Chapter 10
Bacterial Diseases of Potato

Amy Charkowski, Kalpana Sharma, Monica L. Parker, Gary A. Secor, and John Elphinstone

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.
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 β-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).

![Phylogenetic relatedness of strains within the *Ralstonia solanacearum* species complex](image)

**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 solanaceous 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 Caribbean, 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](https://example.com/image.png) 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 \textit{R. solanacearum} and \textit{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 \textit{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 \textit{hrpB} is induced, in response to plant signals, activating other \textit{hrp} (hypersensitive response and pathogenicity) genes in construction (\textit{hrpK} and \textit{hrpY}) and regulation (\textit{hrpB} and \textit{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. \textit{HrpB} positively regulates expression not only of \textit{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 subsistence 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 β-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. brasiiliense, 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. betavascularum*, 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. peruviana*, *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. parmentieri* 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 β-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
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.

**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)
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 (subsp. *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 find-
ings 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 environ-
ment. 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
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 $-40 \, ^\circ C$. 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.

**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.)
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, *chp-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 hydrolyzed 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 solanaceous 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 programmed 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 \textit{Streptomyces} pathogenicity determinants and the toxin thaxtomin appears to be required for pathogenicity (for a recent review, see Bignell et al. 2014). Thaxtomin, 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 \textit{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 \textit{S. scabies}, but not by some other \textit{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 \textit{Streptomyces} pathogenicity. \textit{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 \textit{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 \textit{S. scabies} (Padilla-Reynaud et al. 2015). \textit{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 \textit{Streptomyces} species may also affect susceptibility to common scab (Wanner 2007).

\section*{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 \textit{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 \textit{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 \textit{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
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.
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.

### References

Acuna IA, Strobel GA, Jacobsen BJ, Corsini DL (2001) Glucosylation as a mechanism of resistance to thaxtomin A in potatoes. Plant Sci 161(1):77–88

Adeolu M, Alnajar S, Naushad S, Gupta RS (2016) Genome-based phylogeny and taxonomy of the ‘Enterobacteriales’: proposal for Enterobacteriales ord. nov. divided into the families Enterobacteriaceae, Erwiniaiceae fam. nov., Pectobacteriaceae fam. nov., Yersiniaceae fam. nov., Hafniaceae fam. nov., Morganellaceae fam. nov., and Budviciaceae fam. nov. Int J Syst Evol Microbiol 66(12):5575–5599. [https://doi.org/10.1099/ijsem.0.001485](https://doi.org/10.1099/ijsem.0.001485)

Al-Mughrabi KI, Vikram A, Poirier R, Jayasuriya K, Moreau G (2016) Management of common scab of potato in the field using biopesticides, fungicides, soil additives, or soil fumigants. Biocontrol Sci Tech 26(1):125–135

Ananthakrishnan G, Choudhary N, Roy A, Sengoda VG, Postnikova E, Hartung JS, Stone AL, Damsteegt VD, Schneider WL, Munyaneza JE (2013) Development of primers and probes for genus and species specific detection of ‘Candidatus Liberibacter species’ by real-time PCR. Plant Dis 97(9):1235–1243

Appel O (1906) Neue Untersuchungen Über Kartoffel-Und Tomaten-Erkrankungen. Borntraeger, Stuttgart
Beaulieu C, Sidibé A, Jabloune R, Simao-Beaunoir A-M, Lerat S, Monga E, Bernards MA (2016) Physical, chemical and proteomic evidence of potato suberin degradation by the plant pathogenic bacterium Streptomyces scabiei. Microbes Environ 31(4):427–434

Bentley SD, Corton C, Brown SE, Barron A, Clark L, Doggett J, Harris B, Ormond D, Quail MA, May G (2008) Genome of the actinomycete plant pathogen Clavibacter michiganensis subsp. sepedonicus suggests recent niche adaptation. J Bacteriol 190(6):2150–2160

Bignell DRD, Huguet-Tapia JC, Joshi MV, Pettis GS, Loria R (2010) What does it take to be a plant pathogen: genomic insights from Streptomyces species. Antonie Van Leeuwenhoek 98(2):179–194

Bignell DRD, Fyans JK, Cheng Z (2014) Phytotoxins produced by plant pathogenic Streptomyces species. J Appl Microbiol 116(2):223–235

Bischoff V, Cookson SJ, Wu S, Scheible W-R (2009) Thaxtomin A affects CESA-complex density, expression of cell wall genes, cell wall composition, and causes ectopic lignification in Arabidopsis thaliana seedlings. J Exp Bot 60(3):955–965

Borges KM, Rodney Cooper W, Garczynski SF, Thinakaran J, Jensen AS, Horton DR, Munyaneza JE, Cueva I, Barcenas NM (2017) ‘Candidatus Liberibacter solanacearum’ associated with the psyllid, Bactericera maculipennis (Hemiptera: Triozidae). Environ Entomol 46(2):210–216

Bouchek-Mechiche K, Gardan L, Normand P, Jouan B (2000) DNA relatedness among strains of Streptomyces pathogenic to potato in France: description of three new species, S. europaeiscabiei sp. nov. and S. stelliscabiei sp. nov. associated with common scab, and S. reticuliscabiei sp. nov. associated with netted scab. Int J Syst Evol Microbiol 50(1):91–99

Bouchek-Mechiche K, Gardan L, Andrivon D, Normand P (2006) Streptomyces turgidiscabies and Streptomyces reticuliscabiei: one genomic species, two pathogenic groups. Int J Syst Evol Microbiol 56(12):2771–2776

