has been demonstrated to be the reason for reduced sensitivity compared to wild-type isolates (Pasche et al. 2005). The presence of the F129L mutation in *A. solani* populations has been shown in different countries such as the USA (Pasche et al. 2004), Germany (Leiminger et al. 2014), Sweden (Odilbekov et al. 2016), and Belgium (Landschoot et al. 2017b). The loss in sensitivity towards succinate dehydrogenase inhibitor fungicides (SDHIs) like Boscalid can be caused by different point mutations, as the SDH is composed of four subunits and the binding site of the fungicides is formed by three of them (subunits B, C, D). For *A. solani* populations, five possible mutations have been described so far: H278Y and H278R in subunit B, H134R and H134Q in subunit C, and D123R and H133R in subunit D (Mallik et al. 2014; Metz et al. 2019). Landschoot et al. (2017b) describe the presence of two different SDH mutations in one isolate. Frequently, isolates carry both the F129L mutation in the cytb gene and one of the SDH mutations (Landschoot et al. 2017b).

The sensitivity of baseline isolates to demethylation inhibiting fungicides (DMIs) can vary substantially, but nonbaseline isolates remain sensitive, whereas a distinct loss in sensitivity to anilino-pyrimidine (AP) fungicide pyrimethanil exists. The primary mode of action has not yet been discovered for AP chemistries; therefore the resistance mechanism is currently unknown for this established fungicide group (Fonseka and Gudmestad 2016).

Resistance against QoIs in *A. alternata* populations is caused by the G143A mutation in the cytb gene (Ma et al. 2003). Mutations conferring reduced sensitivity to SDHIs for this Alternaria species are the H277R/Y in SDH subunit B, H134R in subunit C, and D123R and H133R in subunit D (Avenot et al. 2008, 2009). Fairchild et al. (2013) detected isolates resistant to pyrimethanil and Fonseka and Gudmestad (2016) stated high DMI EC50 values in baseline isolates.

9.2.5 Management

Integrated pest management to control early blight requires the implementation of several approaches. The disease is primarily controlled by the use of cultural practices (to reduce the soil born inoculum), less susceptible cultivars and the use of pesticides.

Phytosanitary aspects One of the main components in this case is the crop rotation, which influences the occurrence of early blight. The fungus *A. solani* persists as mycelium or spores in plant debris or soil in the field from one potato-growing season to the next. Therefore crop rotation, including the control of host plants such as weeds (black shadow) in the nonhost crops, reduces the initial soil born inoculum. A short crop rotation with host crops (tomato, potato) results in an earlier and more severe early blight epidemic (Shtienberg and Fry 1990). In addition, the removal or burning of infected plant debris reduces the inoculum level.

A further option to reduce the primary inoculum in the soil is biofumigation. Biofumigation means the suppression of soilborne pathogens by isothiocyanates (ITCs), which derive from hydrolization of glucosinolates by myrosinase in disrupted plant cells. Biofumigant plants (e.g. white mustard, leaf radish) can reduce the early blight disease progression in the crop (Volz et al. 2013).

Tuber harvest and storage *Alternaria solani* also can infect the potato tuber. The fungus cannot infect through the intact periderm, and so the risk of tuber infection can be reduced by allowing tubers to fully mature before harvest. Avoiding wounding at harvest and providing storage conditions to promote wound healing can also reduce tuber infection (Venette and Harrison 1973).

Pathogen-free seed The use of disease- and virus-free seed potatoes is the basis for an economical potato production. Virus-infected potato plants are more susceptible to early blight than healthy plants.

Biotic and abiotic stress Potato plants stressed by biotic or abiotic factors are more susceptible to early blight disease compared to nonstressed plants. There are different types of abiotic stress for plants during the growing season, such as drought, high temperature, and overhead irrigation. Additionally, overhead irrigation can prolong the leaf wetness period, allowing successful fungal infection.

Biotic stress is driven by insects (e.g. aphids, Colorado beetle), which are also known to be virus vectors.

Plant nutrition A balanced nutrition for the potato plants during the growing period is the basis for optimal plant growth and potato yield. Ideal soil fertility and plant nutrition can decrease the severity of *A. solani* (Lambert et al. 2005; MacDonald et al. 2007). Under specific conditions, such as drought when plants are unable to get enough nutrients from the soil through the roots, a foliar fertilizer can reduce the nutrient deficiency and reduce plant susceptibility to early blight.

The fertilizer form can also influence the disease progression of *A. solani*. The use of calcium cyanamide results in a delay of early blight disease, as the fungicidal side effects of degradation products of calcium cyanamide can reduce the initial inoculum in the soil (Volz et al. 2013).

Resistant cultivars Cultivars with reduced susceptibility to early blight are available; however, no completely resistant genotypes have been found so far. The observed field resistance of varieties to foliage infection is associated with plant maturity. Early maturing cultivars are in general more susceptible, and late maturing cultivars are more resistant to *A. solani* (Johanson and Thurston 1990; Abuley et al. 2017). There is no correlation between maturity group and the occurrence of the first early blight symptoms on the leaves, but a strong correlation between maturity group and disease progression. The epidemic in early maturing cultivars starts earlier. Interestingly, there are varieties within a maturity group which are more resistant to early blight (Johanson and Thurston 1990; Leiminger and Hausladen 2014). Overall, there is a possibility of influencing disease progression by planting more resistant cultivars.

