Chapter 10
Bacterial Diseases of Potato

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Abstract Bacterial diseases are one of the most important biotic constraints of potato production, especially in tropical and subtropical regions, and in some warm temperate regions of the world. About seven bacterial diseases affect potato worldwide and cause severe damages especially on tubers, the economically most important part of the plant. Bacterial wilt and back leg are considered the most important diseases, whereas potato ring rot, pink eye, and common scab are the minor. Knowledge about zebra chip is extremely rare, as it occurs in a very isolated area and is an emerging disease in New Zealand, Europe, the USA and Mexico. Potato crop losses due to bacterial diseases could be direct and indirect; and they have several dimensions, some with short-term consequences such as yield loss and unmarketability of the produce and others with long-term consequences such as economic, environmental, and social. Some of them are of national and international importance and are the major constraints to clean seed potato production, with considerable indirect effects on trade. This review focuses on *Clavibacter* spp., *Ralstonia* spp., *Pectobacterium* spp., *Dickeya* spp., *Streptomyces* spp., and *Liberibacter* spp. pathogenic to potato, and looks at the respective pathogen in terms of their taxonomy and nomenclature, host range, geographical distribution, symptoms, epidemiology, pathogenicity and resistance, significance and economic losses, and management strategies. Nevertheless, the information collected here deal more with diseases known in developed and developing countries which cause severe economic losses on potato value chain.

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10.1 Brown Rot and Bacterial Wilt of Potato Caused by *Ralstonia solanacearum*

10.1.1 Taxonomy and Nomenclature

The genus *Ralstonia* is classified in the ß-Proteobacteria within the family Burkholderiaceae. The species complex of *Ralstonia solanacearum* has long been recognized as a group of phenotypically diverse strains, originally characterized as pathogenic races and biovars (Buddenhagen 1962; Hayward 1964). More recently, Fegan and Prior (2005) described four phylotypes in the species complex, each comprising multiple phylogenetic variants (sequevars) according to sequence diversity within barcoding genes (including 16S rRNA, *hrpB*, *mutS*, and *egl*). Recently, the complex has been reclassified on the basis of whole genome comparisons into three distinct species (Safni et al. 2014; Prior et al. 2016): *R. solanacearum* (Phylotype II), *R. pseudosolanacearum* (Phylotypes I and III), and *R. syzygii* (Phylotype IV) (Fig. 10.1).

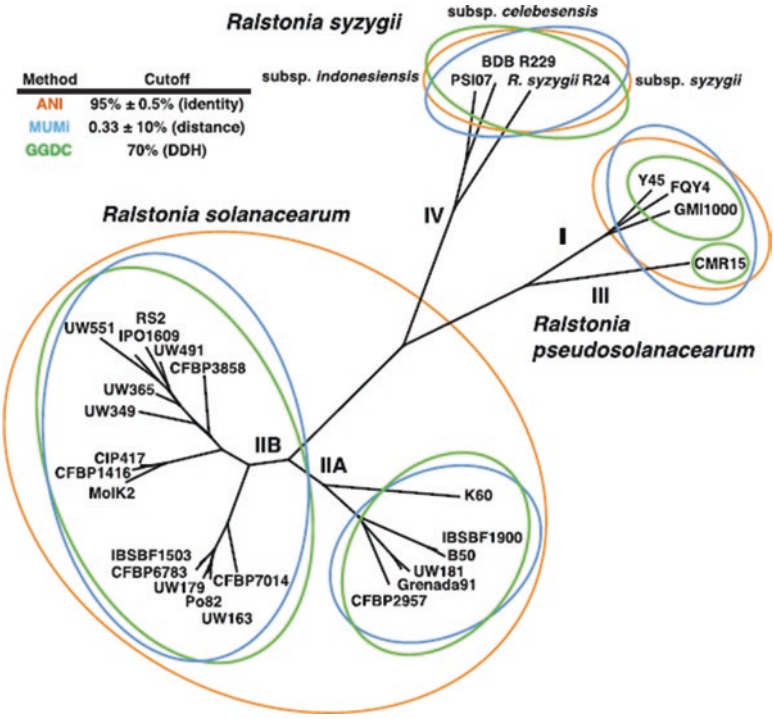

Fig. 10.1 Phylogenetic relatedness of strains within the *Ralstonia solanacearum* species complex, from Prior et al. (2016). Concatenated tree of distance matrices generated from average nucleotide identity (ANI), maximum unique matches index (MUMi), and genome-to-genome distance calculator (GGDC) to compare DNA:DNA homology (DDH)

10.1.2 Host Range

In addition to potato (*Solanum tuberosum* and *S. phureja*), the large range of economically important hosts includes banana and plantain, cucurbits, eggplant, *Eucalyptus,* ginger, groundnut, mulberry, tobacco, tomato, and many ornamental plants. *R. solanacearum*, *R. pseudosolanacearum*, and *R. syzygii* each comprise strains that were originally designated as race 1 and which occur in tropical areas all over the world, attacking a very wide host range of over 250 hosts in 54 botanical families. Some *R. solanacearum* genotypes within Phylotype II (sequevars IIA-6, IIA-24, IIA-41, IIA-53, IIB-3, IIB-4, and IIB-25), originally described as race 2, cause Moko disease of *Musa* spp. (banana and plantain) and *Heliconia*. *R. syzygii* comprises three subspecies: subsp. *syzygii* found on clove, subsp. *celebesensis* the cause of banana blood disease and subsp. *indonesiensis* found on solanaceaous crops (potato, tomato, and chilli pepper) as well as clove.

There are strains within each of the four phylotypes that can cause bacterial wilt and brown rot of potato; however, a single strain of *R. solanacearum* (sequevar 1) within Phylotype IIB (PIIB1), formerly known as race 3/biovar 2, is most widely associated with potato. This genotype has a lower *in planta* temperature optimum (27 °C) than most other genotypes (35 °C), often occurring in latent (symptomless) infections at high altitudes in the tropics and in subtropical and temperate potato-growing areas. This strain can also cause bacterial wilt of tomato and can survive in perennial nightshades, which act as secondary hosts. For example, the PIIB1 strain has overwintered in infected underground stolons of *Solanum dulcamara* (woody nightshade), growing along some European rivers, spreading to potato crops when the bacteria were transmitted in contaminated river water which was used for irrigation (Janse et al. 1998). The same strain has also been spread internationally on geranium cuttings produced in Africa and Central America (Williamson et al. 2002).

10.1.3 Geographical Distribution

Phylotype I strains are regarded to be of Asian origin, Phylotype II strains are thought to be of South American origin, whereas Phylotype III appears to have evolved in Africa and Phylotype IV in Indonesia. The *R. solanacearum* species complex is widely designated as a quarantine organism in many countries in an effort to prevent its movement across geographical borders. Nevertheless, the PIIB1 strain has spread from its origin to many potato-growing areas worldwide, presumably with movement in trade of infected seed tubers (Elphinstone 2005). However, this strain has never been reported on potato in the USA, despite it having been introduced on infected geraniums (Williamson et al. 2002) and findings of other Phylotype II strains on potato and other hosts in the southern states. The PIIB1 strain has in fact been designated as a select agent in the USA because of its perceived potential to pose a severe threat to agriculture. Moko disease-causing Phylotype II strains mainly

occur in South and Central America and the Carribean, but also appear to have spread to the Philippines where the same strains have been found on cooking banana (ABB and BBB genotypes), causing the so-called bugtok disease.

10.1.4 Symptoms

Wilting is a common symptom of infections of most hosts with all phylotypes. The youngest leaves usually wilt first, appearing at the warmest time of day. Wilting may be visible in only one stem, on one side of a plant or even sectoral in part of a leaf, depending where vascular infections are restricted in sectors of stems and leaf petioles. Leaves may become bronzed or chlorotic and epinasty may occur. Wilting of the whole plant may follow rapidly if environmental conditions are favorable for pathogen growth. As the disease develops, a brown discoloration of the xylem vessels in the stem may be observed above the soil line and adventitious roots may develop. A creamy, slimy mass of bacteria exudes from vascular bundles when the stem is cut. Wilting and collapse of whole plants can lead to rapid death.

Symptoms on infected potato tubers may or may not be visible, depending on the state of development of the disease in relation to the prevailing temperature (Fig. 10.2). Cutting a diseased tuber will reveal browning and necrosis of the vascular ring and in adjacent tissues. A creamy fluid exudate usually appears spontaneously from the vascular ring at the cut surface. Bacterial ooze can emerge from the eyes and stem-end attachment of whole tubers, to which soil adheres. If cut stem or tuber vascular tissue is placed in water, threads of bacterial ooze exude.