Brady CL, Cleenwerck I, Denman S, Venter SN, Rodríguez-Palenzuela P, Coutinho TA, De Vos P (2012) Proposal to reclassify Brenneria quercina (Hildebrand and Schroth 1967) Hauben et Al. 1999 into a new genus, Lonsdalea gen. nov., as Lonsdalea quercina comb. nov., descriptions of Lonsdalea quercina subsp. quercina comb. nov., Lonsdalea quercina subsp. iberica subsp. nov. and Lonsdalea quercina subsp. britannica subsp. nov., emendation of the description of the genus Brenneria, reclassification of Dickeya dieffenbachiae as Dickeya dadantii subsp. Dieffenbachiae comb. nov., and emendation of the description of Dickeya dadantii. Int J Syst Evol Microbiol 62(7):1592–1602. https://doi.org/10.1099/ijs.0.035055-0

Buchman JL, Fisher TW, Sengoda VG, Munyaneza JE (2012) Zebra chip progression: from inoculation of potato plants with Liberibacter to development of disease symptoms in tubers. Am J Potato Res 89(2):159–168

Buddenhagen IW (1962) Designations of races in pseudomonas Solanacearum. Phytopathology 52:726

Bukhalid RA, Takeuchi T, Labeda D, Loria R (2002) Horizontal transfer of the plant virulence gene, Nec1, and flanking sequences among genetically distinct Streptomyces strains in the Diastatochromogenes cluster. Appl Environ Microbiol 68(2):738–744

Burkholder WR, Mcfadden LA, Dimock EW (1953) A bacterial blight of chrysanthemums. Phytopathology 43(9). https://www.cabdirect.org/cabdirect/abstract/19541101119

Cao Z, Khodakaramian G, Arakawa K, Kinashi H (2012) Isolation of Borrelidin as a phytotoxic compound from a potato pathogenic Streptomyces strain. Biosci Biotechnol Biochem 76(2):353–357

Charkowski AO (2015) Biology and control of Pectobacterium in potato. Am J Potato Res 92(2):223–229

Charkowski AO (2018) The changing face of bacterial soft-rot diseases. Annu Rev Phytopathol 56(1):269–288. https://doi.org/10.1146/annurev-phyto-080417-045906

Charkowski A, Blanco C, Condemine G, Expert D, Franza T, Hayes C, Hugouvieux-Cotte-Pattat N et al (2012) The role of secretion systems and small molecules in soft-rot Enterobacteriaceae pathogenicity. Annu Rev Phytopathol 50(1):425–449. https://doi.org/10.1146/annurev-phyto-081211-173013
Christie RD, Sumalde AC, Schulz JT, Gudmestad NC (1991) Insect transmission of the bacterial ring rot pathogen. Am Potato J 68(6):363–372

Cicero JM, Fisher TW, Qureshi JA, Stansly PA, Brown JK (2016) Colonization and intrusive invasion of potato Psyllid by ‘Candidatus Liberibacter solanacearum’. Phytopathology 107(1):36–49

Czajkowski R, Pénrombelon MCM, van Veen JA, van der Wolf JM (2011) Control of blackleg and tuber soft rot of potato caused by Pectobacterium and Dickeya species: a review. Plant Pathol 60(6):999–1013. https://doi.org/10.1111/j.1365-3059.2011.02470.x

Czajkowski R, de Boer WJ, van der Wolf JM (2013) Chemical disinfectants can reduce potato blackleg caused by ‘Dickeya solani’. Eur J Plant Pathol 136(2):419–432. https://doi.org/10.1007/s10658-013-0177-8

Czajkowski R, Pénrombelon MCM, Jafra S, Lojkowska E, Potrykus M, van der Wolf JM, Sledz W (2015) Detection, identification and differentiation of Pectobacterium and Dickeya species causing potato blackleg and tuber soft rot: a review. Ann Appl Biol 166(1):18–38. https://doi.org/10.1111/aab.12166

De Boer SH, McCann M (1990) Detection of Corynebacterium sepedonicum in potato cultivars with different propensities to express ring rot symptoms. Am Potato J 67(10):685

De Boer SH, Li X, Ward LJ (2012) Pectobacterium spp. associated with bacterial stem rot syndrome of potato in Canada. Phytopathology 102(10):937–947

de Werra P, Bussereau F, Keiser A, Ziegler D (2015) First report of potato blackleg caused by Pectobacterium carotovorum subsp. Brasiliense in Switzerland. Plant Dis 99(4):551

Dees MW, Wanner LA (2012) In search of better management of potato common scab. Potato Res 55(3–4):249–268

Dees MW, Sletten A, Hermansen A (2013) Isolation and characterization of Streptomyces species from potato common scab lesions in Norway. Plant Pathol 62(1):217–225

Dees MW, Lysøe E, Alsheikh M, Davik J, Brurberg MB (2016) Resistance to Streptomyces turbidiscabies in potato involves an early and sustained transcriptional reprogramming at initial stages of tuber formation. Mol Plant Pathol 17(5):703–713

Dees MW, Lebecka R, Perminow JIS, Czajkowski R, Motyka A, Zoledowska S,Śliwka J, Lojkowska E, Brurberg MB (2017a) Characterization of Dickeya and Pectobacterium strains obtained from diseased potato plants in different climatic conditions of Norway and Poland. Eur J Plant Pathol 148(4):839–851