Use of fungicides The most common method for controlling early blight in potatoes is the use of chemical pesticides. Some fungicides which are used for the control of late blight (Phytophthora infestans) also have some effect on Alternaria solani (e.g. maneb, mancozeb, chlorothalonil, triphenyl tin hydroxide). The most effective fungicides for control of early blight contain active ingredients from the strobilurins group, and azols.

Strobilurins, also known as Quinone outside inhibitors (QoIs), are an important class of fungicides in agriculture because they have a broad-spectrum activity. They inhibit mitochondrial respiration in fungi by binding to the Qo site of the cytochrome b (cytb) complex, blocking electron transfer and inhibiting ATP synthesis (Bartlett et al. 2002).

Carboxamides (SDHI) inhibit the enzyme succinate dehydrogenase (Sdh), a component of complex II in the mitochondrial electron transport chain (Kuhn 1984). Despite the two groups of fungicides having a similar mode of action, SDHI and QoI fungicides show no cross-resistance.

The triazol group belongs to the DMI fungicides, which inhibit one specific enzyme, C14-demethylase. This enzyme plays an important role in ergosterol bio-

synthesis. Ergosterol is important for functional membrane structure and for the development of functional cell walls.

Due to the single site mode of action of strobilurins (QoI) and carboxamides (SDHI), these fungicides have a high risk of development of resistance. In several potato-growing areas, mutants are found which show a reduced sensitivity in in vitro and in vivo trials and also a reduced efficacy in the field (see Pathogen populations).

Most of these fungicides have a very limited curative activity and should be used preventively. EB control is mainly achieved by multiple and frequent application of protectant fungicides. The optimization of fungicide usage for the control of early blight is a considerable challenge. The fungus produces huge amounts of secondary inoculum during the growing season. Therefore different DSS (decision support systems) are available to optimize the use of fungicide applications for the management of early blight in potato. One possibility is to use threshold values based on the disease progress (Leiminger and Hausladen 2012). In some countries, disease management is based on interactive computer-based systems dealing with forecasting favorable weather conditions for infection by Alternaria solani or temperature degree-day thresholds. Alternatively, the recommendation for the use of fungicides is based on plant development (plant size, plant age, onset of potato flowering).

Overall, the combination of plant age and host resistance, disease progress, and calculated weather-based infection risk is used as the basis for an integrated pest management.

9.2.6 Looking Forward

The control of early blight will be a considerable challenge in the future. The increasing importance of early blight in potatoes is due to a number of factors. Climatic change and global warming will result in more conducive conditions for the infection, growth, and disease progress of the fungus in several potato-growing areas. Increasing temperatures during the growing season result in favorable conditions for the pathogen, due to its requirement for higher temperatures. More abiotic stress (drought) increases the susceptibility of the plant, and will therefore also promote disease progress.

In addition, integrated pest management based on continuous pesticide treatments will become more and more ineffective. The occurrence of mutants towards different modes of action will result in reduced efficiency in the control of EB by fungicides. Therefore in the future, successful control of EB requires the implementation of cultural practices, the use of pesticides with a focus on fungicide resistance development, and the cultivation of less susceptible cultivars.

9.3 Wart

Jaroslaw Przetakiewicz

9.3.1 Causal Organism

The pathogen causing potato wart disease, *Synchytrium endobioticum* (Schilb.) Perc., was first discovered by Schilberszky in Hungary. In fact, the pathogen was known firstly in Europe. In 1876, potato wart disease was found for the first time in the UK (Hampson 1993; Flath et al. 2014). In the older classification this species has been included to Protista Kingdom. Nowadays, *Synchytrium endobioticum* (Schilb.) Perc., belongs to Fungi Kingdom, phylum Chytridiomycota, order Chytridiales, family Synchytriaceae, genus *Synchytrium*, and species *endobioticum*. The genus *Synchytrium* included about 200 species which are endobiotic halocarpic organisms that have inoperculate sporangia. All species of the genus *Synchytrium* are parasites but the most important economically and phytosanitary is *S. endobioticum* the causal agent of potato wart disease. The disease is also known by various common names like black wart, cauliflower disease, warty disease, potato tumor, potato cancer, black cancer, or black scab. *S. endobioticum* is an obligate soil-borne biotrophic fungus which is considered to be the most important worldwide quarantine plant pathogen of cultivated potato. Cultivated potato (*Solanum tuberosum*) is the primary host, but the fungus, under experimental conditions, can also infect wild species in genera *Capsicastrum*, *Duboisia*, *Hyoscyamus*, *Lycium*, *Nicotiana*, *Nicandria*, and *Physalis* (Obidiegwu et al. 2014). The pathogen is a primitive fungus which stimulates its host to produce hypertrophic outgrowths on young potato organs, such as eyes, sprouts, young tubers, stolons, stems, leaves, and even flowers but never roots. The fungus does not form hyphae but forms sporangia that produce about 200–300 motile zoospores (Obidiegwu et al. 2014). After infection, *S. endobioticum* produces two different kinds of sporangia in the galls. Summer sporangia (Fig. 9.9) have a thin cell wall and form haploid zoospores which are emerging and steady reinfection of the host tissue like sprouts, tubers, eye tubers, stolons, and roots (only in tomato) (Przetakiewicz 2014a). In appropriate conditions after isogamy of haploid zoospores to diploid zygotes which are able to infect host cells and form winter sporangia which are embedded deeper into the host tissue than the sori (always on the surface). Winter (resting) sporangia (Fig. 9.9) are the dormant structures by which the fungus disperses to establish new infections. They are usually spherical to ovoid in shape and 24–75 μm in diameter with thick-walled (triple wall) structure, which is ornamented with irregularly shaped wing-like protrusions. The spores can survive for a long time without plant hosts. After 43 years, in favorable conditions, disease may develop even from single spores of *S. endobioticum* (Przetakiewicz 2015b, 2016).