Fig. 10.2 Symptoms of potato brown rot with bacteria oozing from cut vascular tissues (**a**) and eyes (**b**) (UK Crown Copyright—Courtesy of Fera Science Ltd.), and wilted plant in the field (**c**) (Courtesy of International Potato Center)

10.1.5 Epidemiology

Although often described as soilborne pathogens, survival is usually short lived at low temperature in bare soil but is significant in alternative wild host plants (especially perennial nightshade species growing in waterlogged conditions or overwintering volunteers from susceptible crops). The bacteria have been shown to survive in a viable but nonculturable (VBNC) form under stress conditions in soil and water (Kong et al. 2014), but the epidemiological relevance of this is unclear. Disease is usually most severe at temperatures of 24–35 °C, although the PIIB1 strain is more cold tolerant than other strains. High soil moisture or periods of wet weather or rainy seasons are associated with high disease incidence. Entry into plants is usually through root injuries from where the bacteria move by colonization of the xylem where they adhere by polar attraction to the vessel walls, or invade the lumen. Blocking of the vessels by bacterial extracellular polysaccharide (EPS) is considered to be the major cause of wilting. The bacteria can also be transmitted mechanically during pruning operations or when cuttings are taken for propagation.

Long distance movement of vegetative propagating material (e.g. seed potatoes, rhizomes of ginger and turmeric, and banana suckers) can carry latent infections. Natural infection of true seed has only been established for groundnut in Indonesia and China. There have been findings of contaminated seed of other hosts (including tomato, Capsicum, eggplant, and soyabean) although seed infection and transmission has not been substantiated. At present, transmission through water or soil and movement of infected vegetative plant parts are considered to be more important for most host plants than transmission via true seed. In contrast, some strains of *R. solanacearum* and *R. syzygii*, which cause Moko disease and blood disease of banana and Sumatra disease of clove are transmitted by insects (including pollinating flies, bees, wasps, and thrips on banana and xylem-feeding spittlebugs of *Hindola* spp. on clove) with potential for rapid spread over several kilometers.

10.1.6 Pathogenicity Determinants and Resistance

Factors determining pathogenicity, virulence, and avirulence in the bacteria have been recently reviewed (Genin and Denny 2012; Meng 2013). After invasion into root intercellular spaces, expression of *hrpB* is induced, in response to plant signals, activating other *hrp* (hypersensitive response and pathogenicity) genes in construction (*hrpK* and *hrpY*) and regulation (*hrpB* and *hrpG*) of a type 3 secretion system (T3SS), a molecular syringe which is essential for pathogenicity. The bacteria proliferate in intercellular spaces with the aid of a variety of effector proteins, secreted through the T3SS, which suppress plant defenses by interfering with host signal pathways. *HrpB* positively regulates expression not only of *hrp* genes but also of genes encoding a number of plant cell wall-degrading exoproteins secreted through a type II secretion system (T2SS). These include polygalacturonases (PehA, PehB,

and PehC), endoglucanase (egl), pectin methylesterase (Pme), and cellobiohydrolase (cbhA), which contribute not only to invasion of xylem vessels, leading to systemic infection, but also to quantitative control of virulence. When cell densities reach a threshold, *PhcA* is activated by build-up of a volatile quorum sensing signal, 3-hydroxy palmitic acid methyl ester (3-OH PAME), inducing genes (*epsABCDEF* & *P*) controlling biosynthesis of exopolysaccharide (EPS), a major virulence factor. Transcriptome analysis (Jacobs et al. 2012) has also confirmed expression of these and other genes encoding various virulence traits at high cell densities during host infection, including genes imparting stress tolerance (*bcp*, *acrA*, *acrB*, and *dps*) and motility and attachment structures (*pilA* and *fliC*).

Available field resistance to the *R. solanacearum* species complex is limited and tends to be unstable under different environmental conditions and/or strain variability. Traditional breeding has not yet yielded new resistant varieties because of the difficulty in transferring multiple unknown genes from wild germplasm with polygenic resistance into cultivars without cotransferring undesirable linked traits. Furthermore, high-level resistance to host colonization as well as to disease development is needed to avoid the risk of spreading the pathogens in symptomless latent infections. Most studies on the genetic basis of resistance to bacterial wilt have been conducted in the model plants *Arabidopsis thaliana* and *Medicago truncatula* (Huet 2014). Quantitative trait loci (QTL) have been identified that include R genes that encode proteins that recognize bacterial effector avirulence (AVR) proteins, triggering resistance to the bacterium. Transfer of selected R genes from *A. thaliana* into tomato conferred immunity to the *Ralstonia* strain with the corresponding AVR gene (Narusaka et al. 2013). Both broad-spectrum and strain-specific quantitative trait loci (QTLs) have been identified in tomato (Wang et al. 2013), tobacco (Qian et al. 2013) and eggplant (Salgon et al. 2017). Discovery of possible resistance/avirulence (R/Avr) gene for gene resistance mechanisms is particularly interesting since known bacterial effectors can be used to screen for homologous resistance genes in related crops, including potato.

10.1.7 Significance and Economic Loss

Recently ranked by international phytobacteriologists as the second most important of all plant pathogenic bacteria after *Pseudomonas syringae* (Mansfield et al. 2012), the plant pathogenic *Ralstonia* spp. have an extremely wide geographic distribution and host range. On potato alone, it is thought to be responsible for approximately US$1 billion in losses each year, affecting some 3 m farm families over 1.5 m ha in around 80 countries (Elphinstone 2005). Moko disease has affected banana and plantain over thousands of square miles in Central and South America, particularly affecting small susbsistence farmers. In many countries in which the organism has quarantine status, important losses occur as a result of regulatory eradication measures and restrictions introduced on further production on contaminated land.

10.1.8 Management

Disease management remains limited and is hampered by the ability of the pathogens to survive in wet environments on plant debris or in asymptomatic weed hosts, which act as inoculum reservoirs. In the absence of any curative chemical control methods, prevention of bacterial wilt largely relies on the availability of pathogen-free planting material and effective surveillance and monitoring to protect areas free from the bacteria. For potato, effective disease management has mainly resulted from the use of limited generation seed multiplication from pathogen-free nuclear stocks with zero tolerances for the disease in official seed certification programs. Regular post-harvest testing of seed potato tubers is usually also necessary to avoid distribution of latent infections. Similarly, for other vegetatively propagated crops, there is a need to ensure planting material has been tested free of infection and that there are restrictions on the movement of planting material from affected to disease-free areas. Disinfection of pruning and harvesting tools is important in preventing spread of disease e.g. in banana and plantain production. In areas where the pathogen could be spread in contaminated irrigation water, prohibition of irrigation with surface water has been an effective control measure. For hydroponic glasshouse production systems, disinfection of recirculating water (e.g. using chlorine dioxide) can prevent spread of the bacterium. This effectively halted international spread of *R. solanacearum* PIIB1 in geranium cuttings produced in Central America and East Africa following export to the USA and Europe.

10.2 Bacterial Blackleg and Tuber Soft Rot Symptoms Caused by *Pectobacterium*

10.2.1 Taxonomy and Nomenclature

The genera *Pectobacterium* is a member of the ß-Proteobacteria in the family *Pectobacteriaceae* within the order *Enterobacterales*. The *Pectobacteriaceae* family also contains the genera *Brenneria*, *Dickeya*, *Lonsdalea*, and *Sodalis* (Adeolu et al. 2016). *Pectobacterium* originally belonged to the genus *Erwinia* (Winslow et al. 1917) with the name *Pectobacterium* being proposed by Waldee (1942). However, the name *Erwinia* persisted until Hauben et al. (1998), using 16S rDNA analysis, re-proposed the name *Pectobacterium*, which has been used since. There are currently 12 species of *Pectobacterium* including *P. aroidearum*, *P. atrosepticum*, *P. betavasculorum*, *P. brasiliense*, *P. cacticida*, *P. carotovorum*, *P. odoriferum*, *P. parmentieri*, *P. peruviense*, *P. polaris, P. punjabense,* and *P. wasabiae* (Dees et al. 2017a, b; Khayi et al. 2016; Nabhan et al. 2013; Sarfraz et al. 2018; Waleron et al. 2018; Zhang et al. 2016a, b).