Dees MW, Lysøe E, Rossmann S, Perminow J, Brurberg MB (2017b) Pectobacterium polaris sp. nov., isolated from potato (Solanum tuberosum). Int J Syst Evol Microbiol 67(12):5222–5229

Duarte V, De Boer SH, Ward LJ, De Oliveira AMR (2004) Characterization of atypical Erwinia carotovora strains causing blackleg of potato in Brazil. J Appl Microbiol 96(3):535–545

Eichenlaub R, Gartemann K-H (2011) The Clavibacter michiganensis subspecies: molecular investigation of gram-positive bacterial plant pathogens. Annu Rev Phytopathol 49:445–464

Elphinstone JG (2005) The current bacterial wilt situation: a global overview. In: Allen C, Piör P, Hayward AC (eds) Bacterial wilt disease and the Ralstonia solanacearum species complex. APS Press, St Paul, MN, pp 9–28

Fegan M, Piör P (2005) How complex is the Ralstonia solanacearum species complex. APS Press, St Paul

Fikowicz-Krosko J, Czajkowski R (2017) Systemic colonization and expression of disease symptoms on bittersweet nightshade (Solanum dulcamara) infected with a GFP-tagged Dickeya solani IPO2222 (IPO2254). Plant Dis 102(3):619–627. https://doi.org/10.1094/PDIS-08-17-1147-RE

Flores-González R, Velasco I, Montes F (2008) Detection and characterization of Streptomyces causing potato common scab in Western Europe. Plant Pathol 57(1):162–169

Frost KE, Groves RL, Charkowski AO (2013) Integrated control of potato pathogens through seed potato certification and provision of clean seed potatoes. Plant Dis 97(10):1268–1280. https://doi.org/10.1094/PDIS-05-13-0477-FE