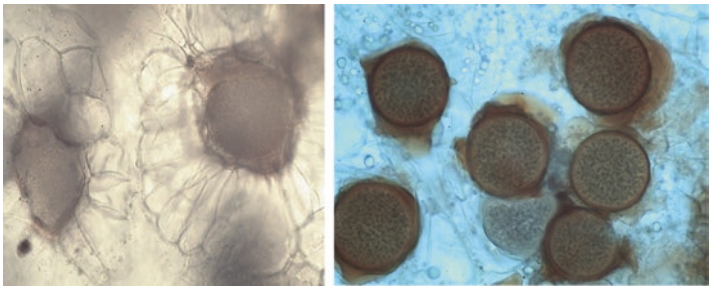

Fig. 9.9 Summer (left) and winter (resting) sporangia (right) of *Synchytrium endobioticum*. Thin-walled summer sporangia of pathotype 3(M1) are enclosed within one membrane forming sorus on surface of wart tissue of cultivar Asche Sämling. Thick-walled winter sporangia of pathotype 8(F1) in wart tissue of cultivar Sonda

Fig. 9.10 Symptoms of potato wart disease above ground (left) and underground (right). The galls on stem and foliage above ground are colored green, while subterranean warts on stem base, stolons, and tubers are colored white. Pathotype 2(Ch1) on cultivar Cykada (left). Pathotype 18(T1) on cultivar Deodara (right)

9.3.2 Symptoms

Usually, the symptoms of potato wart disease are not visible on plants, although there may be a reduction in plant vigor. Only in very suitable conditions small greenish warts (Fig. 9.10) might be visible on the top of plants: stem, foliage and in extremely conditions on inflorescences. (Obidiegwu et al. 2014). In the most cases the symptoms are visible on underground parts of potato (Fig. 9.10) on stolons, stems, bulbs, eye of matured tubers. In soil with the high content of winter sporangia (above 500/g) may lead to infection all eyes of seed potato and develop only warts without any emergences of potato (Przetakiewicz 2014a). The typical symptoms of the disease on tubers are the proliferating warts which may vary markedly in form but are primarily spherical to irregular. The warts are usually whitish

or green if exposed to light, but gradually darken eventually rot and disintegrate. Rarely, the warts can be yellow or purple to brown. The color is dependent on potato variety. If variety produces purple sprouts, then the warts will be purple too. The warts are similar to cauliflower and they usually have the same color but sometimes they are compared to walnut kernel (Przetakiewicz 2014a). The warts are proliferated to gall. Galls vary markedly in form but are primarily spheroid. They are primarily parenchymatous and phytoteratological and not phytooncological. Although, the disease is described as potato tumor or cancer as well as black cancer they should not be referred to as tumors (Hampson 1993). Warts differ in size from pea-sized nodules to the size of a fist. The warts maintain in lab condition (for inoculum production) can reach 220 g from one eye/sprout. At maturity the galls become colored black and lead to total tuber decay (Przetakiewicz 2014a).

Simultaneous germination of all buds in one eye results in wart-like outgrowths very similar to those caused by *S. endobioticum*. However, these pseudo-warts consist of abundant pointed shoots compacted together. No winter sporangia are present in the affected tissue. Symptoms of powdery scab caused by *Spongospora subterranea* f.sp. *subterranea* or potato smut caused by *Thecaphora solani* can be mistaken for wart occurrence. A view under the microscope reveals that spore balls look different from winter sporangia of *S. endobioticum*. The size of warts depends not only on environmental conditions (cool summer, wet soil, etc.) but on potato variety. On extremely susceptible variety specified pathotype of *S. endobioticum* can differ markedly to very big size producing winter sporangia in the last stage of growing. On the slightly susceptible variety specified pathotype of *S. endobioticum* is able to produce very small warts or only to influence on weakly proliferation of host tissue but producing winter sporangia in the first stage of infection. The spores were visible after 13 days after inoculation of slightly susceptible varieties. In uncomfortable conditions (high temperature or dry soil) warts stop growing and begin to produce winter spores (Przetakiewicz 2014a).

9.3.3 Impact

Potato wart disease is so important that, for some 65 years, quarantine and domestic legislations have been in force throughout the world to prevent its spread (Anon 2015). The economic impact of disease caused by this pathogen is not only from disease losses but from loss of international trade markets, long-term quarantines, and regulatory restrictions placed on infested areas and the buffer zones (Przetakiewicz 2014a). Chemical control of *S. endobioticum* is not possible. The only strategies to confine the disease are strict quarantine and phytosanitary measures as well as cultivation of resistant cultivars (Obidiegwu et al. 2014). The availability of resistance cultivars allowed governments to issue regulations prohibiting the cultivation of susceptible cultivars (Baayen et al. 2006). For example, since 1955 only cultivars resistant to pathotype 1(D1) could be registered and grown on the Polish territory. As stated in the Food and Veterinary Office Mission Report, no