10.2.2 Host Range

Pectobacterium species have a wide range of host plants with approximately a third of these overlapping with hosts for *Dickeya* species (Charkowski 2018; Ma et al. 2007a, b). For *Pectobacterium* species, hosts have been identified in at least 20 dicot families in 13 orders and 12 monocot families in 6 orders, often with only a single isolate being associated with a particular family or order. This may be due to lack of reporting rather than a clear difference in the abilities of these two genera to infect (Charkowski 2018; Ma et al. 2007a, b). However, some important specialization may exist since *Pectobacterium* appears to be found more frequently than *Dickeya* on cabbage, cotton, and mango, and *Dickeya* but not *Pectobacterium* on rice and maize. Some species such as *P. atrosepticum*, *P. betavasculariarum*, and *P. parmentieri* appear to have a very narrow host range, and *P. aroidearum* appears to be more virulent on monocots than other *Pectobacterium* species (Nabhan et al. 2013). The *Pectobacterium* species most commonly found on potato include *P. atrosepticum*, *P. brasiliense*, *P. carotovorum*, *P. odoriferum*, *P. parmentieri*, *P. peruviense*, *P. polaris,* and *P. punjabense.*

10.2.3 Geographical Distribution

Pectobacterium species are found on all continents where potato is grown, and are likely to be present as saprophytes in the soil, water, and are also regular inhabitants of plant roots when not causing disease. While there are likely to be some regional differences in the species distribution, some appear to be ubiquitous. For example, *P. atrosepticum, P. brasiliense, P. parmentieri*, and *P. carotovorum* are found on multiple continents (De Boer et al. 2012; Duarte et al. 2004; Kim et al. 2009; Ngadze et al. 2012; Pérombelon and Kelman 1987; Pitman et al. 2008, 2010; She et al. 2017; van der Merwe et al. 2010; van der Wolf et al. 2017; Wang et al. 2017a, b, c).

In Europe, *P. atrosepticum* has been the predominant species responsible for blackleg disease on potato, with *P. carotovorum* responsible a minority of blackleg disease incidents but often associated with soft rot in storage. Recently, at least some of these *P. carotovorum* strains were been reclassified as *P. wasabiae* and subsequently as *P. parmentieri* (Khayi et al. 2016; Nykyri et al. 2012). *P. brasiliense*, which was originally identified as causing disease on potato in Brazil (Duarte et al. 2004), has been common in the United States since at least 2001, as has *P. parmentieri* (Kim et al. 2009; Yap et al. 2004). *P. brasiliense* was not known to cause disease on potato in Europe prior to 2012–2013 but has since increased greatly in its incidence in many European countries (de Werra et al. 2015) and is now recognized as an important pathogen in Africa as well (van der Merwe et al. 2010).

10.2.4 Symptoms

Pectobacterium causes blackleg, which is a stem necrosis that originates from the planted seed tuber (Pérombelon 2002) (Fig. 10.3). Necrotic symptoms often extend several centimeters up the stem and necrotic vascular tissue is typically present inside the stem several centimeters beyond where general stem necrosis occurs. The pith of the stem is often decayed. Plant leaves may turn bright yellow and the plant will eventually wilt and die. Infected plants produce few or no tubers. *Pectobacterium* can enter daughter tubers through the xylem or through wounds caused by insects, frost damage, or harvest equipment. Once inside a tuber, it will decay the inside of the tuber, but not the tuber periderm, causing bacterial soft rot. The bacteria will also decay stems damaged by cultivation equipment or severe weather, causing aerial stem rot. In all cases, it is common to find multiple *Pectobacterium* species or *Pectobacterium* and *Dickeya* together when blackleg, aerial stem rot, or soft rot symptoms are present (Kim et al. 2009; Yap et al. 2004). In the United States, *P. partmentieri* is often found with other decay pathogens, such as *Clavibacter michiganensis* and potato rot nematode.

10.2.5 Epidemiology

The most common *Pectobacterium* strains in a region change from year to year and the strains and species present are also not consistent across a particular continent (Dees et al. 2017a, b). The species also differ in optimal and upper limits of growth temperatures. For example, *P. atrosepticum* and *P. parmentieri* die above 33 °C, but *P. carotovorum* and *P. brasiliense* can grow at temperatures up to 39 °C. Initial seed potato production relies on pathogen-free micropropagated plantlets. These plantlets are grown in greenhouses or screen houses to produce minitubers, which are used for field planting. *Pectobacterium* is sometimes found in or on minitubers, but it is more common on potato ones the tubers have been grown in the field. Each generation of potato multiplication tends to increase *Pectobacterium* incidence on potato tubers. Since *Pectobacterium* is common in the environment and can be found in soil, water, weeds, and insects, it is not feasible to produce potatoes free of this pathogen (Charkowski 2015). The bacteria may also be spread by insects (Kloepper and Schroth 1981), but the importance of insects compared to other routes of spread remains unknown. *Pectobacterium* appears to spread mainly at harvest. Bacterial numbers increase dramatically on senescing vines and the bacteria will contaminate harvest equipment and may become aerosolized during harvest. In tubers, the bacteria are found in lenticels and inside the stolon scare. Asymptomatic infestations are common, so it is not possible to visually assess seed potato lots for risk. Blackleg development is highly dependent on the environment and it is unpredictable, even when a seed lot is known to be contaminated with *Pectobacterium*. Detection protocols useful for studying *Pectobacterium* epidemiology were recently complied (Humphris et al. 2015).

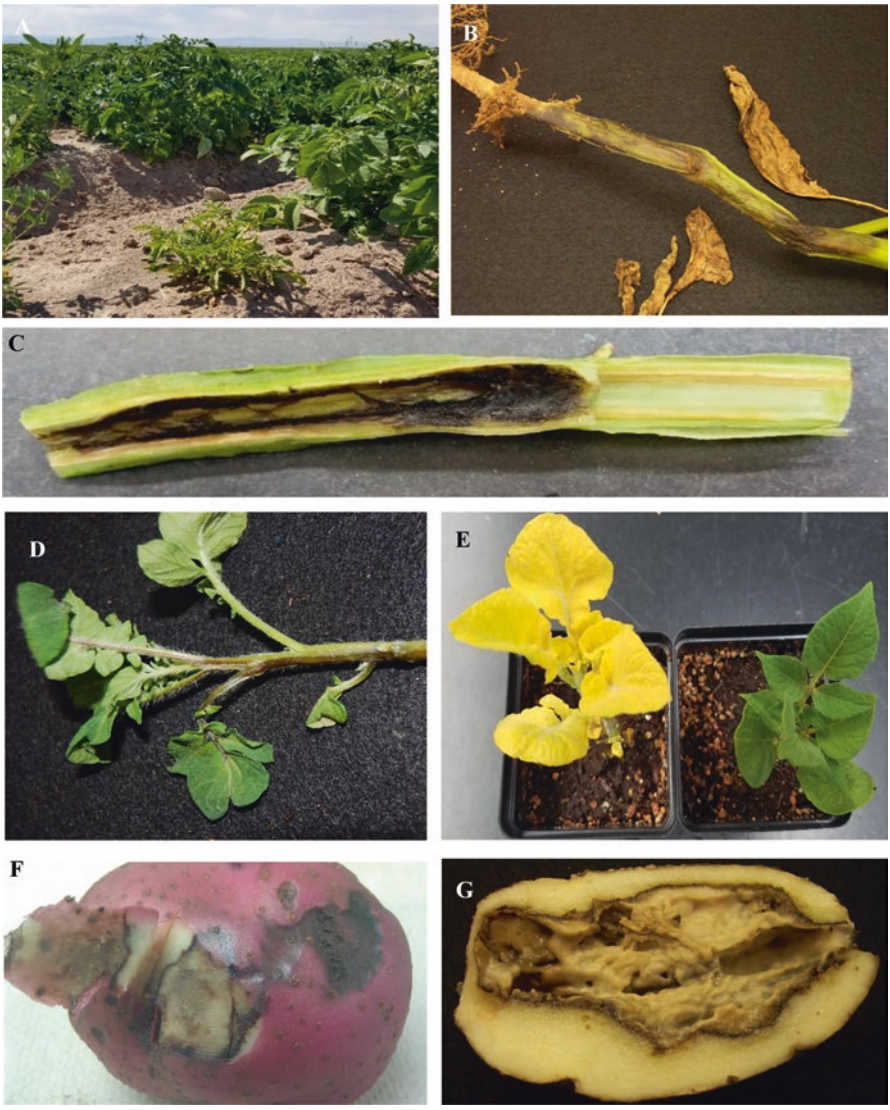

Fig. 10.3 Bacterial blackleg and tuber soft rot symptoms caused by *Pectobacterium* on potato. (**a**) Plants with blackleg are shorter and have curled leaves (A), the stem is blackened on the outside (**b**), the pith inside is decayed and the xylem are brown (**c**). Brown or black decay may spread into leaves (**d**) or leaves may turn bright yellow (**e**). Tubers may have swollen lenticels and sunken lesions (**f**). The soft rot bacteria may enter the tuber through the stolen and decay the center of the tuber (**g**) (Courtesy of Amy O. Charkowski, Colorado State University)

10.2.6 Pathogenicity Determinants and Resistance

Pectobacterium pathogenicity depends upon secreted plant cell wall-degrading enzymes, although several other factors also contribute to virulence (Charkowski et al. 2012). The genetic basis for the observed host range limitation in some *Pectobacterium* species and differences in ability to grow at temperatures above 33 °C remain unknown. There are no examples of gene-for-gene resistance with this necrotrophic pathogen. The antimicrobial peptide Snakin-1 enhances resistance when overexpressed in potato 329–338 and some wild potato species exhibit resistance to *Pectobacterium* (Rietman et al. 2014), but the basis for resistance in wild potato species is poorly understood. There are no resistant commercial potato varieties, but varieties differ in tolerance. There is a large environmental component to disease development for blackleg and little effort has been made to correlate laboratory assays for tolerance with results observed on grower farms. The recent identification of numerous new *Pectobacterium* species suggests that additional novel and potentially high virulent species remain to be discovered and also that this high level of diversity will hinder development of tolerant potato varieties.

10.2.7 Significance and Economic Loss

Pectobacterium has served as a model pathogen for phytobacteriology research for longer than almost any other bacterial pathogen, except *Erwinia amylovora*, and *Pectobacterium* research has resulted in some notable firsts, such as the first demonstration of the role of quorum sensing in bacterial pathogenicity (Pirhonen et al. 1993). It remains an economically significant disease worldwide. Farmers lose millions annually to blackleg, aerial stem rot, and tuber soft rot. Of these, tuber soft rot can be particularly devastating since it occurs after the farmer has invested a full season of inputs into growing the crop.

10.2.8 Management

Pectobacterium management relies mainly on cultural practices (Charkowski 2015; Czajkowski et al. 2011). Growers initiate potato production with micropropagated plantlets that are free of *Pectobacterium*, but tubers are quickly contaminated once they are planted in fields. To reduce the risk of disease at planting, growers should fully suberize seed if they are using cut seed and they should not plant cold seed into wet ground. During the growing season, they should irrigate with ground water if possible and not overfertilize with nitrogen. Rouging infected plants is likely to spread the

disease, so this is not recommended. At harvest, the bacteria will multiply on the vines as they senesce, so quickly killing potato vines may aid in reducing disease incidence the following year. Tubers should be allowed to heal before cooling storages. Good airflow and high humidity in potato warehouses will also aid in reducing soft rot in storage. High levels of carbon dioxide in warehouses will promote soft rot development.

10.3 Blackleg and Soft Rot of Potato Caused by *Dickeya*

10.3.1 Taxonomy and Nomenclature

The genus *Dickeya* is a member of the ß-Proteobacteria in the family *Pectobacteriaceae* within the order *Enterobacterales*. The *Pectobacteriaceae* family also contains the genera *Brenneria*, *Lonsdalea Pectobacterium*, and *Sodalis* (Adeolu et al. 2016). Members of the *Dickeya* genus originally belonged to the genus *Erwinia* represented by strains within species *E. chrysanthemi* (Burkholder et al. 1953). Later this species was reclassified as *Pectobacterium chrysanthemi* (Hauben et al. 1998), until Samson et al. (2005) elevated the species to the genus *Dickeya* with six species. There have since been some changes and additions to these species, which currently include *D. aquatica*, *D. chrysanthemi*, *D. dadantii*, *D. dianthicola*, *D. fangzhongdai*, *D. paradisiaca*, *D. solani*, and *D. zeae* (Brady et al. 2012; Parkinson et al. 2014; Samson et al. 2005; Tian et al. 2016).

10.3.2 Host Range

Dickeya has a broad host range and can infect plant species in at least 12 dicot families in 10 orders and 10 monocot families in 5 orders, and include ornamentals such as chrysanthemum, carnation, dahlia, and calla lily as well as important crops including carrot, tomato, and, the most economically important, potato (Charkowski 2018; Ma et al. 2007a, b; Samson et al. 2005). While all *Dickeya* species, with the exception of *D. paradisiaca*, have been found on ornamentals in Europe, only *D. dianthicola* and *D. solani* have caused significant economic losses on potato (Toth et al. 2011). In both cases, the lack of genetic diversity between isolates on potato and ornamental hosts suggests the organisms may have spread to potato from such a host (Parkinson et al. 2009; Slawiak et al. 2009). Only *D. aquatica*, which was isolated from waterways in the UK (Parkinson et al. 2014) and Maine (J. Hao, personal communication), has not yet been associated with a plant disease.

10.3.3 Geographical Distribution

As with *Pectobacterium*, *Dickeya* species have been reported on a wide range of hosts in different countries around the world (Samson et al. 2005). While *D. zeae, D. solani,* and *D. dianthicola* have wide geographic distributions, *D. paradisiaca* appears to be restricted to Colombia (Samson et al. 2005; Toth et al. 2011).

D. dianthicola was the first *Dickeya* species to be associated with plant disease in Europe, occurring on *Dianthus* in the Netherlands, Denmark, and the UK and later spreading to other nations (Hellmers 1958). It was later associated with other ornamentals and crops in a number of European countries, including potato. In some cases, *D. dianthicola* replaced *P. atrosepticum* as the dominant blackleg pathogen (Parkinson et al. 2009; Toth et al. 2011). *D. solani* was recognized independently as a new *Dickeya* pathogen on potato by several groups from 2004 through 2010 (Laurila et al. 2008; Parkinson et al. 2009; Slawiak et al. 2009). Isolates of both *D. dianthicola* and *D. solani* show little genetic diversity compared to isolates from ornamentals, and within these species there is a high degree of genetic similarity. Therefore, it seems likely that these pathogens have independently jumped host from an ornamental onto potato (Toth et al. 2011).

10.3.4 Symptoms

Although *Dickeya* can cause tuber soft rot, it primarily causes blackleg on potato. Blackleg symptoms include necrosis of the potato stem, originating from the mother tuber and spreading several centimeters above ground (Fig. 10.4). Plant leaves will wilt and curl as the disease develops and the plant vascular system will become necrotic. The pith of the stem is often decayed. *D. dianthicola* can also cause severe seed decay and lack of plant emergence in severe cases. Infected plants produce few or no tubers and any tubers produced may decay prior to harvest. Both *Dickeya* and *Pectobacterium* may be present together in diseased plants. In the United States, *P. parmentieri* is the most common species found together with *Dickeya*.

10.3.5 Epidemiology

Initial seed potato production relies on pathogen-free micropropagated plantlets. These plantlets are grown in greenhouses or screenhouses to produce minitubers, which are used for field planting (Frost et al. 2013). *Dickeya* will kill micropropagated plants within a few days and is not typically found in greenhouses or screenhouses. It appears to contaminate potatoes after they have been grown for at least one generation in the field, with the risk of contamination increasing with each

Fig. 10.4 Foliar symptoms of *Dickeya dianthicola* on potato. Initial symptoms are either a lack of emergence or leaf curling (**a**). The base of the stem turns dark brown or black and this necrosis can extend several centimeters from the soil line (**b**). The pith inside symptomatic stems is often decayed and the xylem are necrotic for several centimeters above the external stem necrosis and the pith decay (**c**). Disease symptoms may only develop on one stem of a multi-stem plant (**d**) (Courtesy of Amy O. Charkowski, Colorado State University)

generation in the field. *Dickeya* does not appear to survive in soil, but it can contaminate waterways and survive for long periods in surface water (Toth et al. 2011). It may also survive in weeds (Fikowicz-Krosko and Czajkowski 2017) or volunteer potatoes and spread by insects (Rossmann et al. 2018). Like *Pectobacterium*, *Dickeya* appears to spread mainly at harvest, where it can spread from infected vines and tubers to previously uncontaminated tubers. The bacteria are mainly found on tuber lenticels, but may also be present in the tuber stolon scar. Asymptomatic infestations are common, so it is not possible to visually assess seed potato lots for risk.

Blackleg development is highly dependent on the environment and it is unpredictable, even when a seed lot is known to be contaminated with *Dickeya*. Plants grown from infested seed lots planted in warm, humid areas tend to develop disease, while plants grown from the same infested seed lot planted in cooler, drier climates may remain healthy. Temperatures above 30 °C during the growing season appear to be particularly conducive to disease development. Co-contamination with *Pectobacterium* and *Dickeya* appears to lead to disease development more frequently than when only *Dickeya* is present.

10.3.6 Pathogenicity Determinants and Resistance

Dickeya pathogenicity relies mainly on pectate lyases and other plant cell wall-degrading enzymes secreted by the bacterial cell, although several other virulence genes are known (Charkowski et al. 2012). Although both *Pectobacterium* and *Dickeya* use plant cell wall-degrading enzymes, there are some important differences in enzyme genes and gene regulation between the genera that may account for some of the differences in disease symptoms. There are no examples of gene-for-gene resistance with *Dickeya* and the basis for resistance to *Dickeya* in wild potato species or for host range is poorly understood. There are no resistant commercial potato varieties, but varieties do differ in tolerance.

10.3.7 Significance and Economic Loss

The relative importance of *Dickeya* as a potato pathogen appears to be increasing (Toth et al. 2011). *D. solani* caused severe losses in the early 2000s in multiple countries and in 2015 *D. dianthicola* was in up to 20% of seed potato lots in some states in the US. Recent development of species-specific PCR assays for *Dickeya* will likely reveal that it is widespread in potato. As with *Pectobacterium*, farmers lose millions annually to blackleg caused by *Dickeya*.

10.3.8 Management

Cultural practices are important for *Dickeya* management and the recommendations are essentially the same as for *Pectobacterium* (Czajkowski et al. 2011, 2013). Growers initiate potato production with micropropagated plantlets that are free of *Dickeya*, but tubers may become contaminated once they are planted in fields. To reduce the risk of disease spread, growers should sanitize equipment thoroughly between seed fields, especially if blackleg is present. At planting, growers should fully suberize seed if they are using cut seed, and they should not plant seed that is too cold or into saturated ground. During the growing season, they should irrigate with ground water if possible and not overfertilize with nitrogen. Rouging infected plants is likely to spread the pathogen if diseased plants are present. At harvest, the *Dickeya* may multiply on the vines as they senesce, so quickly killing potato vines may aid in reducing disease incidence the following year. Tubers should be allowed to heal before cooling storages. Good airflow and high humidity in potato warehouses will also aid in reducing soft rot in storage. High levels of carbon dioxide in warehouses will promote soft rot development. Seed potatoes may be tested for *Dickeya* prior to planting (Czajkowski et al. 2015; Humphris et al. 2015) and

growers should avoid planting contaminated seed lots in areas where growing conditions are conducive to blackleg.

10.4 Potato Ring Rot Caused by *Clavibacter michiganensis* Subsp. *sepedonicus*

10.4.1 Taxonomy and Nomenclature

Clavibacter michiganensis subsp. *sepedonicus* is a Gram positive, coryneform, aerobic, non-spore-forming bacterium in the Microbacteriaceae family of the Actinobacteria. *C. michiganensis* is the only species currently recognized within the genus; all six of its subspecies (subspp. *Insidiosus*, *michiganensis*, *nebraskensis*, *phaseoli*, *sepedonicus*, and *tesselarius*) are plant pathogens. *C. michiganensis* subsp. *sepedonicus* (*Cms*) was formerly known under the synonyms *Corynebacterium sepedonicum*, *Corynebacterium michiganense* pv. *sepedonicum*, and *Corynebacterium michiganense* subsp. *sepedonicum*.

10.4.2 Host Range

The only economically important host is potato (*Solanum tuberosum*), although natural infection was recently reported for the first time on tomato (van Vaerenbergh et al. 2016). Many members of the Solanaceae, including tomato and eggplant, are susceptible after artifical inoculation. Some solanaceous weeds, e.g. hairy nightshade (*Solanum sarrachoides*) and buffalobur (*S. rostratum*), may harbor the bacterium following potato crops with ring rot (van der Wolf et al. 2005a).

10.4.3 Geographical Distribution

First reported after an outbreak in Germany in 1905 (Appel 1906), it is one of the few major plant pathogens that is not present in the area where the crop evolved, i.e. Andean South America. In North America it was first reported in Quebec (Canada) in 1931 and by 1940 it had spread to all important potato-producing districts in Canada and the USA due to movement in trade of infected seed potato tubers. Subsequent zero tolerances imposed in quarantine and seed certification controls in most potato-growing areas have effectively limited the numbers of findings, although total eradication is difficult. Currently, it tends to occur sporadically in cool, northern latitudes of North America (Northern USA and Canada) with only a single report in Mexico. Strict regulation in Europe has also reduced findings in

annual surveys, especially in certified seed crops, with only occasional recent findings in some countries (Bulgaria, Czech Republic, Estonia, Finland, Germany, Greece, Hungary, Latvia, Lithuania, Netherlands, Norway, Slovakia, Sweden, and Turkey). Isolated former outbreaks have been declared eradicated in Austria, Belgium, Cyprus, Denmark, France, Spain, and UK (England and Wales). However, it remains prevalent in areas where formal seed certification is absent (parts of northern, western, and central Russia, Ukraine, Poland, and Romania). It is also reported in Asia (several provinces of China, Japan, Kazakhstan, Korea, Nepal, Pakistan, and Uzbekistan) although its distribution is not clearly defined. Ring rot has never been confirmed in Africa, Australasia, or South America.

In the field, foliar symptoms are not always observed or may occur only at the end of the season when they are difficult to distinguish in the senescing plant and are easily missed during crop inspections. Unlike bacterial wilt, caused by *Ralstonia,* wilting due to the ring rot bacterium is usually slow and initially limited to the leaf margins (Fig. 10.5). Young infected leaves expand more slowly in the infected zones and become distorted. Leaves affected by xylem blockages further down the stem often develop chlorotic, yellow to orange, interveinal areas. Infected leaflets, leaves, and even stems may eventually die. Leaves and tubers may simply be reduced in size and occasionally whole plants can be stunted.

10.4.4 Epidemiology

Factors affecting development and spread of potato ring rot were reviewed in detail by van der Wolf et al. (2005b). Seed potato tubers infected or contaminated with *Cms* are the primary source of infection. Inadvertent dissemination of the bacterium to new places of production occurs with the movement and planting of latently infected seed tubers. The bacterium also spreads from infected tubers through direct contact and via contaminated surfaces of equipment used in potato production, such as seed cutters, planters, harvesters, graders, and transport vehicles as well as in contaminated stores and containers. Plant-to-plant spread in the field is usually low but there is some experimental evidence that insects can transmit the disease (Christie et al. 1991) although the full significance of this is not understood. *Cms* survives for extended periods of many months to years in a dry and cool environment. Its persistence on farm equipment, in stores, and on transport vehicles is an important means by which the bacterium is maintained and spread within farm units and disseminated to other production units.

The bacteria migrate systemically from seed tubers to the stems via the vascular tissue, and subsequently into progeny tubers through the stolons. The pathogen population density increases during the growing season but sometimes can be detected in stems within 3–4 weeks after planting infected seed. Survival of *Cms* in soil is not thought to contribute greatly to ring rot epidemiology although it can overwinter in the field in volunteer tubers (groundkeepers) and in potato tissue

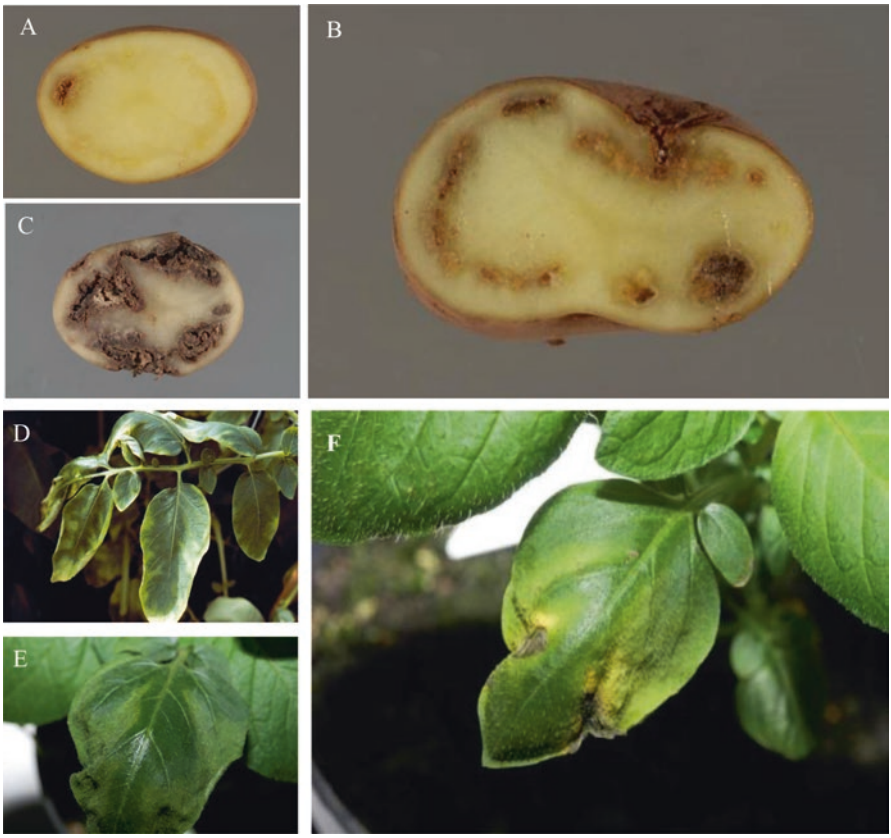

Fig. 10.5. Symptoms of potato ring rot: initial tuber symptoms (water-soaked vascular ring, bacterial exudate, and start of vascular necrosis) (**a**), necrosis around vascular ring (**b**), advanced necrosis and secondary rotting (**c**), interveinal chlorosis and wilting/epinasty at leaf margin (**d**, **e**), leaf distortion (**f**). (UK Crown Copyright—Courtesy of Fera Science Ltd.)

debris. Survival is longest in cold dry conditions. The bacterium survives particularly well when dried in smears of decayed tuber tissue on equipment, machinery, potato sacks, and storage containers and can remain infectious in the dried state for at least 18 months at temperatures from 5 to −40 °C. *Cms* has been reported to be associated with solanaceous weeds, but any role of these potential inoculum sources in the epidemiology of the ring rot disease of potato is unclear. *Cms* has a low optimum growth temperature (21–23 °C) and is confined mainly to cooler potato-growing regions.

10.4.5 Pathogenicity Determinants and Resistance

The genome of *Cms* was first sequenced by Bentley et al. (2008). In addition to the 3.26 Mb chromosome, which is highly similar amongst all *Clavibacter* subspecies, a 50-kb circular plasmid (pCS1) and a 90-kb linear plasmid (pCSL1) are carried by all *Cms* strains. The plasmid pCS1 is essential for symptom development, but genes required for host recognition, efficient colonization, infection, and evasion or suppression of plant defense are located on the chromosome (Eichenlaub and Gartemann 2011). Two proteins, CelA and Chp-7, have been shown to be required for full virulence (Laine et al. 2000; Nissinen et al. 2001). The gene *celA* is located on the plasmid pCS1 and encodes the cellulase endo-β-1,4-glucanase. Located on the chromosome, c*hp-7* encodes a serine protease effector that directly elicits a hypersensitive response in nonhost tobacco plants (Lu et al. 2015). Gene expression studies in *Cms* cells growing in either potato tissue or rich media (Holtsmark et al. 2008) have identified other putative virulence genes. In addition to *celA*, a homologous gene *celB*, two serine proteases and a xylanase was also upregulated in the plant tissue. Three other serine protease genes, including the *chp-7*, were downregulated. Unlike Gram-negative bacterial pathogens of potato, the gram-positive *Clavibacter* pathogens do not have a type 3 secretion system (T3SS) to translocate effectors into host plant cells.

Eichenlaub and Gartemann (2011) have described the likely infection process by *Clavibacter*. During infection the bacteria enter the xylem vessels of host plants, and subsequently spread systemically to colonize the whole plant. *Clavibacter* can be considered as a biotrophic phytopathogen that recruits nutrients (carboxylic acids and sugars) from the xylem fluid. Following growth and colonization, the cell walls of the xylem vessels and surrounding parenchymatic cells are then hydolyzed by expression of cellulases and other extracellular enzymes, potentially including polygalacturonase, pectic lyase, xylanases, and other endoglucanases, leading to symptom development.

There are no currently available potato cultivars with immunity or useful resistance to ring rot. The concept of cultivar tolerance to ring rot is not yet understood and little is known of the status of most commonly grown cultivars with respect to their susceptibility to infection and colonization under varying environmental conditions. Although potato cultivars vary in their propensities to express ring rot symptoms, less variation between cultivars in their susceptibilities to latent infections is observed (De Boer and McCann 1990). Since tolerant cultivars, which tend not to develop symptoms, can act as symptomless carriers of *Cms*, they have been removed from seed certification schemes in North America (Manzer and McKenzie 1988). Laurila et al. (2003) demonstrated that an accession (PI472655) of the wild potato species *Solanum acaule* was susceptible to latent infection by *Cms* at 15 °C but appeared immune to infection at 25 °C.

10.4.6 Significance and Economic Loss

Direct losses due to wilting and tuber rotting in field and store are usually moderate, especially where modern seed certification systems are in place. Nevertheless, ring rot constitutes a constraint on seed potato production, with considerable indirect effects on trade. These result from statutory measures taken against ring rot outbreaks, which include loss of certification, restrictions on further cropping, purchase of new seed stocks, costs of disinfection, and disposal of infected and associated crops and subsequent effects on reputation and export trade.

10.4.7 Management

In the absence of effective chemical or biological control measures, or potato cultivars with adequate levels of resistance, management of potato ring rot must rely on the production and safe distribution of seed potatoes that are free from infection. Control is achieved primarily through strict application of quarantine and seed certification regulations, which involve a zero tolerance for the disease during seed and import inspections and for the pathogen during regular testing of consignments. By laboratory testing for latent infections, infected lots can be detected early and eliminated from seed programs before further spread of the pathogen occurs. Phytosanitary measures must be aimed at the entire potato production system on account of the insidious nature of the disease. Seed potatoes should be imported only from countries which can show, by regular surveys and tests, that they operate a seed-potato production and distribution system free from ring rot.

Implementation of crop rotation, disinfection, and other sanitation practices is most important whenever the disease has occurred to prevent recurrence of the disease and spread of the pathogen. Ring rot infected crops, and any adjacent crops that may have become contaminated should be eliminated from the production system and new certified seed should be acquired for any future production. Disinfectants effective against *Cms* include quaternary ammonia-, chlorine-, or iodine-containing compounds. These should be applied, after cleaning of equipment and other contaminated surfaces, to ensure a minimum of 10 min contact under low organic load. Control of potato volunteers and solanaceaous weeds is also important. The use of whole rather than cut seed helps to reduce any potential spread of the disease.

10.5 Common Scab of Potato Caused by *Streptomyces* Species

10.5.1 Taxonomy and Nomenclature

Streptomyces is a gram positive, aerobic, filamentous, spore-forming bacterium in the Streptomycetaceae family of the Actinobacteria. The filamentous mycelia have few or no cross walls. Spores are formed in spiral chains at the tips of hyphae. *Streptomyces* is the largest genus in the Actinobacteria and nearly 600 species are recognized. Most *Streptomyces* are soil-dwelling saprophytes and some species have a beneficial symbiosis with eukaryotes, including plants. At least 12 *Streptomyces* species cause common scab, netted scab, and/or pitted scab on potato. The names of several pathogenic *Streptomyces* species, such as *S. scabies*, were grammatically incorrect when they were first named and the scientific community has only recently begun using corrected names, such as *S. scabiei*.

Common scab is usually caused by *S. scabiei* (Thaxter 1892; Lambert and Loria 1989b), *S. acidiscabiei* (Lambert and Loria 1989a), or *S. turgidiscabiei* (Miyajima et al. 1998). Other species that cause scab symptoms on potato include the pitted scab pathogen *S. caviscabiei* (Goyer et al. 1996), three species first reported in France, including *S. europaeiscabiei*, *S. reticuliscabiei*, and *S. stelliscabiei* (Bouchek-Mechiche et al. 2000), three species first reported in Korea, including *S. luridiscabiei*, *S. niveiscabiei*, and *S. puniciscabiei* (Park et al. 2003), and one species reported in Japan, *S. cheloniumii* (Oniki et al. 1986a, b). The species *S. reticuliscabiei* is genomically the same as *S. turgidiscabiei*, but causes netted scab symptoms rather than typical common scab lesions (Bouchek-Mechiche et al. 2000, 2006). *S. diastatochromogenes* was recently reported as a common scab pathogen of potato, but there is no information available on its relative importance and the species identification was based solely on 16S rDNA sequence (Yang et al. 2017). A related species, *S. ipomeae*, causes root rot of sweet potato. Additional *Streptomyces* species capable of causing common scab have been isolated, but not yet described as species (see Table 1 in Bignell et al. 2014) and additional pathogenic species certainly remain to be discovered. Nonpathogenic strains exist within the pathogenic species, and none of the nonpathogenic strains appear to encode the phytotoxin thaxtomin (Wanner 2006, 2007, 2009).

10.5.2 Host Range

Potato is the most economically important host of plant pathogenic *Streptomyces* species. Plant pathogenic species are also able to cause disease on root crops, such as carrot, beet, parsnip, radish, sweet potato, and turnip (Goyer and Beaulieu 1997), and on peanut pods (Kritzman et al. 1996), but the economic impact of *Streptomyces* on these crops is less important than other diseases that infect these root crops.

10.5.3 Geographical Distribution

Pathogenic *Streptomyces* are present in soils wherever potato is grown and, as the name denotes, the disease it causes is one of the most common and most important potato diseases worldwide. Multiple species are present in individual fields and tubers (Wanner 2009; Lehtonen et al. 2004; Dees et al. 2013). Some species have only been reported from limited geographical regions, but no comprehensive global surveys have been done, so the distribution of pathogenic *Streptomyces* species remains mostly unexplored.

The spread of this pathogen is managed mainly through quality regulations which prohibit planting or shipping of severely affected seed, so there are essentially no limits on the spread of pathogenic *Streptomyces* through seed potatoes. Establishment of new *Streptomyces* strains in field soil is dependent on numerous complex factors, including soil chemistry and resident soil microbes, making establishment of pathogenic *Streptomyces* strains transported on seed potatoes unpredictable.

10.5.4 Symptoms

Streptomyces can cause necrosis on all underground parts of a potato (Fig. 10.6), including roots, stolons, and stems, and it can reduce growth of roots from seed tubers (Han et al. 2008). This pathogen can also cause necrosis on and kill potato seedlings grown from true potato seed. It does not directly cause foliar symptoms, although plant vigor may be reduced due to root necrosis caused by *Streptomyces*.

There is a wide variation in tuber symptoms caused by *Streptomyces*, including pitted scab, erumpent scab, and mild netted scab and symptom type depends, at least in part, on which toxins the infecting strain produces and the potato genotype. The pathogen colonizes tubers as they initiate, often entering the tube through lenticels. Whitish-grey bacterial mycelia and spores are sometimes visible in pitted scab lesions at harvest. The disease does not progress in storage, although tubers with severe pitted scab lesions will dehydrate and will not sprout the following season.

Fig. 10.6 Common scab symptoms on potato (Courtesy of AHDB Potatoes Sutton Bridge Crop Storage Research)

10.5.5 Epidemiology

Streptomyces has a relatively complex life cycle compared to many bacterial pathogens. It grows vegetatively as filamentous mycelia-like cells. When resources are depleted, the vegetative cells undergo programed cell death, nutrients are transferred to aerial reproductive hyphae, and spores are formed. These hyphae are sometimes visible without magnification inside scab lesions. Pathogenic *Streptomyces* grow best in soils with a pH between 5.2 and 8.0, and temperature of 20–22 °C, which are conditions that also favor potato growth.

Streptomyces survives and disperses mainly through cylindrical spores formed at hyphal tips. The spores can disperse in water, on soil-dwelling invertebrates, and on seed tubers. *Streptomyces* spores can survive in soil for 20 or more years and the spores are heat resistant. The pathogen spores germinate and enter the plant through natural openings, such as lenticels, or through wounds. Tubers are most susceptible to *Streptomyces* colonization during the first month of development. *Streptomyces* cannot cause lesions on mature tubers and lesion size and severity does not progress during storage, although tubers with severe pit scab may become dehydrated and will not sprout the following season.

Because multiple *Streptomyces* species are present in field soil and on diseased plants, epidemiological studies now rely on molecular detection of the species present in order to understand the impacts of management methods, soil characteristics, or biocontrol strains. PCR assays capable of distinguishing *Streptomyces* species are available (Wanner 2009). PCR assays designed to detect genes encoding thaxtomin are also used in epidemiological studies because detection of thaxtomin DNA is correlated with ability of an isolate to cause common scab (Wanner 2006, 2007, 2009; Flores-González et al. 2008) and with development of common scab symptoms in field soils (Qu et al. 2008).

Soils that suppress common scab exist and ongoing work is aimed at identifying the communities that lead to suppressiveness. Soils that suppress common scab have high *Streptomyces* populations. These saprophytic streptomycetes produce antibiotics that inhibit pathogenic *Streptomyces* or that compete with pathogenic *Streptomyces* for resources, thereby reducing common scab (for a comprehensive review, see Schlatter et al. 2017).

10.5.6 Pathogenicity Determinants and Resistance

Bacteria in this genus have unusually large linear genomes of 10–12 Mb and they produce diverse secondary metabolites. In the plant pathogenic *Streptomyces*, large pathogenicity islands encompassing several hundred genes encode virulence genes required for production of secondary metabolites, such as toxins, cytokinin, nitric oxide, and secreted proteins (Bignell et al. 2010; Joshi and Loria 2007). At least two of these pathogenicity islands are mobile (Bukhalid et al. 2002) and one of them can

mobilize at least one otherwise nonmobile pathogenicity island (Zhang and Loria 2017). As a result, pathogenicity can be transferred to previously nonpathogenic species (Zhang and Loria 2017).

Phytotoxins are the main *Streptomyces* pathogenicity determinants and the toxin thaxtomin appears to be required for pathogenicity (for a recent review, see Bignell et al. 2014). Thaxtomins, which are nitrated dipeptides (tryptophan and phenylalanine), are required for the development of common scab symptoms (King et al. 1989, 1991; Kinkel et al. 1998). Thaxtomin appears to weaken plant cell walls and cause plant cell hypertrophy through inhibition of cellulose synthesis and cell wall acidification (Fry and Loria 2002; Bischoff et al. 2009). This toxin can be used in potato breeding since seedling tolerance to thaxtomin is correlated with tolerance to common scab in the field (Hiltunen et al. 2011).

The other types of toxins produced by pathogenic *Streptomyces*, including coronatine-like toxins (Fyans et al. 2015), concanamycin (Natsume et al. 2017), borrelidin (Cao et al. 2012), and FD-891 (Natsume et al. 2005), are not necessarily produced by all pathogenic strains and production of these toxins may affect whether an individual strain produces pitted, net, or erumpent common scab symptoms. For example, concanamycin, a type of toxin produced by *S. scabies*, but not by some other *Streptomyces* species, may be required for formation of pitted scab lesions and appears to be synergistic with thaxtomin (Natsume et al. 2017).

Enzymes may also play a role in *Streptomyces* pathogenicity. *Streptomyces* lesions typically do not autofluoresce, suggesting that suberin formation is either inhibited or digested. Two genes that encode potential suberinases are present in the *S. scabies* genome and biochemical evidence supports that suberin is degraded (Beaulieu et al. 2016; Komeil et al. 2013). Degradation of suberin also appears to increase expression of the numerous cellulases produced by *S. scabies* (Padilla-Reynaud et al. 2015). *Streptomyces* toxin production is induced by plant-derived molecules, including the disaccharide cellobiose, a breakdown product of cellulose.

Little is known about the genetic basis of resistance to common scab. Suggested mechanisms include phellum layer thickness (Thangavel et al. 2016), phellum suberization (Thangavel et al. 2016; Khatri et al. 2011), detoxification of thaxtomin (Acuna et al. 2001), or sustained expression of disease defense genes (Merete Wiken Dees et al. 2016). Differences in ability of potato varieties to support growth of nonpathogenic *Streptomyces* species may also affect susceptibility to common scab (Wanner 2007).

10.5.7 Significance and Economic Loss

Common scab can cause complete loss, although this is usually associated with mismanagement of the crop, such as adding too much lime to a field, insufficient irrigation, or highly susceptible varieties planted in fields with high disease pressure.

Direct losses occur annually, however, worldwide, and common scab is often listed among the most important potato diseases (for example, Hill and Lazarovits 2005).

10.5.8 Management

The best option is disease tolerance or resistance, but currently there are limited options for potato varieties with high tolerance to common scab. Common scab symptom development is affected by soil moisture and chemistry, the soil microbial community, crop rotation, and host genetics in a complex manner that has made predicting common scab severity and managing this disease difficult. A comprehensive review of these challenges was published by Dees and Wanner (2012). Recommendations for management of common scab usually include adequate irrigation during tuber formation, and low soil pH (<5.2). Typically, sulfur fertilizers are used to reduce soil pH and this can reduce disease severity (Pavlista 2005). However, these methods sometimes fail to provide adequate management and can lead to other production problems. For example, over-irrigation during tuber formation can lead to development of powdery scab and several other potato diseases, and low soil pH limits farmer options for crop rotations and selects for *S. acidiscabies*.

Chemical treatments can work for a season, but are often expensive and damaging to the soil, making this the least sustainable disease management option. Some commonly used effective chemicals include fludioxonil as a seed piece treatment, chloropicrin as a soil fumigant, and pentachloronitrobenzene as an in-furrow treatment (Al-Mughrabi et al. 2016; Powelson and Rowe 2008). Fluazinam may also provide some control of common scab (Santos-Cervantes et al. 2017).

Crop rotation choices can also reduce common scab severity (Powelson and Rowe 2008; Larkin and Halloran 2014; Larkin et al. 2011; Larkin and Griffin 2007). These crop rotations tend to include brassica crops as a biofumigant and commonly planted green manures that are allelopathic and that help control multiple soil-borne potato diseases. Soil amendments, such as rice bran, chelated iron, or peat can decrease common scab, likely by increasing the population of nonpathogenic streptomycetes (Tomihama et al. 2016; Sarikhani et al. 2017). Some soil amendments, such as manure, which increases soil pH, will increase common scab severity.

Biocontrol with nonpathogenic *Streptomyces* strains also shows promise and the mechanism of biocontrol is likely similar to that seen in suppressive soils, which is thought to be due to both resource completion and antibiotic production (Schlatter et al. 2017). Suppressive soils develop through repeated monoculture of potato, but this practice results in accumulation of other soil-borne pathogens. However, repeated inoculations of soils with a single antagonistic *Streptomyces* strain can result in common scab suppression in as little as 3 years, and suppressive lasted for 2 years beyond the last inoculation (Hiltunen et al. 2017).

10.6 Zebra Chip of Potato Caused by *Liberibacter*

10.6.1 Taxonomy and Nomenclature

The genus "*Candidatus* Liberibacter" is a gram-negative bacterium in the Rhizobeaceae family. At least seven *Ca. Liberibacter* species exist. Of these, "*Candidatus* Liberibacter solanacearum" (Lso), which is a phloem-limited pathogen, is the only known potato-infecting species. There are at least five Lso haplotypes, with haplotypes A and B causing disease on potato and the remaining three haplotypes infecting carrots and celery (Nelson et al. 2011; Teresani et al. 2014). At 1.26 Mbp, the circular Lso genome is relatively small (Hong Lin et al. 2011) and there are relatively few genomic differences among Lso haplotypes (Wang et al. 2017a, b, c). Compared to related free-living bacteria, such as *Agrobacterium*, Lso has a low G + C content and lacks many genes involved in metabolism.

10.6.2 Host Range

Potato is the most economically important host of Lso haplotypes A and B, but Lso can also infect other solanaceous crops and weeds. All Ca. Liberibacter are spread by *Bactericera* species and Lso also infects its vector and can reduce vector fitness (Yao et al. 2016). Although Lso is only spread in potato by *B. cockerelli*, but it can also be found in other *Bactericera* species, suggesting that vector feeding preferences limit the species of vectors important for zebra chip and not Lso-vector interactions (Borges et al. 2017).

10.6.3 Geographical Distribution

Potato psyllids are native to North and Central America, and it recently invaded New Zealand (Teulon et al. 2009). The bacterial pathogen has spread with its vector and can be found wherever potato psyllids are found. The highest disease incidence is typically found in the Southwestern United States, Mexico, and Central America.

10.6.4 Symptoms

Zebra chips symptoms are severe on both the foliage and the tubers. The upper parts of infected plants have leaf curling, chlorosis, shortened internodes, aerial tubers, and early necrosis and death (Buchman et al. 2012) (Fig. 10.7). The tubers appear to have glassy or brown streaks that darken when they are fried, giving the

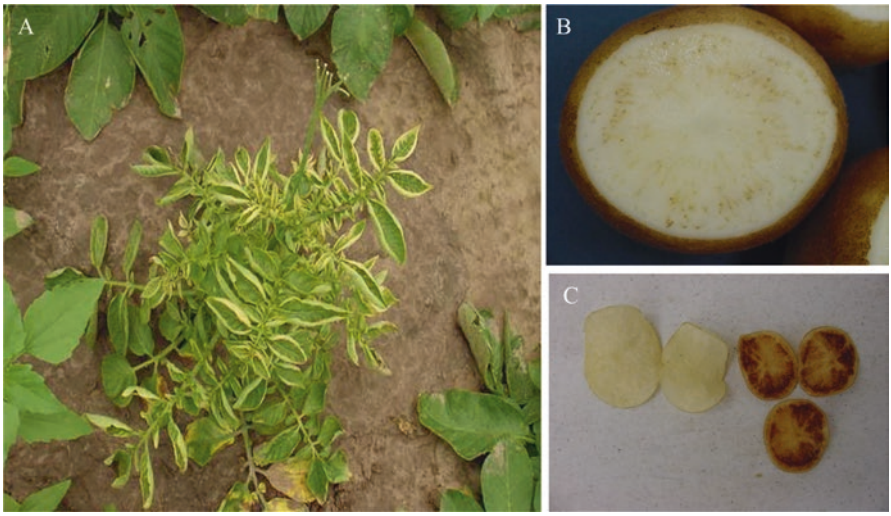

Fig. 10.7 Typical Lso symptoms on potato. Leaf curl symptoms on foliage (**a**); Necrotic specks in tuber (**b**); Blackened splotches on a fried chip (healthy on left, and diseased on right) (**d**) (Courtesy of Gary A. Secor, North Dakota State University)

disease its name, zebra chip. Tuber development slows or ceases in symptomatic plants, resulting in yield losses. Lso appears to reduce protease inhibitor levels in tubers, and as a result, tubers from infected plants have less protein (Kumar et al. 2015). Infected tubers either do not sprout or have only hair sprouts (Rashed et al. 2015). If plants emerge from infected tubers, they die shortly after emergence. Storage temperature affects symptom development, with cooler storage (3 °C) resulting more tuber symptoms than warmer storage temperatures (6 or 9 °C) (Wallis et al. 2017).

10.6.5 Epidemiology

Lso is transmitted solely by *B. cockerelli*, which feeds on phloem with its piercing-sucking mouthparts. The pathogen is transmitted in a persistent, propagative, and circulative fashion and it is also transmitted transovarially in the psyllid (Cicero et al. 2016; Hansen et al. 2008). A 2-week latent period occurs between psyllid acquisition of Lso and ability to transmit the pathogen (Sengoda et al. 2014). In most regions in North America where the pathogen and vector are present, potatoes are infected late in the season. Surprisingly, Lso reduces the fitness of its insect vector, with haplotype B resulting in more insect mortality than haplotype A (Yao et al. 2016).

Once transmitted into a leaf, the pathogen does not cause symptoms for at least 3 weeks. It is also not evenly distributed in plants, which makes it difficult to detect

prior to symptom development and this has hampered epidemiological studies. Lso is not culturable, which also makes epidemiological studies more challenging and as a result, researchers rely mainly on PCR assays for pathogen detection (Ananthakrishnan et al. 2013; Secor et al. 2009). Based on symptom development, Lso appears to be sensitive to temperatures above 32 °C and to thrive at 27–32 °C. Solanaceous weeds serve as important reservoirs for Lso and can provide a green bridge between potato crops (Thinakaran et al. 2015).

Infected tubers rarely sprout and when they do, they tend to develop hair sprouts. As a result, this disease is poorly transmitted through seed potatoes and insect transmission remains the most important mode of spread. For this reason, zebra chip is not currently regulated through seed potato certification in North America. It has, however, impacted export of potatoes from North America.

10.6.6 Pathogenicity Determinants and Resistance

Liberibacter pathogenicity determinants were recently thoroughly reviewed (Wang et al. 2017a, b, c). There are no resistant potato varieties, although timing and severity of symptoms differ among varieties (Lévy et al. 2015). Tolerant lines still support Lso levels similar to those found in susceptible varieties, but the Lso has less impact on plant physiology and symptom development in tubers in tolerant lines (Rashidi et al. 2017; Wallis et al. 2015). Recent results also suggest that psyllids are not able to transmit the pathogen with equal efficiency into all potato lines (Rashidi et al. 2017).

10.6.7 Significance and Economic Loss

Zebra chip has caused millions in losses in North America, and although seed tubers are not a major source of inoculum, it has affected the potato export market. The spread of this pathogen and its vector to New Zealand has also caused significant losses there. In addition to losses in yield and quality, the high cost of vector management has added to financial losses caused by *L. solanacearum*.

10.6.8 Management

Insecticides are the main management method used for control of zebra chip. Growers in North America monitor psyllids and determine when psyllids appear and the percentage of Lso-infected psyllids present. They may spray insecticides a dozen or more times during the growing season to protect the potato crop, with imidocloprid and spirotetramat among the most commonly used (Guenthner et al.

2012). Since the insects tend to be present on the underside of leaves, effectively covering the underside of the leaves is essential. These sprays are expensive and the potential for insecticide resistance and loss of natural enemies due to frequent sprays makes this approach unsustainable in the long term.

10.7 Concluding Remarks

Bacterial diseases of potato have remained an economically significant disease worldwide. Farmers lose millions of dollars annually due to bacterial diseases. Bacterial wilt, soft rot, and ring rot have got international attention as they constitute a huge constraint on seed potato production, with considerable indirect effects on trade. Rigorous seed certification and testing programs in developed countries have limited the impact of these diseases within their value chains, while developing countries commonly lack these safeguards. Lack of certified disease-free planting material in many developing country contexts contributes to further distribution of these pathogens via latently infected tubers, as well as tuber quality and yield degeneration caused by farmers replanting diseased seed year to year. Bacterial disease management efforts in developing countries should follow the systems approach that incorporates specific operational practices to reduce the likelihood of incursion, establishment, and growth of these pathogens in potato crops. This includes training farmers in proper production practices, on-farm management tools, using healthy seed tubers, and planting in clean soils. Additional factors to consider in controlling these diseases can include sanitation, cultural practices, crop rotation with nonhost plants, and the use of tolerant or resistant varieties.

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