Fry BA, Loria R (2002) Thaxtomin A: evidence for a plant cell wall target. Physiol Mol Plant Pathol 60(1):1–8
Fyans JK, Altowairish MS, Li Y, Bignell DRD (2015) Characterization of the coronatine-like phyto-toxins produced by the common scab pathogen Streptomyces scabies. Mol Plant-Microbe Interact 28(4):443–454
Genin S, Denny TP (2012) Pathogenomics of the Ralstonia solanacearum species complex. Annu Rev Phytopathol 50:67–89
Goyer C, Beaulieu C (1997) Host range of Streptomyces strains causing common scab. Plant Dis 81(8):901–904
Goyer C, Faucher E, Beaulieu C (1996) Streptomyces Caviscabies sp. nov., from deep-pitted lesions in potatoes in Québec, Canada. Int J Syst Evol Microbiol 46(3):635–639
Guenthner J, Goolsby J, Greenway G (2012) Use and cost of insecticides to control potato psyllids and zebra chip on potatoes. Southwest Entomol 37(3):263–270
Han L, Dutilleul P, Prasher SO, Beaulieu C, Smith DL (2008) Assessment of common scab-inducing pathogen effects on potato underground organs via computed tomography scanning. Phytopathology 98(10):1118–1125
Hansen AK, Trumble JT, Stouthamer R, Paine TD (2008) A new Huanglongbing species, ‘Candidatus Liberibacter Psyllaurous,’ found to infect tomato and potato, is vectored by the Psyllid Bactericera Cockerelli (Sulc). Appl Environ Microbiol 74(18):5862–5865
Hauben L, Moore ERB, Vauterin L, Steenackers M, Mergaert J, Verdonck L, Swings J (1998) Phylogenetic position of phytopathogens within the Enterobacteriaceae. Syst Appl Microbiol 21(3):384–397. https://doi.org/10.1016/S0723-2020(98)80048-9
Hayward AC (1964) Characteristics of Pseudomonas solanacearum. J Appl Bacteriol 27(2):265–277
Hellmers E (1958) Four wilt diseases of perpetual-flowering Carnations in Denmark. Dansk botanisk Arkiv 18(2):1–100. https://www.cabdirect.org/cabdirect/abstract/19581101855
Hill J, Lazarovits G (2005) A mail survey of growers to estimate potato common scab prevalence and economic loss in Canada. Can J Plant Pathol 27(1):46–52
Hiltunen LH, Alanen M, Laakso I, Kangas A, Virtanen E, Valkonen JPT (2011) Elimination of common scab sensitive progeny from a potato breeding population using Thaxtomin A as a selective agent. Plant Pathol 60(3):426–435
Hiltunen LH, Kelloniemi J, Valkonen JPT (2017) Repeated applications of a nonpathogenic Streptomyces strain enhance development of suppressiveness to potato common scab. Plant Dis 101(1):224–232
Holtsmark I, Takle GW, Brurberg MB (2008) Expression of putative virulence factors in the potato pathogen Clavibacter michiganensis subsp. sepedonicus during infection. Arch Microbiol 189(2):131–139
Huet G (2014) Breeding for resistances to Ralstonia Solanacearum. Front Plant Sci 5:715
Humphris SN, Cahill G, Elphinstone JG, Kelly R, Parkinson NM, Pritchard L, Toth IK, Saddler GS (2015) Detection of the bacterial potato pathogens Pectobacterium and Dickeya Spp. using conventional and real-time PCR. In: Lacomme C (ed) Plant pathology: techniques and protocols, Methods in molecular biology. Springer, New York, pp 1–16. https://doi.org/10.1007/978-1-4939-2620-6_1
Jacobs JM, Babujee L, Meng F, Milling A, Allen C (2012) The in planta transcriptome of Ralstonia solanacearum: conserved physiological and virulence strategies during bacterial wilt of tomato. MBio 3(4):e00114–e00112
Janse JD, Araluppan FAX, Schans J, Wenneker M, Westerhuis W (1998) Experiences with bacterial brown rot Ralstonia solanacearum biovar 2, race 3 in the Netherlands, Bacterial wilt disease. Springer, Berlin, pp 146–152
Joshi MV, Loria R (2007) Streptomyces turgidiscabies possesses a functional cytokinin biosynthetic pathway and produces leafy galls. Mol Plant-Microbe Interact 20(7):751–758
Khatri BB, Tegg RS, Brown PH, Wilson CR (2011) Temporal association of potato tuber development with susceptibility to common scab and Streptomyces scabiei-induced responses in the potato periderm. Plant Pathol 60(4):776–786
Khayi S, Cigna J, Chong TM, Quêtu-Laurent A, Chan K-G, Hélias V, Faure D (2016) Transfer of the potato plant isolates of Pectobacterium wasabiae to Pectobacterium parmentieri sp. nov. Int J Syst Evol Microbiol 66(12):5379–5383
Kim H-S, Ma B, Perna NT, Charkowski AO (2009) Phylogeny and virulence of naturally occurring type III secretion system-deficient Pectobacterium strains. Appl Environ Microbiol 75(13):4539–4549
King RR, Harold Lawrence C, Clark MC, Calhoun LA (1989) Isolation and characterization of phytotoxins associated with Streptomyces scabies. J Chem Soc Chem Commun 13:849–850
King RR, Harold Lawrence C, Clark MC (1991) Correlation of phytotoxin production with pathogenicity of streptomyces scabies isolates from scab infected potato tubers. Am Potato J 68(10):675–680
Kinkel LL, Bowers JH, Shimizu K, Neeno-Eckwall EC, Schottel JL (1998) Quantitative relationships among Thaxtomin A production, potato scab severity, and fatty acid composition in streptomyces. Can J Microbiol 44(8):768–776
Kloeper JW, Schroth MN (1981) Relationship of in vitro antibiosis of plant growth-promoting rhizobacteria to plant growth and the displacement of root microflora. Phytopathology 71: 1020–1024
Komeil D, Simao-Beaunoir A-M, Beaulieu C (2013) Detection of potential suberinase-encoding genes in Streptomyces scabiesi strains and other actinobacteria. Can J Microbiol 59(5):294–303
Kong HG, Bae JY, Lee HJ, Joo HJ, Jung EJ, Chung E, Lee S-W (2014) Induction of the viable but nonculturable state of Ralstonia solanacearum by low temperature in the soil microcosm and its resuscitation by catalase. PLoS One 9(10):e109792
Kritzman G, Shani-Cahani A, Kirshner B, Riven Y, Bar Z, Katan J, Grinstein A (1996) Pod wart disease of peanuts. Phytoparasitica 24(4):293–304
Kumar GNM, Knowles LO, Richard Knowles N (2015) Zebra chip disease decreases tuber (Solanum tuberosum L.) protein content by attenuating protease inhibitor levels and increasing protease activities. Planta 242(5):1153–1166