potato plants or tubers with symptoms of *S. endobioticum* pathotype 1(D1) have been detected in Poland since the 1950s–early 1960s. This is a result of growing only resistant cultivars to mentioned pathotype of *S. endobioticum* (Przetakiewicz 2008). Worldwide prevention is based on the control of disease spread and pathogen exclusion via regulatory action. *S. endobioticum* has a very limited capacity for natural spread, which is principally why it has been possible to control it so effectively by statutory means. Nevertheless, *S. endobioticum* is a classic example of the distribution of plant pathogens by man. Regulatory action has largely restricted the spread of the disease within potato-growing regions, as the seriousness of the disease was quickly recognized (Hampson 1993). Once *S. endobioticum* has been introduced into a field, the whole crop may be rendered unmarketable and moreover the fungus is so persistent that potatoes cannot be grown again safely for many years, nor can the land be used for any plants intendent for export. Based on the results obtained by Przetakiewicz (2015b), winter sporangia of *S. endobioticum* are very persistent and capable of retaining viability for as long as 46 years. The single spores from the inter-host period were still infective after 43 years. These results should be taken into consideration when de-scheduling previously infested plots even after 40 years or longer, especially in the mountainous areas. *S. endobioticum* is still of great economic importance in cool areas and wet mountainous regions. The detection of potato wart disease on Prince Edward Island during the 2000 growing season resulted in an estimated $30 million loss to the island's economy in the first year.

9.3.4 Pathogen Populations and Distribution

Potato wart disease appears to have arisen in the potato-growing area of Andean South America (Hampson 1993). Wart-like outgrowths on early Peruvian tuber-shaped pottery were interpreted as potato wart disease. The disease is likely to have arrived in Europe from South America because the Great Potato famine of the 1840s in Ireland induced European growers to import potato germplasm from South America. The introduction of the pathogen to Europe was possible by diseased tubers, infested soil or contaminated bags along with shipments of guano (Hampson 1993). *S. endobioticum* spread at the end of the nineteenth century from the center of origin in the Andes first to Europe and North America and subsequently across whole potato-growing areas of Asia, Africa and Oceania. Historic account has it that potato wart disease entered England in 1876 or 1878 while another view upholds that the disease has been present in the Liverpool province of England in 1876 or 1878 (Obidiegwu et al. 2014). In 1901, the disease was officially recognized in the UK. Potato wart disease and its causal agent were described by Schilberszky who received in 1888 warted tubers of cultivar Maercker-Zwiebel that had been grown locally from seed tubers imported from England. It spread widely in Europe, but statutory measures finally restricted its distribution and it has spread only to a limited extent to other parts of the world. According to EPPO Pest Quarantine Database

(Anon 2015), *S. endobioticum* occurs locally in almost all EPPO countries. The distributions are fragmentary as a result of statutory control. According to national reports, it has been found but is not established in Belgium, France, Luxembourg as well as in Lebanon. Found in the past but eradicated in Portugal (unconfirmed). In Asia countries *S. endobioticum* occurs in Armenia, Bhutan, China, India, Georgia, Nepal, Turkey. In Africa: South Africa, Tunisia, Algeria, Zimbabwe, and Egypt (absent, unreliable record). North America: Canada, Mexico (absent formerly present), USA (eradicated). South America: Bolivia, Chile (eradicated), Ecuador, Falkland Islands, Peru, Uruguay (absent, confirmed by survey). Oceania: New Zealand (South Island). Numerous pathotypes of the fungus occur and are defined by their virulence on differential potato cultivars. In Europe, more than 40 pathotypes of *S. endobioticum* have already been identified (Obidiegwu et al. 2014; Przetakiewicz 2014b, 2015a). A pathotype is characterized by its pattern of virulence or avirulence to a series of differential cultivars of potato. Pathotypes 1(D_1), 2(G_1), 6(O_1), 8(F_1), and 18(T_1) are the most relevant in Europe. Other pathotypes occur mainly in the rainy mountainous areas of central and eastern Europe (Alps, Carpathians). They persist mainly in small garden potato plots and not in commercial potato crops (Przetakiewicz 2014a). Mitochondrial genomic variation shows that *S. endobioticum* has been introduced into Europe multiple times, that several pathotypes emerged multiple times, and that isolates represent communities of different genotypes (van de Vossenberg et al. 2018a).

9.3.5 Resistance

The biggest discovery was finding resistant cultivars of potato to *S. endobioticum* among cultivated ones. Systematic studies on resistance of potato cultivars to *S. endobioticum* started in England in 1909. Resistance sources to *S. endobioticum* were found in old cultivars such as Snowdrop and Flourball, which facilitated resistance breeding. Conventional breeding programs were successful in controlling potato wart disease through the development of resistant cultivars early in the twentieth century (Obidiegwu et al. 2014). Breeding for resistance was successful, thanks to the arability of a dominant gene that blocked development and reproduction of originally introduced pathotype 1(D1) of *S. endobioticum*. Unfortunately, in Europe wart development on resistant potato cultivars was first discovered in 1941 in Germany [pathotype 2(G1)] and former Czechoslovakia [pathotype 3(S1)]. The new pathotypes have been proved to be difficult to control and eradicate then the original pathotype 1(D1) (Baayen et al. 2006). Molecular mapping studies provide evidence that wart resistance to pathotype 1(D1) of *S. endobioticum* can be conferred by a single locus from different sources. Hehl et al. (1999) mapped the single dominant gene *Sen 1* for resistance to pathotype 1(D1) in diploid mapping population. The gene *Sen 1* is located on potato chromosome IX. Brugmans et al. (2006) also used a diploid potato linkage map to locate *Sen1-4*, a second dominant gene for resistance to pathotype 1(D1). This gene is located on the long arm of