Laine MJ, Haapalainen M, Wahlroos T, Kankare K, Nissinen R, Kassuwi S, Metzler MC (2000) The cellulase encoded by the native plasmid of Clavibacter michiganensis ssp. sepedonicus plays a role in virulence and contains an expansin-like domain. Physiol Mol Plant Pathol 57(5):221–233
Lambert DH, Loria R (1989a) Streptomyces scabies sp. nov., nom. rev. Int J Syst Evol Microbiol 39:387–392
Lambert DH, Loria R (1989b) Streptomyces acidiscabies sp. nov. Int J Syst Evol Microbiol 39:393–396
Larkin RP, Griffin TS (2007) Control of Soilborne potato diseases using Brassica green manures. Crop Prot 26(7):1067–1077
Larkin RP, Halloran JM (2014) Management effects of disease-suppressive rotation crops on potato yield and soilborne disease and their economic implications in potato production. Am J Potato Res 91(5):429–439
Larkin RP, Wayne Honeycutt C, Griffin TS, Modesto Olaya O, Halloran JM, He Z (2011) Effects of different potato cropping system approaches and water management on soilborne diseases and soil microbial communities. Phytopathology 101(1):58–67
Laurila J, Metzler MC, Ishimaru CA, Rokka V-M (2003) Infection of plant material derived from Solanum acaule with Clavibacter michiganensis ssp. sepedonicus: temperature as a determining factor in immunity of S. acaule to bacterial ring rot. Plant Pathol 52(4):496–504
Laurila J, Ahola V, Lehtinen A, Joutsjoki T, Hannukkala A, Rahkonen A, Pirhonen M (2008) Characterization of Dickeya strains isolated from potato and river water samples in Finland. Eur J Plant Pathol 122(2):213–225. https://doi.org/10.1007/s10658-008-9274-5
Lehtonen MJ, Rantal S, Kreuze JF, Bang H, Kuisma L, Koski P, Virtanen E, Vihlman K, Valkonen JPT (2004) Occurrence and survival of potato scab pathogens (Streptomyces species) on tuber lesions: quick diagnosis based on a PCR-based assay. Plant Pathol 53(3):280–287
Lévy JG, Scheuring DC, Koym JW, Henne DC, Tamborindeguy C, Pierson E, Creighton Miller J (2015) Investigations on putative zebra chip tolerant potato selections. Am J Potato Res 92(3):417–425
Lin H, Lou B, Glynn JM, Doddapaneni H, Civerolo EL, Chen C, Duan Y, Zhou L, Vahling CM (2011) The complete genome sequence of ‘Candidatus Liberibacter solanacearum’, the bacterium associated with potato zebra chip disease. PLoS One 6(4):e19135
Lu Y, Hatusgai N, Katagiri F, Ishimaru CA, Glazebrook J (2015) Putative serine protease effectors of Clavibacter michiganensis induce a hypersensitive response in the apoplast of Nicotiana species. Mol Plant-Microbe Interact 28(11):1216–1226
Ma B, Hibbing ME, Kim HS, Reedy RM, Yedidia I, Breuer J, Breuer J, Glasner JD, Perna NT, Kelman A, Charkowski AO (2007a) Host range and molecular phylogenies of the soft rot enterobacterial genera Pectobacterium and Dickeya. Phytopathology 97(9):1150–1163. https://apsjournals.apsnet.org/doi/abs/10.1094/PHYTO-97-9-1150
Ma B, Hibbing ME, Kim H-S, Reedy RM, Yedidia I, Breuer J, Breuer J, Glasner JD, Perna NT, Kelman A (2007b) Host range and molecular phylogenies of the soft rot enterobacterial genera Pectobacterium and Dickeya. Phytopathology 97(9):1150–1163
Mansfield J, Genin S, Magori S, Citovsky V, Satriyanum M, Ronald P, Dow MAX, Verdier V, Beer SV, Machado MA (2012) Top 10 plant pathogenic bacteria in molecular plant pathology. Mol Plant Pathol 13(6):614–629
Manzer FE, McKenzie AR (1988) Cultivar response to bacterial ring rot infection in Maine. Am Potato J 65(6):333–339
Meng F (2013) The virulence factors of the bacterial wilt pathogenRalstonia solanacearum. J Plant Pathol Microbiol 4(168):10:4172
Miyajima K, Tanaka F, Takeuchi T, Kuninaga S (1998) Streptomyces turdigiscabies sp. nov. Int J Syst Evol Microbiol 48(2):333–339
Nabhan S, De Boer SH, Maiss E, Wydra K (2013) Pectobacterium aroidearum sp. nov., a soft rot pathogen with preference for monocotyledonous plants. Int J Syst Evol Microbiol 63(7):2520–2525
Narasaka M, Kubo Y, Hatakeyama K, Imamura J, Ezura H, Nanasato Y, Tabei Y, Takano Y, Shirasu K, Narusaka Y (2013) Interfamily transfer of dual NB-LRR genes confers resistance to multiple pathogens. Int J Syst Evol Microbiol 48(2):495–502
Natsume M, Komiyama M, Koyanagi F, Tashiro N, Kawai H, Abe H (2005) Phytotoxin produced by Streptomyces sp. causing potato russet scab in Japan. J Gen Plant Pathol 71(5):364–369
Natsume M, Tashiro N, Doi A, Nishi Y, Kawai H (2017) Effects of concanamycins produced by Streptomyces scabies on lesion type of common scab of potato. J Gen Plant Pathol 83(2):78–82
Nelson WR, Fisher TW, Munyanzea JE (2011) Haplotypes of ‘Candidatus Liberibacter solanacearum’ suggest long-standing separation. Eur J Plant Pathol 130(1):5–12
Ngadze E, Brady CL, Coutinho TA, Van der Waals JE (2012) Pectinolytic bacteria associated with potato soft rot and blackleg in South Africa and Zimbabwe. Eur J Plant Pathol 134(3):533–549
Nissinen R, Kassuwi S, Peltoila R, Metzler MC (2001) In planta-complementation of Clavibacter michiganensis subsp. sepedonicus deficient in cellulase production. Eur J Plant Pathol 107(2):175–182
Nykyri J, Niemi O, Koskinen P, Nokso-Koivisto J, Pasanen M, Broberg M, Plyusnin I, Törönen P, Holm L, Pirhonen M (2012) Revised phylogeny and novel horizontally acquired virulence determinants of the model soft rot phytopathogen Pectobacterium wasabiae SCC3193. PLoS Pathog 8(11):e1003013
Oniki M, Suzui T, Araki T, Sonoda RI, Chiba T, Takeda T (1986a) Causal agent of russet scab of potato. Bull Nat Inst of Agro Environ Sci 2:45–60
Oniki M, Suzui T, Araki T, Sonoda R, Chiba T, Takeda T (1986b) Causal agent of russet scab of potato [Streptomyces sp.]. Bull Nat Inst Agro-Environ Sci. http://agris.fao.org/agris-search/search.do?recordID=JP870464388
Padilla-Reynaud R, Simao-Beaunoir A-M, Lerat S, Bernards MA, Beaulieu C (2015) Suberin regulates the production of cellulytic enzymes in Streptomyces scabiei, the causal agent of potato common scab. Microbes Environ 30(3):245–253