chromosome IV. In these two mentioned populations, the resistance segregated as a monogenic trait. Ballvora et al. (2011) discovered the first loci for virulent pathotypes 2(G1), 6(O1), and 18(T1) in two tetraploid half-sib families, in which the resistance to pathotype 1(D1) also segregated. In contrary to earlier studies in diploid populations (Hehl et al. 1999; Brugmans et al. 2006), the phenotypic distribution of wart resistance appeared quantitative in the two mapping populations analyzed (Ballvora et al. 2011). The quantitative resistance locus (QRL) *Sen2/6/18* on chromosome I expressed resistance to pathotypes 2(G1), 6(O1), and 18(T1). The QRL *Sen18* on chromosome IX expressed resistance to pathotype 18(T1). And the third QRL Sen1 on chromosome XI expressed resistance mainly to pathotype 1(D1) (Ballvora et al. 2011). Groth et al. (2013) mapped quantitative trait loci (QTL) for resistance to pathotype 1(D1), 2(G1), 6(O1), and 18(T1). The QRL for all four pathotypes were located on chromosomes II, VI, VIII, and IX, and QRL for pathotypes 2(G1), 6(O1), and 18(T1) on chromosomes VII and X. The QRL detected in this study were different from the ones in Ballvora et al. (2011). The cultivar Panda used in this study (Groth et al. 2013) as a resistant parent to pathotype 1(D1), 2(G1), 6(O1) and 18(T1), is in fact slightly susceptible to all four pathotypes. These results were confirmed in CORNET project (acronym SynTest) (Przetakiewicz, unpublished results 2014) Consequently, breeding is hampered by a lack of dominant major genes for resistance to virulent pathotypes. The wart resistance has several sources and diverse backgrounds which resulted in a few potato cultivars resistant to virulent pathotypes of *S. endobioticum*. Nevertheless, recent results indicated for identify a newly locus *Sen2* located on chromosome XI which provides resistance to at least seven various virulent pathotypes of *S. endobioticum* (Plich et al. 2018).

9.3.6 Management

The first acts were issued in 1908 in Ireland, and a few years later in Scotland, England, and Germany. The first legislation related primarily to prohibit the import from other countries of potatoes infected by *S. endobioticum.* In that time the acts did not take into account the possibility of the spread of the disease within the country. It was a reason of increasing number of outbreaks in such a short time in many countries. In consequence of the wide spread of potato wart disease was the development of research on the biology controlling of the fungus. EPPO includes *S. endobioticum* into the A2 quarantine list because of its long persistence in soil and the severe losses it inflicts to potato crops. The United State Department of Agricultural (USDA) has the fungus on its official list of selected agents and toxin. The European Union issued a specific requirement in the Council Directive 69/29/EC of 8 December 1969 on control of Potato wart disease and the Council Directive 2000/29/EC of 8 May 2000 on protective measures against the introduction into the Community of organisms harmful to plant or plant products and against their spread within the Community (Obidiegwu et al. 2014).

9.3.7 *Looking Forward*

Although *S. endobioticum* seems to be in remission in Europe, new foci have appeared in countries with warmer continental climate (Turkey, Georgia, Bulgaria, and Greece) (Anon 2015; Gorgiladze et al. 2014; Vloutoglou pers comm. 2015). This may suggest adaptation of *S. endobioticum* to warmer and/or dryer climate as well as the lack of adequate controls in countries where the disease has not been present before. Moreover, new pathotypes have been discovered recently (Çakir et al. 2009; Przetakiewicz 2015a). It can be expected that new pathotypes of *S. endobioticum* might appear, since there are many reasons for new pathotypes to arise (Melnik 1998). The newest Pathotype 39(P_1) was detected in the rainy mountainous area, in small garden potato plots, when the old traditional cultivars of potato are cultivated without any crop rotations. It seems therefore that where climatic conditions are suitable for *S. endobioticum* to take place and the growing of slightly susceptible cultivars is possible, the development of new pathotype is favored (Przetakiewicz 2015a). Although, local pathotypes persist mainly in small garden potato plots in economically unimportant potato-growing regions, they are still very important for quarantine and phytosanitary measures as spores can spread in rain water (Przetakiewicz 2015b). Phenotypic assessment of resistance to *S. endobioticum* is laborious and, time-consuming. Diagnostic DNA-based markers closely linked with or, even better, located within wart resistance genes would greatly facilitate the early detection and combination of different resistance sources and are therefore highly desirable (Obidiegwu et al. 2014). Natural DNA variation in wild and cultivated potato germplasm provides an excellent platform for the discovery of diagnostic tools for marker-assisted selection and resistance gene cloning (Obidiegwu et al. 2014). A new dominant gene *Sen2* on chromosome XI provides extreme resistance to pathotypes 1(D1), 2(G1), 2(Ch1), 3(M1), 6(O1), 8(F1), 18(T1), and 39(P1). In the future, this gene will offer potentials for the efficient selection of new commercial cultivars that are resistant to multiple *S. endobioticum* pathotypes. EPPO Standard PM 7/28 (2) (Anon 2017) recommends various biotests using differential potato cultivars for the identification only 4 pathotypes [1(D1), 2(G1), 6(O1) and 18(T1)]. Pathotype determination is labor-intensive and time-consuming too, especially for the identification of local pathotypes which require more differential cultivars (Przetakiewicz 2017; Przetakiewicz and Plich 2017). Molecular diagnostic tools (TaqMan PCR method) are currently available for the identification of pathotype 1(D1) and its discrimination from non-1(D1) pathotypes (Bonants et al. 2015; van de Vossenberg et al. 2018b). However, this method requires costly probes and cannot be used for the identification of virulent pathotypes. The recent report revealed no sequence polymorphisms between the five *S. endobioticum* pathotypes, indicating that *S. endobioticum* pathotypes may be exceptionally similar to each other. One reason for this may be that the development of new *S. endobioticum* pathotypes is caused by very limited changes in avirulence factors rather than extensive genetic recombination between divergent genotypes (Busse et al. 2017). Recently, polymorphic microsatellite markers were used to assess the