Park DH, Kim JS, Kwon SW, Wilson C, Yu YM, Hur JH, Lim CK (2003) Streptomyces luridiscabiei sp. nov., Streptomyces puniciscabiei sp. nov. and Streptomyces niveiscabiei sp. nov., which cause potato common scab disease in Korea. Int J Syst Evol Microbiol 53(6):2049–2054

Parkinson N, Stead D, Bew J, Heeney J, Tsror (Lahkim) L, Elphinstone J (2009) Dickeya species relatedness and clade structure determined by comparison of RecA sequences. Int J Syst Evol Microbiol 59(10):2388–2393. https://doi.org/10.1099/ijs.0.009258-0

Parkinson N, DeVos P, Pirhonen M, Elphinstone J (2014) Dickeya aquatica sp. nov., isolated from waterways. Int J Syst Evol Microbiol 64(7):2264–2266. https://doi.org/10.1099/ijs.0.058693-0

Pavlista AD (2005) Early-season applications of sulfur fertilizers increase potato yield and reduce tuber defects. Agron J 97(2):599–603

Pérombelon MCM (2002) Potato diseases caused by soft rot erwiniias: an overview of pathogenesis. Plant Pathol 51(1):1–12

Pérombelon MCM, Kelman A (1987) Blackleg and other potato diseases caused by soft rot erwiniias: proposal for revision of terminology. Plant Dis 71(3):283–285

Pirhonen M, Flego D, Heikinheimo R, Tapio Palva E (1993) A small diffusible signal molecule is responsible for the global control of virulence and exoenzyme production in the plant pathogen Erwina carotovora. EMBO J 12(6):2467–2476

Pitman AR, Wright PJ, Galbraith MD, Harrow SA (2008) Biochemical and genetic diversity of pectolytic enterobacteria causing soft rot disease of potatoes in New Zealand. Australas Plant Pathol 37(6):559–568

Pitman AR, Harrow SA, Visnovsky SB (2010) Genetic characterisation of Pectobacterium wasbiae causing soft rot disease of potato in New Zealand. Eur J Plant Pathol 126(3):423–435

Powelson ML, Rowe RC (2008) Managing diseases caused by seedborne and soilborne fungi and fungus-like pathogens. Potato Health Manage:2183–2195

Prior P, Ailloud F, Dalsing BL, Remenant B, Sanchez B, Allen C (2016) Genomic and proteomic evidence supporting the division of the plant pathogen Ralstonia solanacearum into three species. BMC Genomics 17(1):90

Qian Y-l, Wang X-s, Wang D-z, Zhang L-n, Chao-long Z, Gao Z-l, Zhang H-j, Wang Z-y, Sun X-y, Yao D-n (2013) The detection of QTLs controlling bacterial wilt resistance in tobacco (N. Tabacum L.). Euphytica 192(2):259–266

Qu X, Wanner LA, Christ BJ (2008) Using the TxtAB operon to quantify pathogenic Streptomycetes in potato tubers and soil. Phytopathology 98(4):405–412

Rashed A, Workneh F, Paetzold L, Rush CM (2015) Emergence of ‘Candidatus Liberibacter solanacearum’-infected seed potato in relation to the time of infection. Plant Dis 99(2):274–280

Rashidi M, Novy RG, Wallis CM, Rashed A (2017) Characterization of host plant resistance to zebra chip disease from species-derived potato genotypes and the identification of new sources of zebra chip resistance. PLoS One 12(8):e0183283

Rossmann S, Dees MW, Perminow J, Meadow R, Brurberg MB (2018) Soft rot Enterobacteriaceae are carried by a large range of insect species in potato fields. Appl Environ Microbiol 84(12):e00281–e00218. https://doi.org/10.1128/AEM.00281-18

Safni I, Cleenwerck I, De Vos P, Fegan M, Sly L, Kappler U (2014) Polyphasic taxonomic revision of the Ralstonia solanacearum species complex: proposal to emend the descriptions of Ralstonia solanacearum and Ralstonia syzygii and reclassify current R. syzygii strains as Ralstonia syzygii subsp. syzygii subsp. nov., R. solanacearum phylotype IV strains as Ralstonia syzygii subsp. Indonesiensis subsp. nov., Banana blood disease bacterium strains as Ralstonia syzygii subsp. Celebesensis subsp. nov. and R. solanacearum phylotype I and III strains as Ralstonia pseudosolanacearum sp. nov. Int J Syst Evol Microbiol 64(9):3087–3103
Salgon S, Jourda C, Sauvage C, Daunay M-C, Reynaud B, Wicker E, Dintinger J (2017) Eggplant resistance to the Ralstonia solanacearum species complex involves both broad-spectrum and strain-specific quantitative trait loci. Front Plant Sci 8:828

Samson R, Legendre JB, Christen R, Saux MF-L, Achouak W, Gardan L (2005) Transfer of Pectobacterium chrysanthemi (Burkholder et Al. 1953) Brenner et Al. 1973 and Brenneria paradiisaca to the genus Dickeya gen. nov. as Dickeya chrysanthemi comb. nov. and Dickeya paradiisaca comb. nov. and delineation of four novel species, Dickeya dadantii sp. nov., Dickeya dianthicola sp. nov., Dickeya dieffenbachiae sp. nov. and Dickeya zeae sp. nov. Int J Syst Evol Microbiol 55( Pt 4):1415–1427. https://doi.org/10.1099/ijs.0.02791-0.