genetic diversity of potato wart at the intraspecific level for the first time and will certainly contribute to a better understanding of the evolutionary history of this pathogen in the years to come (Gagnon et al. 2016). The molecular methods for *S. endobioticum* detection based on the RealTime PCR (Smith et al. 2014; Bonants et al. 2015) or PNA-based hybridization assay (Duy et al. 2015) have several advantages over the traditional method of microscopic examination in routine diagnostic testing. Molecular methods can significantly reduce the time to disease diagnosis and prevent the spread of *S. endobioticum* to other locations.

9.4 Powdery Scab

Alison Lees, Francisco Bittara, and Gary A. Secor

9.4.1 Introduction and Future Perspectives

Spongospora subterranea causes root hyperplasia (root galling) and tuber powdery scab, leading to losses in seed and ware crops of potato worldwide and is also important as the natural vector of Potato mop-top virus (PMTV), an economically important tuber blemish disease of potato found in some regions. Powdery scab is particularly favored by cool, damp conditions and is an intractable disease. Additionally, *S. subterranea* is an unculturable biotroph, making research difficult.

Progress in understanding many aspects of the biology of *S. subterranea* has been slow relative to other plant pathogens and even compared with other plasmodiophorids. However, the development of quantitative molecular assays and the increasing availability of sequence information have allowed recent progress to be made in understanding various aspects of the epidemiology of powdery scab and root galling, and biology of the pathogen.

The pathogen can survive for many years and very low levels of inoculum can cause relatively severe disease outbreaks, making control elusive: it is generally accepted that an integrated approach to disease control will prove most effective as no single control method is totally effective. Additional knowledge of inoculum-based risk, pathogen variation and infection conditions will all contribute towards such a risk-based integrated control system.

The tripartite potato/*S. subterranea* /PMTV interaction is a difficult system to manipulate and there has been little progress in understanding the basis of host specificity and molecular mechanisms of virus transmission. In recent years some progress has been made in developing molecular markers and genomic sequence information for the plasmodiophorids. In addition, host genomics has advanced with the discovery of genetic markers and identification of novel resistance. The availability of in vivo root culture systems for *S. subterranea* propagation, high-throughput nucleotide sequencing, comparative genomics and advanced

imaging technologies will no doubt be applied in due course in order to advance knowledge of this intractable and understudied disease and the virus that it vectors. It is envisaged that availability of plasmodiophorid genomes will, in the future, lead to molecular interaction studies between *S. subterranea* and compatible host plants, and will allow the genes or molecular mechanisms involved in host recognition, infection of host cells, multiplication of *S. subterranea* within roots and the development of sporangial or sporogenic phases to be identified.

9.4.2 Causal Organism

The taxonomy of the plasmodiophorids is complicated and has previously been described in detail (Braselton 1995; Dick 2001; Down et al. 2002). The genus *Spongospora*, once ascribed to the fungi, is now considered to be a member of the family *Plasmodiophoridae* within the Super-group Rhizaria, Phylum Cercozoa, under the Class Phytomyxea, which includes members parasitic to higher plants and Stramenopila and which usually cause hypertrophy in the host cells (Bulman and Braselton 2014; Neuhauser et al. 2010). Traditionally, the species *S. subterranea* has been divided into two *formae speciales: Spongospora subterranea* (Wallr.) Lagerheim f.sp. *nasturtii* Tomlinson (the cause of crook root of watercress) and *Spongospora subterranea* (Wallr.) Lagerheim f.sp. *subterranea* (the cause of powdery scab and root galling on potato) (Neuhauser et al. 2010). However, host specificity characteristics, differences in sporangial states and habit as well as molecular data have provided additional evidence to support their placement into the species rank (i.e. *S. subterranea* and *S. nasturtii*; Bulman and Braselton 2014; Dick 2001; Gau et al. 2013; Neuhauser et al. 2010; Qu and Christ 2004) and they will be referred to as such hereafter. Due to the uncertain taxonomy of the group, the collective term "plasmodiophorids" is also commonly used (Braselton 1995). Key features of the plasmodiophorids are that they have zoospores with two anterior undilopodia ("flagella"), multinucleated protoplasts (plasmodia), and environmentally resistant resting spores, and that they are biotrophic parasites. Plasmodiophorids are a monophyletic group with cruciform nuclear division, obligate intracellular parasitism, biflagellated zoospores, and environmentally resistant resting spores (Bulman et al. 2001; Qu and Christ 2004).

A detailed description of life cycle of *S. subterranea* was given by Harrison et al. (1997). There are two major phases in the life cycle of *Spongospora*, each initiated by host cell infection through a single uninucleate plasmodium: in the sporogenic (spore-producing) phase, sporogenic plasmodia are located within infected plant tissue, either in tuber lesions or root, shoot or stolon galls. Following nuclear divisions within the plasmodium, thick-walled resting spores are produced, each being around 3.5–4.5 μm diameter (Jones 1978). These resting spores are aggregated together in sponge-like sporosori which vary in size from 19 to 85 μm. Falloon et al.

(2011) determined the mean numbers of resting spores in sporosori to be about 700. Resting spores may then persist in the soil, where they are able to survive in a dormant state in the absence of potato cultivation for many years, or on tubers. Under suitable conditions, the resting spores germinate and each releases a single biflagellate primary zoospore which can then infect host cells. The mechanisms of host infection are not well understood.

9.4.3 Symptoms

Spongospora subterranea can infect all underground organs of potato (i.e. stolons, tubers, and roots) where the pathogen stimulates the enlargement and division of host cells leading to the appearance of symptoms. Depending on environmental conditions initial tuber symptoms, which take 4–8 weeks to develop, are purplish brown lesions (1–2 mm diameter) that subsequently enlarge into raised mature lesions which burst, exposing large masses of sporosori resulting in characteristic symptoms of the disease (Figs. 9.11 and 9.12).

Sometimes infection of tuber buds can stimulate tubers to swell in that area, forming outgrowths or cankers which may also be infected through lenticels and be covered with scab lesions. Infection of roots or stolons can sometimes lead to development of galls (hyperplasia) (Fig. 9.13), where infected tissue is stimulated to grow and sporosori are formed inside the gall (Fig. 9.14). Galls burst when mature and release the sporosori into the soil. Root or stolon galls can be easily overlooked in the field and there is evidence that varieties with resistance to tuber infection can be susceptible to root or stolon gall production (Falloon 2008), resulting in an unseen build-up of inoculum in the soil.

Fig. 9.11 Characteristic lesions of powdery scab on cultivar Agria

Fig. 9.12 A typical powdery scab lesion

Fig. 9.13 Root galling of potato caused by *Spongospora subterranea*

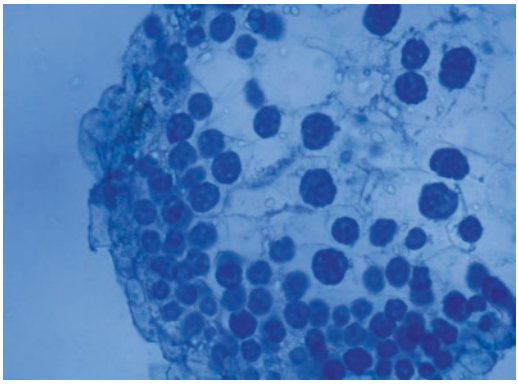

Fig. 9.14 Sporosori inside root gall

9.4.4 Impact

The main impact of powdery scab is cosmetic, due to a reduction in quality caused by lesions on the tuber surface, thus decreasing their value for either fresh or processing purposes. The disease also affects seed tuber certification, with the extent of the impact dependent on certification tolerances which vary from country to country (Falloon 2008; Wale 2000). Reports of losses due to unmarketable tubers can be as high as 50% in Australia (Hughes 1980) to 100% of the harvested product in Venezuela (Bittara et al. 2009). In addition, root infection by *S. subterranea* can reduce water absorption and nutrient intake (Shah et al. 2012). Some studies have reported a reduction in tuber yield and mean tuber weight due to disease caused by *S. subterranea* on either *Solanum tuberosum* spp. *andigena or S. tuberosum* spp. *tuberosum* (Gilchrist et al. 2011; Shah et al. 2012). In contrast, field studies using the cultivars Shepody and Umatilla Russet (*S. tuberosum* spp. *tuberosum*) showed no detrimental effect on either tuber yield or mean tuber weight due to the development of symptoms on roots and tubers (Johnson and Cummings 2015). *Spongospora subterranea* is also the vector of *Potato mop-top virus* (PMTV), one of the causes of spraing, a tuber blemish disease (Jones and Harrison 1969). PMTV is carried within zoospores and *S. subterranea* can remain viruliferous for many years. Little is known, however, about the virus–vector interactions, particularly with respect to the conditions conducive to transmission of the virus and the differential symptom expression of one or other of the diseases where infection by both organisms has occurred.

9.4.5 Pathogen Populations and Distribution

Since the first description of *Spongospora subterranea* under the name *Erysibe subterranea* by F. W. Wallroth in 1841, the pathogen has been reported in most potato-growing regions of the world, including hot and dry countries where farming is conducted at high altitudes or under irrigation. The number of first reports continues to increase across the world (Harrison et al. 1997; Merz and Falloon 2009; Wale 2000).

Few attempts have been made to characterize variation in *S. subterranea*, an unculturable biotroph with poorly understood genetics. It is difficult to obtain sufficient "clean" DNA for commonly used molecular marker techniques, sequence information is limited and assumptions about what constitutes an isolate or "strain" (sporosorus, resting spore or zoospore) are made. It is not demonstrated that sporosori occur as a result of sexual recombination, although this is thought to be the case (Braselton 1995). In general, analyses have been made on a single sporosori, which may also comprise many genotypes in the form of individual resting spores.

Genetic variation in Internal Transcribed Spacer sequences revealed limited differences among collections from Europe and Australasia and those from South

America (Bulman and Marshall 1998). Three ribotypes (I, II, and III) of *S. subterranea* were identified, with ribotype I, II and III is found in South America, ribotype II in North America and Australasia and ribotypes I and II in Great Britain (Osorio-Giraldo et al. 2012; Qu and Christ 2004). Most recently, Gau et al. (2013) applied SSR markers and ITS and actin sequence data to approximately 700 samples of *S. subterranea* obtained from 19 countries, different resting spore sources of the pathogen (root galls or tuber lesions) and from different potato host (sub) species. They described an overall low genetic diversity in *S. subterranea*, comprising three sample clusters; two occurring in South America (root galls and tuber lesions) and the third group comprising samples from elsewhere, independent of the resting spore source, and suggested that an ecological adaption in the native region due to coevolutionary processes and/or competitive exclusion may have taken place. South American populations were more diverse than those from other regions but no relationship between host species and pathogen diversity was noted.

9.4.6 Host Resistance

Resistance to powdery scab exists in some cultivars worldwide (Torres et al. 1995; Genet et al. 1996; Christ 1987; Falloon et al. 2003; Nitzan et al. 2008; Merz et al. 2012; Bittara et al. 2016). Falloon et al. (2003) found that although cultivars ranged from very susceptible to resistant, all developed zoosporangia and root galls and therefore none possessed immunity. Susceptibility of individual cultivars to root and tuber infection is not always closely correlated (Hughes 1980; Falloon et al. 2003; Bittara et al. 2016), particularly with regards to the relationship between root and tuber infection and root galling (Falloon et al. 2003; van de Graaf et al. 2007). The use of certain cultivars with high tuber resistance may therefore still maintain populations of *S. subterranea* in the soil.

Despite the availability of disease resistant cultivars, genetic resistance to *S. subterranea* currently plays a minor role in disease control as cultivars are usually selected by growers for characteristics other than their ability to resist powdery scab.

Resistance to powdery scab has also been demonstrated in other Solanum species, for example, *S. acaule* was reported to show resistance to pathogen infection in roots (Mäkäräinen et al. 1994) and among potato genotypes reported to have resistance to the formation of root galls at the Columbia Basin (WA) resistant clones were derived from the introgression of *S. bulbocastanum* and the resistant cultivar Summit Russet appeared more than once in their parental background (Nitzan et al. 2008). Although the mechanisms involved in resistance to disease caused by *S. subterranea* are poorly studied, evidence supports the hypothesis that root and tuber resistance to the disease is under control of multiple genes (Bittara et al. 2016; Falloon et al. 2003; Merz et al. 2012). In addition, disease resistance is inherited in an additive manner (Wastie et al. 1988) and is stable across environments (Bittara et al. 2016; Nitzan et al. 2010).

9.4.7 Management

Management of powdery scab is particularly difficult, and no single strategy currently controls powdery scab or root gall formation effectively, making an integrated approach essential (Falloon 2008). Disease avoidance using uncontaminated seed in uninfested soil represents the best method of disease prevention. The relative importance of soil inoculum level in causing disease on tubers was conclusively demonstrated by Brierley et al. (2013) who showed that when arbitrary soil inoculum threshold values of 0, <10 and >10 sporosori/g soil were set, it was observed that the number of crops developing powdery scab increased with the level of inoculum quantified in the field soil preplanting. In field trials carried out to investigate the link between the amount of inoculum added to the soil and disease development, disease incidence and severity on progeny tubers was found to be significantly ($P < 0.01$) greater in plots with increasing levels of inoculum. This information allows disease risk assessments to be made by taking soil inoculum concentration, in combination with other biotic and abiotic factors, into account.

The use of host resistance represents the most effective, sustainable and cost-effective approach for disease management; in addition, genetic resistance is especially suitable on pathogen populations with low genetic diversity (Gau et al. 2013). In a study performed across five European countries, no evidence of genotype x pathogen interaction was observed as disease development as was similar for all locations (Merz et al. 2012). Nevertheless, the use of host resistance is affected by factors defined by the consumer market (Harrison et al. 1997).

Resting spores produced by the pathogen are highly resistant to environmental stresses and can remain viable for >10 years (Merz and Falloon 2009), therefore up to 7 years of crop rotation or more that excludes potatoes might be required to reduce the risk of disease development once the pathogen has stablished in the field (Sparrow et al. 2015). Crop rotation may also have an influence on *S. subterranea* soil infestation as detectable inoculum was shown to be greater following a potato/wheat rotation compared to a potato/pea rotation (Shah et al. 2014). Additionally, a study by Qu and Christ (2006), found four of 16 crop and weed species infected with *S. subterranea* subsequently produced root galls, with sporosori detected on three host species (yellow mustard, oats and tomato). Similarly, Shah et al. (2010) confirmed the presence of sporosori in root galls formed on the solanaceous weeds hairy and black nightshade and subsequently demonstrated pathogenicity on potato with sporosorus inoculum derived from hairy nightshade. It is therefore possible for *S. subterranea* to complete its lifecycle in the absence of potatoes.

It is broadly accepted that high soil water content encourages zoospore release and that soil in which most pore spaces are filled with water facilitates movement of zoospores towards the host, and subsequent infection and disease development. This is supported by associations between seasons of high (or low) rainfall or irrigation treatments with high (or low) levels of powdery scab.

The chemical management of powdery scab is difficult and, in some cases, economically unsuitable. The use of seed and soil-applied chemicals to control powdery scab was previously reviewed in some detail by Merz and Falloon (2009).

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