Santos-Cervantes ME, Felix-Gastelum R, Herrera-Rodríguez G, Espinoza-Mancillas MG, Mora-Romero AG, Leyva-López NE (2017) Characterization, pathogenicity and chemical control of Streptomyces acidiscabies associated to potato common scab. Am J Potato Res 94(1):14–25

Sarfraz S, Riaz K, Oulghazi S, Cigna J, Sahi ST, Khan SH, Faure D (2018) Pectobacterium punjabense sp. nov., isolated from blackleg symptoms of potato plants in Pakistan. Int J Syst Evol Microbiol 68(11):3551–3556

Sarikhani E, Sagova-Mareckova M, Omelka M, Kopecky J (2017) The effect of peat and iron supplements on the severity of potato common scab and bacterial community in tuberosphere soil. FEMS Microbiol Ecol 93(1)

Schlatter D, Kinkel L, Thomashow L, Weller D, Paulitz T (2017) Disease suppressive soils: new insights from the soil microbiome. Phytopathology 107(11):1284–1297

Secor GA, Rivera VV, Abad JA, Lee I-M, Clover GRG, Liefting LW, Li X, De Boer SH (2009) Association of ‘Candidatus Liberibacter solanacearum’ with zebra chip disease of potato established by graft and psyllid transmission, electron microscopy, and PCR. Plant Dis 93(6):574–583

Sengoda VG, Rodney Cooper W, Swisher KD, Henne DC, Munyaneza JE (2014) Latent period and transmission of ‘Candidatus Liberibacter solanacearum’ by the potato Psyllid Bactericera cockerellii (Hemiptera: Triozidae). PLoS One 9(3):e93475

She XM, Lan GB, Tang YF, He ZF (2017) Pectrobacterium caratovorum subsp. Brasilienise causing pepper black spot disease in China. J Plant Pathol 99(3):769–772

Sławiak M, van Beckhoven JRCM, Speksnijder AGCL, Czajkowski R, Grabe G, van der Wolf JM (2009) Biochemical and genetical analysis reveal a new clade of biovar 3 Dickeya spp. strains isolated from potato in Europe. Eur J Plant Pathol 125(2):245–261. https://doi.org/10.1007/s10658-009-9479-2

Teresani GR, Bertolini E, Alfaro-Fernández A, Martínez C, Tanaka FAO, Kitajima EW, Roselló M, Sanjuán S, Ferrándiz JC, López MM (2014) Association of ‘Candidatus Liberibacter solanacearum’ with a vegetative disorder of celery in Spain and development of a real-time PCR method for its detection. Phytopathology 104(8):804–811

Teulon DAI, Workman PJ, Nielsen MC, Thomas KL, Anderson DP (2009) Bactericera cockerelli: incursion, dispersal and current distribution on vegetable crops in New Zealand. New Zealand Plant Prot 62:136–144

Thangavel T, Tegg RS, Wilson CR (2016) Toughing it out—disease-resistant potato mutants have enhanced tuber skin defenses. Phytopathology 106(5):474–483

Thaxter R (1892) Potato scab. Potato Scab:153–160. https://www.cabdirect.org/cabdirect/abstract/20057000599

Thinakaran J, Pierson E, Kunta M, Munyaneza JE, Rush CM, Henne DC (2015) Silverleaf nightshade (Solanum Elaeagnifolium), a reservoir host for ‘Candidatus Liberibacter solanacearum’, the putative causal agent of zebra chip disease of potato. Plant Dis 99(7):910–915

Tian Y, Zhao Y, Yuan X, Yi J, Fan J, Zhigang X, Baishi H, De Boer SH, Li X (2016) Dickeyafangzhongdai sp. nov., a plant-pathogenic bacterium isolated from pear trees (Pyrus Pyrifolia). Int J Syst Evol Microbiol 66(8):2831–2835. https://doi.org/10.1099/ijsem.0.001060

Tomihama T, Nishi Y, Mori K, Shirao T, Iida T, Uzushashi S, Ohkuma M, Ikeda S (2016) Rice bran amendment suppresses potato common scab by increasing antagonistic bacterial cellular levels in the rhizosphere. Phytopathology 106(7):719–728
Toth IK, van der Wolf JM, Saddler G, Lojkowska E, Hélias V, Pirhonen M, Tsror (Lahkim) L, Elphinstone JG (2011) Dickeya species: an emerging problem for potato production in Europe. Plant Pathol 60(3):385–399. https://doi.org/10.1111/j.1365-3059.2011.02427.x

van der Merwe JJ, Coutinho TA, Korsten L, van der Waals JE (2010) Pectobacterium carotovorum subsp. brasiliensis causing blackleg on potatoes in South Africa. Eur J Plant Pathol 126(2):175–185

Van der Wolf JM, Elphinstone JG, Stead DE, Metzler M, Müller P, Hukkanen A, Karjalainen R (2005a) Epidemiology of Clavibacter michiganensis subsp. sepedonicus in relation to control of bacterial ring rot. Report/Plant Research International 95—38

Van der Wolf JM, Van Beckhoven JRCM, Hukkanen A, Karjalainen R, Müller P (2005b) Fate of Clavibacter michiganensis ssp. sepedonicus, the causal organism of bacterial ring rot of potato, in weeds and field crops. J Phytopathol 153(6):358–365

Van der Wolf JM, de Haan EG, Kastelein P, Krijger M, de Haas BH, Velvis H, Mendes O, Kooman-Gersmann M, van der Zouwen PS (2017) Virulence of Pectobacterium carotovorum subsp. brasiliense on potato compared with that of other Pectobacterium and Dickeya species under climatic conditions prevailing in the Netherlands. Plant Pathol 66(4):571–583

Van Vaerenbergh J, De Paepe B, Hoedekie A, Van Malderghem C, Zaluga J, De Vos P, Maes M (2016) Natural infection of Clavibacter michiganensis subsp. sepedonicus in tomato (Solanum Lycopersicum). New Dis Rep 33:7–7

Waldee EL (1942) Comparative studies of some peritrichous phytopathogenic bacteria. Retrospective Theses and Dissertations. p 14150. https://lib.dr.iastate.edu/rtd/14150

Waleron M, Misztak A, Waleron M, Franczuk M, Wielgomas B, Waleron K (2018) Transfer of Pectobacterium carotovorum subsp. carotovorum strains isolated from potatoes grown at high altitudes to Pectobacterium peruvianum sp. nov. Syst Appl Microbiol 41(2):85–93

Wallis CM, Munyaneza JE, Chen J, Noye R, Bester G, Buchman JL, Nordgaard J, Hest PV (2015) ‘Candidatus Liberibacter solanacearum’ titers in and infection effects on potato tuber chemistry of promising germplasm exhibiting tolerance to zebra chip disease. Phytopathology 105(12):1573–1584

Wallis CM, Rashad A, Workneh F, Paetzold L, Rush CM (2017) Effects of holding temperatures on the development of zebra chip symptoms, ‘Candidatus Liberibacter solanacearum’ titers, and phenolic levels in ‘Red La Soda’ and ‘Russet Norkotah’ tubers. Am J Potato Res 94(4):334–341

Wang J-F, Ho F-I, Truong HTH, Huang S-M, Balatero CH, Dittapongpitch V, Hidayati N (2013) Identification of major QTLs associated with stable resistance of tomato cultivar ‘Hawaii 7996’ toRalstonia solanacearum. Euphytica 190(2):241–252

Wang J, Haapalainen M, Schott T, Thompson SM, Smith GR, Nissinen AI, Pirhonen M (2017a) Genomic sequence of ‘Candidatus Liberibacter solanacearum’ haplotype C and its comparison with haplotype A and B genomes. PLoS One 12(2):e0171531

Wang N, Pierson EA, Setubal JC, Xu J, Levy JG, Zhang Y, Li J, Rangel LT, Jr JM (2017b) The Candidatus Liberibacter–host interface: insights into pathogenesis mechanisms and disease control. Annu Rev Phytopathol 55:451–482

Wang J, Wang YH, Dai PG, Chen DX, Zhao TC, Li XL, Huang Q (2017c) First report of tobacco bacterial leaf blight caused by Pectobacterium carotovorum subsp. brasiliense in China. Plant Dis 101(5):830–830

Wanner LA (2006) A survey of genetic variation in Streptomyces isolates causing potato common scab in the United States. Phytopathology 96(12):1363–1371

Wanner LA (2007) High proportions of nonpathogenic Streptomyces are associated with common scab-resistant potato lines and less severe disease. Can J Microbiol 53(9):1062–1075

Wanner LA (2009) A patchwork of Streptomyces species isolated from potato common scab lesions in North America. Am J Potato Res 86(4):247–264

Williamson L, Nakaho K, Hudelson B, Allen C (2002) Ralstonia solanacearum race 3, biovar 2 strains isolated from geranium are pathogenic on potato. Plant Dis 86(9):987–991
Winslow C-EA, Jean B, Buchanan RE, Krumwiede C Jr, Rogers LA, Smith GH (1917) The families and genera of the bacteria: preliminary report of the Committee of the Society of American Bacteriologists on characterization and classification of bacterial types. J Bacteriol 2(5):505

Yang FY, Yang DJ, Zhao WQ, Liu DQ, Yu XM (2017) First report of Streptomyces diastatochromogenes causing potato common scab in China. Plant Dis 101(1):243–243

Yao J, Saenkham P, Levy J, Ibanez F, Noroy C, Mendoza A, Huot O, Meyer DF, Tamborindeguy C (2016) Interactions ‘Candidatus Liberibacter solanacearum’—Bactericera cockerelli: haplotype effect on vector fitness and gene expression analyses. Front Cell Infect Microbiol 6:62

Yap M-N, Barak JD, Charkowski AO (2004) Genomic diversity of Erwinia carotovora subsp. carotovora and its correlation with virulence. Appl Environ Microbiol 70(5):3013–3023

Zhang Y, Loria R (2017) Emergence of novel pathogenic Streptomyces species by site-specific accretion and cis-mobilization of Pathogenicity Islands. Mol Plant-Microbe Interact 30(1):72–82

Zhang Y, Bignell DRD, Zuo R, Fan Q, Huguet-Tapia JC, Ding Y, Loria R (2016a) Promiscuous pathogenicity islands and phylogeny of pathogenic Streptomyces spp. Mol Plant-Microbe Interact 29(8):640–650

Zhang Y, Fan Q, Loria R (2016b) A re-evaluation of the taxonomy of phytopathogenic genera Dickeya and Pectobacterium using whole-genome sequencing data. Syst Appl Microbiol 39(4):252–259

Open Access  This chapter is licensed under the terms of the Creative Commons Attribution 4.0 International License (http://creativecommons.org/licenses/by/4.0/), which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license and indicate if changes were made.

The images or other third party material in this chapter are included in the chapter’s Creative Commons license, unless indicated otherwise in a credit line to the material. If material is not included in the chapter’s Creative Commons license and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder.