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INVITED REVIEW

Potato Common Scab: a Review of the Causal Pathogens, Management Practices, Varietal Resistance Screening Methods, and Host Resistance

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Abstract Potato common scab is a widespread disease in which scab-like lesions develop on tubers. The disease is caused by pathogenic *Streptomyces* species, which synthesize the phytotoxin thaxtomin. The *txtAB* operon, responsible for thaxtomin production, can be used as a marker to identify pathogenic strains of the bacterium. Screening methods to assess scab susceptibility in breeding programs are time-consuming and can produce variable results. Management practices to control the disease vary and include crop rotation, tolerant varieties, monitoring soil pH, avoiding low soil moisture at tuber initiation, and application of soil- and/or seed-applied pesticides. There is a wide range in levels of tolerance among potato varieties. Many public research programs are committed to breeding for scab-tolerant varieties and evaluating management methods. Topics reviewed target readers focused on breeding and disease management objectives to reduce the incidence and severity of potato common scab.

Resumen La roña común de la papa es una enfermedad ampliamente distribuida, que desarrolla lesiones tipo pústulas en los tubérculos. La enfermedad es causada por especies patogénicas de *Streptomyces*, que sintetizan la fitotoxina thaxtomin. El operón txtAB, responsable de la producción de thaxtomin, puede usarse como marcador para identificar variantes patogénicas de la bacteria. Los métodos de análisis para evaluar la susceptibilidad a la roña en los programas de mejoramiento se llevan mucho tiempo y pueden producir resultados variables. Las prácticas de manejo para controlar la enfermedad varían, e incluyen rotación de cultivos, variedades tolerantes, monitoreo del pH del suelo, evitando baja humedad en el suelo al principio de la tuberización, y la aplicación de plaguicidas al suelo y/o a la semilla. Existe una gran amplitud de niveles de tolerancia entre las variedades de papa. Muchos programas públicos de investigación están comprometidos al mejoramiento para variedades tolerantes a la roña y en la evaluación de métodos de manejo. Los tópicos revisados conducen a lectores enfocados en objetivos de mejoramiento y manejo de la enfermedad para reducir la incidencia y la severidad de la roña común de la papa.

Keywords *Streptomyces* · *Solanum tuberosum* · Disease resistance

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Introduction

Potato common scab (CS) is caused by the saprophytic, filamentous, gram-positive, soil-borne bacterium *Streptomyces scabies* and other pathogenic *Streptomyces* species (Loria 2001; St-Onge et al. 2008; Hao et al. 2009; Wanner 2009). Symptoms of this disease include scab-like surface, raised, and/or pitted lesions on the tuber (Bukhalid and Loria 1997), which can cause a substantial reduction in marketable yield for potato growers. Requirements for potato grades and state seed certification consider the severity and amount of scab on tuber

lots. The United States Department of Agriculture (USDA) grading requirements consider potato tubers with an aggregate surface area greater than 5% to be damaged. For pitted scab, a potato tuber is considered damaged when removal of lesions causes a loss of more than 5% of the total weight of the tuber or covers the surface in an aggregation of greater than a half inch (1.3 cm). Potato tubers are considered seriously damaged when the removal of tissue affected by pitted scab causes a loss of more than 10% of the total weight of the potato, or when scab affects an aggregate area of greater than one inch (2.5 cm). For surface scab, the tuber is considered to have serious damage when the aggregate surface area is greater than 25%.

It is important to note that of the several hundred bacterial species belonging to the genus *Streptomyces*, only a few are able to infect developing potato tubers and other underground plant structures such as the tap roots of radish, parsnip, carrot, and beet (Goyer and Beaulieu 1997; Stevenson et al. 2001). This point is perhaps of great importance when considering the soil microbial community in our overall understanding of the pathogen and its effects on host plants, as well as management of the disease it causes.

Disease Cycle of *Streptomyces scabies*

Thaxter was the first to describe and isolate the causal agent of CS in North America as *Oospora scabies* (Thaxter 1891). The change in name to *Streptomyces scabies* occurred in 1948, was revived in 1989, but then changed to *S. scabiei* in 1997 to follow grammatical convention (Lambert and Loria 1989a). A 2007 opinion paper published by Saddler et al. requests the approval of continued use of *S. scabies* in reference to the potato pathogenic *S. scabiei*. While no final decision has been made on this taxonomic concern, a rebuttal to Saddler's paper was published in 2008. In this work, we have chosen to use *S. scabies* due to its widely accepted usage especially among the plant breeding and disease management researchers.

Streptomyces scabies is a bacterium that resembles a fungus due to its filamentous morphology. The mycelium is composed of thin (approximately 1 μm diameter), branched hyphae with few or no cross walls. Cylindrical spores (0.5 by 0.9–1.0 μm) are produced in mature spiral chains containing 20 or more spores (Lambert and Loria 1989a). Spores are released from the tip of the hyphae. The bacterium is dispersed by spores and survives on seed, in soil, and in soil water (Agrios 2005). The hydrophobic characteristic of the spores allows them to also be transported by arthropods and nematodes (Loria et al. 2006). The spores germinate and enter plant tissues through wounds, larval feeding sites, stomata, and lenticels (Locci 1994; Agrios 2005). Penetration of tubers by *S. scabies* and other pathogenic *Streptomyces* species is thought to take place though young lenticels, probably because they have not yet formed a layer of protective suberin (Locci 1994). Loria et al. (2003) demonstrated that penetration and growth occurs through the cell walls. Young tubers are most susceptible up until three to four weeks from tuber initiation and will be discussed later in this review (Khatri et al. 2011; Agrios 2005). Thaxtomin, a phytotoxin produced by *Streptomyces* genotypes that cause common scab, may aid penetration of rapidly growing plant cells, such as expanding internodes (Loria et al. 2003). Tegg et al. (2005) demonstrated that thaxtomin is more effective in young, physiologically active tissues, which would include expanding internodes. Studies suggest that thaxtomin results in a compromised cell wall (Fry and Loria 2002; Scheible et al. 2003; Tegg et al. 2005), allowing for penetration. The tuber is the only known tissue type displaying symptoms on potato (Powelson and Rowe 2008). However, Han et al. (2008) demonstrated that *S. scabies* affects the emergence and growth of roots at early stages of development.

When exposed to a tuber, the pathogen grows through the outer few cell layers symplastically and apoplastically (Loria 2001; Agrios 2005). As host cells die, they provide nutrients for the bacterium. Subsequently, the living host cells around the area of the infection divide and produce layers of cork cells that eventually push outward and form a scab lesion on the tuber. Surface (russet scab), raised (erumpent scab), and pitted (pitted scab) lesions may be observed on the same tuber, and can vary in size; lesions may also coalesce to form large scabby areas on a tuber. The types of lesions formed and the extent to which a tuber is covered with lesions appears to be influenced by environmental factors, cultivar susceptibility, and complexities of the soil microbe community, including *Streptomycetes* spp. virulence factor profiles (Locci 1994; Loria et al. 1997; Boucheck-Mechiche et al. 2000b).

Distribution and Morphology of *Streptomyces* Species Pathogenic on Potato

Streptomyces species that cause CS symptoms on potato tubers have a wide host range and can result in stunting in both monocots and dicots, including wheat, corn, beet, parsnip, carrot and radish (Hooker 1949; Locci 1994; Leiner et al. 1996; Goyer and Beaulieu 1997; Wanner 2004). *Streptomyces scabies* is distributed globally, though a recent survey did not find *S. scabies* in Norway (Lambert and Loria 1989a, b; Dees et al. 2013). CS caused by *S. scabies* is most severe in soil with a pH range of 5.5 to 7.5 (Powelson and Rowe 2008). *Streptomyces scabies* forms gray, smooth, melanin-containing spores in spiral chains of 20 or more (Lambert and Loria 1989a).

Streptomyces scabies strains can be distinguished based on physiological characteristics. Most of the strains evaluated by Lambert and Loria (1989a) did not degrade xanthine and were susceptible to 20 μg of streptomycin per ml and 0.5 μg of crystal violet per ml. Melanin is produced on tyrosine agar

and peptone iron agar. Growth of *S. scabies* does not generally occur below pH 5 (Lambert and Loria 1989a). Current evidence suggests that *S. scabies* is a heterogeneous species carrying differing genetic clusters (Goyer and Beaulieu 1997). There is also variation for pathogenicity within *S. scabies* (Goyer and Beaulieu 1997).

Streptomyces acidiscabies is an acid-tolerant species that causes scab on potatoes in acidic soils below pH 5.2 (Lambert and Loria 1989b), such as those found in the northeastern United States (Powelson and Rowe 2008). It has been reported in Japan, China, Korea, the United Kingdom, and North America (Lambert and Loria 1989b; Toth et al. 2001; Song et al. 2004; St-Onge et al. 2008; Thwaites et al. 2010; Zhao et al. 2010; Dees and Wanner 2012). Although CS symptoms due to *S. acidiscabies* are indistinguishable from *S. scabies*, the pathogens are distinguishable in culture (Lambert and Loria 1989b). *Streptomyces acidiscabies* has flexuous spore chains and produces a red or yellow pH-sensitive diffusible pigment. The spore mass color (white to orange red) is growth-medium dependent. Additionally, it grows on agar media at a pH of 4.0, does not use raffinose as a carbon source, and will tolerate higher concentrations (relative to *S. scabies*) of penicillin G, streptomycin, thallium acetate, crystal violet, and oleandomycin (Lambert and Loria 1989b).

Streptomyces turgidiscabies causes scab lesions on potato in eastern Japan, Finland, China, the United Kingdom, Sweden, North America, and Korea (Miyajima et al. 1998; Kim et al. 1999; Kreuze et al. 1999; Lehtonen et al. 2004; Zhao et al. 2008; Wanner 2009; Thwaites et al. 2010; Dees et al. 2012; Dees and Wanner 2012). In culture, this *Streptomyces* species is gray in color and produces flexuous spore chains, with spores that are smooth and cylindrical. Additionally, it does not produce diffusible pigments (including melanin), and does not grow on agar media at pH 4.0. It utilizes raffinose and inulin as carbon sources. *Streptomyces turgidiscabies* is sensitive to streptomycin, penicillin G, polymyxin B, and thallium acetate (Miyajima et al. 1998). *Streptomyces acidiscabies* and *S. turgidiscabies* are each thought to have emerged via a horizontal gene transfer event from *S. scabies* (Bukhalid et al. 1998; Healy et al. 1999).

Other *Streptomyces* species have been reported to cause scab lesions on potato. *S. europaeiscabiei*, causing common or netted scab (depending on the strain), has frequently been reported in North America, Western Europe, and Korea (Boucheck-Mechiche et al. 2000a; Song et al. 2004; Flores-Gonzalez et al. 2008; Wanner 2009; Dees et al. 2012; Dees and Wanner 2012). *Streptomyces stelliscabiei*, causing common scab, has been reported in the United States, France, and South Africa (Boucheck-Mechiche et al. 2000a; Wanner 2009). Park et al. (2003) reported that *S. luridiscabiei*, *S. puneiescabiei*, and *S. niveiscabiei* cause common scab in Korea. In North America, groups *Streptomyces sp. IdahoX* and sp. SD3024 have also been described (Wanner 2007; Hao et al. 2009).

Species Causing Common Scab in North America

A few robust, broad-geographic, sample-intensive surveys of pathogenic *Streptomyces* species resulting in CS have been conducted to provide a comprehensive inventory in North America. Most of the potentially pathogenic species are *Streptomyces scabies* and *S. europaeiscabiei*, followed by *S. stelliscabiei*, and species/strain *S. sp. IdahoX*, then *S. acidiscabies* and *S. turgidiscabies*; others are less common, such as *S. bottropensis* (Wanner 2009). Species distribution in North America is patchy, with the exception of *S. europaeiscabiei*, which is found mainly in the northwest (Alaska, Idaho, Ontario) and *S. sp. IdahoX* in the west. *S. scabies* is predominant in the middle and eastern Midwest (Minnesota, Missouri, Wisconsin, Indiana, and Michigan) as well as Washington, Oregon, and New York. In Maine and Pennsylvania, *S. scabies* and *S. europeiscabiei* are found in approximately equal proportions. *Streptomyces stelliscabiei* has been reported to predominate in some field locations, but not in any particular region. Species distribution is variable on a local and regional basis, where a single species will often predominate in a field that may differ from neighboring fields (Wanner 2009). An array of *S. scabies* strains can be present in a single field within a year. Moreover, the strain profiles within a field may shift from year to year (Wanner 2008). The lack of a pattern for geographical distribution has also been reported in Norway for *S. europaeiscabiei* and *S. turgidiscabies*, where both could be found in the same field and lesion (Dees et al. 2013). Since potato *Streptomyces* pathogens can be introduced into new fields on seed potatoes, field populations may not be stable over time.

Lesions containing more than one species have also been reported (Lehtonen et al. 2004; Wanner 2009). Non-pathogenic isolates from scabby tubers have been reported for *S. scabies*, *S. acidiscabies*, and *S. turgidiscabies* (Faucher et al. 1992; Lindholm et al. 1997; Wanner 2009). The presence of non-pathogenic isolates in scabby tubers combined with the possible diversity of pathogenic isolates within a field speaks to the complexity faced by growers in reducing the incidence and severity of the disease as well as the challenges to researchers as they breed for resistant clones and optimize management methods.

Thaxtomin, a Pathogenicity Factor

Pathogenic *Streptomyces* species synthesize the phytotoxin thaxtomin, which elicits scab symptoms (King and Lawrence 1989; Lawrence et al. 1990; King et al. 1992), and is the primary pathogenicity determinant in common scab-causing species (Lawrence et al. 1990; Healy et al. 2000). Although the target of thaxtomin is unknown, the compound inhibits cellulose biosynthesis. Arabidopsis seedlings treated with thaxtomin exhibit reduced accumulation of

crystalline cellulose in cell walls (Scheible et al. 2003) and decreased concentrations of cellulose synthases in the plasma membrane (Bischoff et al. 2009). Thaxtomin production is induced by plant-derived compounds, including cellobiose, formed by the partial hydrolysis of cellulose, and suberin, present on the surface of potato tubers (Beauséjour et al. 1999; Lerat et al. 2009). Thaxtomin is a nitrated dipeptide composed of phenylalanine and tryptophan and was first isolated and identified by King and Lawrence (1989). The predominant phytotoxin produced by *S. scabies* is thaxtomin A, followed by thaxtomin B (Lawrence et al. 1990; King et al. 1991, 1992). A study by Kinkel et al. (1998) reported a positive correlation between symptom expression and thaxtomin A production in culture, where each 1 μg/mL increase in thaxtomin A corresponded to an 11% increase in potato tuber surface area infected.

Thaxtomin A induces plant cell hypertrophy in expanding tissues (Fry and Loria 2002; Scheible et al. 2003), resulting in a reduction in seedling growth (Leiner et al. 1996; Scheible et al. 2003). Thaxtomin also results in an increased intracellular Ca^{2+} concentration, leading to cell wall acidification and a compromised cell wall (Tegg et al. 2005). This Ca^{2+} increase is necessary to trigger thaxtomin induced programmed cell death (PCD) in Arabidopsis cell suspensions (Errakhi et al. 2008). Unlike most PCD pathways in plant cells, this one does not involve typical defense responses such as the expression of defense-related genes, production of reactive oxygen species, and induction of the hypersensitive response (Duval et al. 2005).

Genes for the biosynthesis of thaxtomin are located on a pathogenicity island (PAI) that is conserved and mobile (Bukhalid and Loria 1997; Kers et al. 2005; Loria et al. 2006). The PAI is 660 kb in *S. turgidiscabie*s and can be transferred from pathogenic *Streptomyces* to nonpathogenic *Streptomyces* via conjugation. In *S. scabies*, the pathogenicity island is split into two distinct regions, a 'toxicogenic region' containing genes for thaxtomin biosynthesis and a 'colonization region' which contains the *nec1* and *tomA* genes that contribute to virulence (Kers et al. 2005; Lerat et al. 2009). These regions are 55 and 110 kb, respectively. The genes for thaxtomin biosynthesis include nitric oxide synthase (NOS), encoded by *txtD* (Kers et al. 2004), and *txtE*, a P450 monooxygenase (Barry et al. 2012). Genes *txtA* and *txtB* (*txtAB*) encode non-ribosomal peptide synthetases (Healy et al. 2000), that produce a dipeptide from phenylalanine and the nitrated tryptophan. The dipeptide is then hydroxylated by cytochrome P450 monooxygenase, encoded by *txtC* (Healy et al. 2002). Thaxtomin biosynthesis is regulated by the TxtR protein (encoded by *txtR*), a member of the AraC/XylS family of transcriptional regulators and is embedded in the biosynthetic pathway of thaxtomin (Joshi et al. 2007). A figure of thaxtomin biosynthesis may be found in Barry et al. 2012.

The gene *nec1* encodes a novel protein that induces necrosis in plant tissue (Bukhalid and Loria 1997) and contributes to virulence (Bukhalid et al. 1998; Joshi et al. 2007), but is not required for pathogenicity (Wanner 2009). Cloning *nec1* into the nonpathogenic *Streptomyces lividans* is sufficient to allow colonization and necrosis of potato tuber disks (Bukhalid et al. 1998). Since the Nec1 protein is produced prior to thaxtomin synthesis, it has been suggested that *nec1* suppresses plant defenses stimulated by thaxtomin (Joshi et al. 2007).

The *tomA* gene homolog is responsible for synthesizing a tomatinase enzyme that has been identified in phytopathogenic fungi and targets the antimicrobial glycoalkaloid tomatine as a possible way to disarm host response to infection (Kers et al. 2005; Seipke and Loria 2008). Tomatinases are common in plant pathogenic fungi (Morrissey and Osbourn 1999) and have been found in the plant pathogenic bacterium *Clavibacter michiganensis* subsp. *michiganensis* (Kaup et al. 2005). While the genes *nec1* and *tomA* are not essential for disease (Seipke and Loria 2008; Wanner 2009), they do contribute to virulence (Lerat et al. 2009).

Scheible et al. (2003) have identified an Arabidopsis mutant resistant to thaxtomin, where resistance is due to a decreased rate of thaxtomin uptake. The *TXR1* gene is thought to be a regulator of thaxtomin transport and plays a required role in normal root growth at increased temperatures and normal shoot growth at all temperatures (Scheible et al. 2003). The region identified in this study may be helpful for breeders to evaluate quantitative trait loci (QTL) associated with resistance. Breeding for resistance to common scab is discussed later in this review.

Pathogenic Strain Identification

Conventional polymerase chain reaction (PCR) assays based on 16S rRNA genes can be used to detect and distinguish among most species of *Streptomyces* isolated from CS symptomatic tubers (Lehtonen et al. 2004; Wanner 2009). With this assay, there is high sequence similarity between *S. scabies* and *S. europascabiei.* To distinguish, the internal transcribed spacer region of the 16S operon must also be amplified and digested with the restriction enzyme *Hpy99I* according to the methods described by Flores-Gonzalez et al. (2008) and Song et al. (2004). Type strains with the restriction site present (*Hpy99I*+) include *S. scabies*, while strains without (*Hpy99I*-) are indicative of *S. europascabiei*.

Species designations do not indicate pathogenicity because there may be both pathogenic and nonpathogenic strains of a species (Wanner 2006). The pathogenicity of a strain can be confirmed by the presence of thaxtomin (Lawrence et al. 1990; Leiner et al. 1996; Healy et al. 2000; Fry and Loria 2002). The detection of *txtAB* was perfectly correlated with pathogenicity in more than 100 isolates (Wanner 2006, 2007).

The *txtAB* operon has been used as a marker to determine pathogenic species (Wanner 2006, 2007; Flores-Gonzalez et al. 2008; Qu et al. 2008, 2011), to quantify pathogenic species in soil and tubers using a real-time polymerase chain reaction assay (Qu et al. 2008), and to distinguish between symptoms due to pathogenic *Streptomyces* species and those caused by the powdery scab pathogen in potato (Qu et al. 2011). Powdery scab (PS), while similar in disease name, is caused by *Spongospora subterranea*, a soilborne fungus-like protozoan that produces disease symptoms that may be confused with CS. PS lesions are tan, blister-like swellings that may rupture and result in outgrowths that look like CS lesions.

Exposure of susceptible seedlings to putative *Streptomycete* pathogens can also provide a pathogenic screening approach for isolates in question (Flores-Gonzalez et al. 2008; Dees et al. 2013). Such assays have been used to support molecular and biochemical thaxtomin tests and continue to provide a phenotypic validation of genotypic investigations.

Relationship between Disease Severity and Inoculum Levels

Under greenhouse conditions, CS severity generally increases with an increase in inoculum concentration. However, the slope of the regression line is affected by cultivar resistance and strain virulence (Keinanth and Loria 1991). An increase in the population of *S. scabies* in the rhizosphere may be dependent on the strains tested and the inoculum density. Keinanth and Loria (1991) reported an increase in the rhizosphere population of *S. scabies* with an increase in the initial soil population by assaying four inoculum levels of two different pathogenic strains. In contrast, (Ryan and Kinkel 1997) found no significant increase in pathogen populations in the rhizosphere by assaying with an inoculum source that contained five different *S. scabies* strains and higher population densities than that used by Keinath and Loria. As suggested by Ryan and Kinkel (1997), the higher population densities may have inhibited *S. scabies* reproduction. Interestingly, the rhizosphere population of *S. scabies* is not consistently related to the severity, incidence, strain virulence, or cultivar resistance in the greenhouse (Keinanth and Loria 1991) and are topics of research for future studies. It is important to note that the success of inoculation in greenhouse studies and for both antagonistic and pathogenic strains is dependent on the application technique. For example, Keinanth and Loria (1991) prepared inoculum in liquid media, whereas Ryan and Kinkel (1997) cultivated inoculated species with vermiculite. Additional assays and scoring methods for common scab are described later in this review.

Increased CS is also correlated with increased concentrations of pathogenic *Streptomyces* in the field. Specifically, the percent of tubers with CS increased as *nec1* copy number increased (Manome et al. 2008; Koyama et al. 2006). Using real-time PCR of *txtAB*, Qu et al. (2008) found that fields where CS occurred have a concentration of 10^3–10^6 colony forming units (CFU) of *Streptomyces* per gram of soil. However, the presence of *txtAB* was not correlated with scab incidence. Conn et al. (1998) isolated *Streptomyces* spp. from two potato fields in Oklahoma, and found that approximately 4 % of the species produced thaxtomin, corresponding to approximately 20,000 CFU of pathogenic *Streptomyces* per gram of soil. Neither Koyama et al. (2006) nor Qu et al. (2008) found evidence of pathogenic *Streptomyces* in fields where CS did not occur.

Tuber Periderm and Infection Period

Tubers are most susceptible to infection just after tuber initiation up until three to four weeks after tuber initiation (Khatri et al. 2011). Specifically, Khatri et al. (2011) reported a higher percentage of infected tubers (68%) and percent surface area (11.7%) when inoculum was added at two weeks after tuber initiation compared to just 4% infection and area (0.004%) when inoculum was added at eight weeks after tuber initiation. The mean lesion depth was greatest on tubers that were infected at two to three weeks after initiation of tuberization, in contrast to tubers that were inoculated at six to eight weeks, which resulted in mostly superficial lesions (Khatri et al. 2011). Tuber internodes that are formed first are more susceptible to infection than internodes that are formed later (Adams 1975; Khatri et al. 2010, 2011). The first internodes formed have the greatest amount of time to expand, resulting in more severe lesions when exposed to *S. scabies* (Lapwood et al. 1970).

Exposure to *S. scabies*, or other potato-pathogenic *Streptomyces* species, results in an increased number of cell layers and thickness of the phellem in both tolerant and susceptible genotypes when observed at harvest. In the presence of the pathogen, the thickness of the phellem, the number of cell layers, and suberin content are greater at 10 days after tuber initiation (Khatri et al. 2011). The thickening occurs in response to inhibition of the biosynthesis of cellulose in developing tuber cells by thaxtomin A (King and Lawrence 1989; Loria et al. 2006; Bischoff et al. 2009; King and Calhoun 2009; Khatri et al. 2011).

A study by Khatri et al. (2011) reported that exposure of a susceptible cultivar (Desiree) to the pathogen results in increased suberin deposition, phellum thickness, and number of cell layers in the phellem. An increased deposition of suberin can be found in tubers that have been exposed at seven or 21 days after tuber initiation (28% and 38% higher, respectively) when assessed seven days after exposure to *S. scabies*. Desiree also has significantly increased phellem thickness and number of cell layers three weeks after exposure to *S. scabies* when inoculated at seven days after tuber initiation. When

inoculated at 21 days after tuberization, there is a significant increase in phellem thickness and number of cell layers at two to three weeks after exposure. Desiree tubers that are exposed to the pathogen seven days after tuber initiation are more susceptible to CS (27% of tubers with disease) than tubers inoculated 21 days after tuber initiation (<10%). Future studies that characterize the pathogen population within a field over time accounting for virulence genes and phenotype of the developing tuber, including the development of phellem, may provide valuable insight for growers and breeders in developing management responses.

Resistance Scoring Methods

A number of assays have been developed to screen for common scab resistance or to study host-pathogen interactions. The standard methods are typically field and greenhouse assays. In the field assay, potatoes are grown in an infested field until they senescence, and then tubers are harvested and scored for scab symptoms (Langton 1972; Marais and Vorster 1988; Haynes et al. 2010; Sedlákova et al. 2013). This assay has been adapted for use in greenhouses and growth chambers in order to better control environmental conditions and both inoculum dose and composition (Bjor and Roer 1980; Marais and Vorster 1988; Boucheck-Mechiche et al. 2000b; Wanner and Haynes 2009). In these assays, potatoes are planted in potting medium that has been inoculated or in naturally infested soil.

Other screening assays include a soil-less, nutrient film hydroponic method utilized by Khatri et al. (2010) which allows the nondestructive visualization of tuber and symptom development. In this method, tubers were sprayed with a *S. scabies* spore suspension and harvested at plant senescence, at which time symptoms were noted. Inoculating tubers between three and twenty days after tuber initiation produced good symptoms. Khatri et al. (2011) also developed a novel pot system that used a screen to spatially separate roots and stolons, allowing for nondestructive examination and inoculation of tubers. Of these two methods, the novel pot system resulted in more infection than the hydroponic method (Khatri et al. 2011).

Hiltunen et al. (2011) created an assay to eliminate only the most susceptible genotypes by scoring seedlings inoculated with thaxtomin A and validating it using field and greenhouse assays. Cuttings from 120 seedlings were placed in tissue culture media containing thaxtomin A. After four weeks, plant height and number of roots were recorded. Eight highly sensitive and eight highly tolerant plants were selected. These plants were screened using one greenhouse assay and field assays in three separate fields. Shoot height from the in vitro assay was significantly correlated with the scab index obtained in the greenhouse and field assays.

There are a number of different methods for measuring CS disease severity. These include assessing surface area affected, lesion type, and percent of tubers infected (Leach et al. 1938; Caligari and Wastie 1985; Haynes et al. 1997; Driscoll et al. 2009; Wanner and Haynes 2009; Khatri et al. 2011). Percent surface area is visually determined on a scale of 0 to 100%. Lesion type may be scored on a scale of 0 to 5, based on whether the lesion is superficial, raised, or pitted and whether lesions are discrete or coalescing (Wanner and Haynes 2009). In general, tubers with a smaller lesion-covered surface area have a less severe lesion type (Leach et al. 1938). Surface area and lesion type correlation coefficients are positive and range from 0.30 to 0.85 (Bjor and Roer 1980; Lambert et al. 2006).

The assay and scoring method can affect the sensitivity of screening results. Leach et al. (1938) found that scoring each tuber separately and then averaging the scores of all tubers in the hill was more effective than scoring and averaging only the largest or most diseased tubers. Goth et al. (1993) reported that a cluster analysis on the combination of percent surface area and lesion type is superior to an overall scab index for distinguishing between resistant and susceptible potato clones. A cluster index for these two traits provides an efficient method to classify relative resistance of the tested clone to known cultivar standards (Goth et al. 1993). Caligari and Wastie (1985) reported a reduction in error variance and wider variation among cultivars when tubers were grown in naturally infested sandy soil diluted to 50% in the greenhouse than when using undiluted soil. Pasco et al. (2005) obtained a reliable cultivar response using a sterile soil mixture composed of one part sand and two parts peat. In this same assay, a range in aggressiveness among different isolates was detected.

Screens for CS resistance in the greenhouse and growth chamber have many sources of variation to consider. Within-greenhouse pot variation may equal the between-pot and between-replicate experiment variation when considering both lesion type and percent surface area, despite efforts to inoculate with a single isolate, control the inoculum density and maintain a controlled environment in a growth chamber (Wanner and Haynes 2009). Control measures in experiments such as using standard sets of clones, adequate replication, and rank by pathogen assessment are important parameters to evaluate the results due to the challenges listed above in the greenhouse and in field studies.

There are also a number of assays to test for the pathogenicity of *Streptomyces* strains. An assay based on mini tubers obtained by the leaf-bud protocol developed by Lauer (1977), has been used in several studies to assess pathogenicity of *Streptomyces* species (Babcock et al. 1993; Lorang et al. 1995; Lindholm et al. 1997; Kinkel et al. 1998). In these assays, mini-tubers produced from cuttings are inoculated with spore suspensions or liquid culture and evaluated for CS after a few days to three weeks. In a seedling assay, radish seeds are grown on one week-old *Streptomyces* plates and

evaluated for stunting and necrosis after about a week (Flores-Gonzalez et al. 2008; Dees et al. 2013). However, these methods have not been used to screen potatoes for resistance to common scab.

Conditions Conducive to Disease

Streptomyces species that are pathogenic on potato can potentially cause lesions on the tubers of susceptible genotypes given the occurrence of favorable conditions including a pH higher than 5.2, temperatures of 20–22 °C, and a soil moisture below field capacity during early tuberization (Archuleta and Easton 1981). Water develops a microfilm around the developing tuber to limit infection (Gudmestad 2008). *Streptomyces scabies* will generally cause disease in soils with less than 65–70% soil moisture (Gudmestad 2008). As a management technique, growers can maintain soil moisture in the 80–85% range from tuber initiation until tubers are 25 to 38 mm in diameter (Davis et al. 1976; Gudmestad 2008; Powelson and Rowe 2008). Environmental conditions may further influence the types of lesions observed (shallow, raised, or pitted) (Goyer et al. 1996). However, lesion type has also been associated with pathogen species and/or the presence of one or more virulence factors (Boucheck-Mechiche et al. 2000b). Multiple species and/or strains of *Streptomyces* may be present within the tuber sphere microbial community, making it difficult to discern tight associations among these factors.

Environmental factors that are conducive to CS caused by *S. scabies* include low soil moisture during tuber initiation, daytime temperatures above 70 °F, and a soil pH range of 5.5 to 7.5. The severity of scab lesions increases as the soil pH increases from pH 5.2 to 8.0. Above pH 8.0, scab severity decreases again (Agrios 2005). The recommendation for potato growers is to reduce soil pH to 5.2 or below (reviewed by Locci 1994). Sulfur, acid-forming fertilizers, and gypsum applied before or at planting reduces the soil pH to between 5.0 and 5.2, which helps to suppress CS caused by *S. scabies* (Locci 1994; Powelson and Rowe 2008). Further, growers are encouraged to carefully monitor and manage soil moisture to field capacity during early tuberization in fields with a history of common scab or in fields planted to highly susceptible potato cultivars with the potential for carrying seed-introduced inoculum.

Management of Common Scab

The management of common scab requires an integrated approach including cultural and, in some instances, chemical considerations. In this section, we review the influence of green manures, crop rotation, soil moisture management, and chemical and biological inputs on common scab.

Green manures that inhibit *S. scabies* and other pathogenic species include buckwheat, canola, fallow, oat, rye, and millet (Kinkel 2008; Powelson and Rowe 2008). Soil incorporated red clover and animal manure, such as poultry manure, which increases soil pH, may exacerbate CS symptoms. Pulp and municipality wastes have been shown to reduce the severity of CS (Powelson and Rowe 2008). Increased microbial activity is thought to suppress *S. scabies* in the soil. However, the genera of active microbes are not well understood and have yet to be harnessed and broadly utilized in commercial field management of CS (Kinkel 2008).

A somewhat effective management practice for reducing CS is a disease-suppressive rotation, such as that of a three year, mustard/rapeseed – rye/sudangrass – potato (Larkin et al. 2011). Larkin et al. (2011) found that this rotation, although difficult to carry out in practice due to economic constraints, reduced CS in all three years under irrigated and non-irrigated conditions by 25–40%. Other cropping systems that reduced scab in some years were a two-year rotation of barley/clover-potato and a three year rotation of barley/timothy-timothy-potato with an additional year of forage, limited tillage, and straw mulch after potato. Compost amendments to the latter rotation were not as effective at reducing disease levels.

Interestingly, soils suppressive to scab are also those that have supported potato monoculture for several years (Menzies 1959; Lorang et al. 1989). Microbial communities have been noted to have site-specific characteristics in soil and tuber periderm. Moreover, soils with relatively less C, N, Ca, and Fe have been reported to lower common scab severity. The quality of soil organic matter is also related to disease suppression (Sagova-Mareckova et al. 2015). Several studies have reported disease suppression on potato due to the presence of nonpathogenic *Streptomyces* species, as well as presence of bacilli and fluorescent pseudomonads (Liu et al. 1995; Lorang et al. 1995; Bowers et al. 1996; Wanner 2007; Hiltunen et al. 2008; Meng et al. 2012).

While the use of host resistance in an integrated disease management program is foundational, common scab resistance in potato has been challenging to identify and develop in commercial cultivars. Manipulation of environmental conditions, and implementation of non-host crop rotation, in addition to adoption of cultural practices, such as reducing soil pH and managing soil moisture, have only a seemingly partial impact on common scab. In the absence of host resistance, crop protection inputs become necessary to produce a healthy and marketable crop. Chemical and antimicrobial treatments have been used with varying levels of success. Historically, formaldehyde, urea formaldehyde, and manganese sulphate have also been used for common scab control, but these treatments are no longer applied in production systems (Locci 1994). Because CS is not caused by a fungus, the disease is not typically managed through seed-applied fungicides. However, seed treatment with mancozeb-containing fungicides can aid in limiting other seed-borne or soil-borne

pathogens that cause diseases such as *Rhizoctonia*, *Fusarium*, and *Phytophthora infestans*, which can influence seed health, emergence, and overall crop stand/vigor (Gullino et al. 2010). In a 2-year Canadian study, seed-applied fludioxonil resulted in a 57.8% reduction of common scab severity, and use of a seed-applied biopesticide containing *Bacillus subtilis* resulted in a 56.1% reduction (Al-Mughrabi et al. 2016). Some production regions have adopted pentachloronitrobenzene (PCNB) (common name Quintozene, trade name Blocker, AMVAC), an in-furrow soil treatment, to suppress CS (Davis et al. 1976; Powelson and Rowe 2008). Chloropicrin, a broad-spectrum soil biocidal fumigant, has had some usage in U.S. potato production systems to improve control of soilborne diseases, including common scab, in select fields with histories of high pressure (Webster et al. 2013). While we have addressed just a few of the chemical and biological inputs tested for common scab in this review, a growing body of research has been amassed which provides extensive information on effectiveness of various soil amendments, seed-applied, in-furrow applied, and foliar-applied treatments from many locations, over many years. Unfortunately, few to no treatments provide highly effective and reliable control of the disease across locations (Powelson and Rowe 2008; Lerat et al. 2009).

Sources of Resistance

Host plant resistance is considered to be one of the best options for managing CS (Mishra and Srivastava 2001; Jansky and Rouse 2003; Pasco et al. 2005; Wanner and Haynes 2009). In the past, a few German cultivars have been reported to be resistant to common scab, including Charlotte, Richter's Jubel and Hindenburg (Dionne and Lawrence 1961; Pasco et al. 2005). Resistance has also been reported in the Dutch cultivars Sirtema and Monalisa, and the French cultivar Belle de Fontenay (Pasco et al. 2005). In addition, some historic russet cultivars, such as Russet Burbank and Russet Rural are considered resistant (Darling 1937). In recent decades, breeding for resistance to common scab has become a priority for North American potato breeders. Their efforts have paid off. New cultivars with resistance include Alta Crown, Freedom Russet, GemStar Russet, Kalkaska, Liberator, Marcy, McBride, Megachip, Millenium Russet, Owyhee Russet, Premier Russet, Summit Russet, Teton Russet, and Western Russet (Douches et al. 2001, 2009; Love et al. 2005, 2006a, b; De Jong et al. 2006; Groza et al. 2005, 2007, 2009; Novy et al. 2014; Novy et al. 2008; Bizimungu et al. 2011; Yilma et al. 2012). Despite these advances, breeders still place a high priority on breeding for resistance to CS, especially in combination with other important traits such as processing quality.

Wild relatives of potato provide another potential source of novel germplasm for resistance for CS. Wild *Solanum* species have made important contributions to cultivar development (Maxted et al. 2012). Moreover, most wild species can hybridize with the cultivated potato (Hanneman 1989; Camadro 2010). Hosaka et al. (2000) screened one hundred accessions of 18 wild diploid potato species and selected several (322) resistant genotypes. The wild species *S. bukasovii*, *S. canasense*, and *S. multidissectum* produced the most resistant clones. Resistance has also been identified in the cultivated diploid *S. tuberosum* Phureja Group (Alam 1972; De Maine et al. 1993), dihaploids of *S. tuberosum* (Cipar and Lawrence 1972) and the wild species *S. chacoense* (Dionne and Lawrence 1961; Braun 2013).

Somatic cell selection in the cultivar Iwa using thaxtomin A produced several potato clones with improved resistance to common scab (Wilson et al. 2009). However, in leaf and tuber bioassays, the resistant plants were not consistent in their response to thaxtomin A. Consequently, resistance was not likely the result of selection for tolerance to the phytotoxin. In a follow-up study, however, Wilson et al. (2010) reported that thaxtomin A tolerance was associated with disease resistance in somatic variants isolated from the cultivar Russet Burbank.

Mechanisms of Resistance

Despite decades of research on scab resistance in potato, little is known about the mechanisms of resistance. One possible mechanism is physical, with the production of barriers to pathogen penetration. Phellum suberization was reported to be greater in the tolerant cultivar Russet Burbank than the susceptible cultivar Desiree, as discussed earlier (Khatri et al. 2011). In addition, Russet Burbank had more cell layers in tuber phellum and they were thicker than in Desiree. Similarly, Thangavel et al. (2016) found that somaclones selected for resistance to thaxtomin A had more phellum cell layers with more suberin polyphenols than those that were not resistant. Lenticels may be another structural feature that contribute to scab resistance. Darling (1937) reported that the lenticels of susceptible clones are larger and more loosely arranged than those of resistant clones (Darling 1937; Atiq et al. 2013).

Another potential resistance mechanism is chemical, through the detoxification of thaxtomin A. This can be accomplished through glucosylation. The resistant cultivar Nooksack was found to have higher levels of glucose transferase activity than susceptible Ranger Russet (Acuna et al. 2001). Apparently, scab resistant tubers were able to convert thaxtomin to the glucose conjugate of thaxtomin, which is a less toxic form. In contrast, Tegg and Wilson (2010) found no relationship between resistance to CS and tolerance to thaxtomin A. While Russet Burbank and Atlantic are moderately resistant to CS, only Atlantic was tolerant to thaxtomin A. In contrast, both Bismarck and Tasman are susceptible to CS but tolerant of thaxtomin A.

A few studies have evaluated gene expression in response to pathogen infection. Tai et al. (2013) reported a correlation between scab resistance and the expression of two *MYB* and three *bHLH* transcription factor genes. The expression of one *MYB* gene increased when plants were grown in a location with high scab pressure. The other genes were constitutively expressed and may be involved in defense via plant growth hormone regulation. In another study, a resistant cultivar was able to mount an early defense that was sustained through early tuber development, whereas the defense system failed to sustain itself in the susceptible cultivar (Dees et al. 2016). In addition, an isoform of the *TXR1* (thaxtomin resistance-1) gene was present in the resistant but not the susceptible cultivar. Alternative splicing of up-regulated genes in infected plants was higher than in uninfected controls, indicating that this form of gene regulation is an important component of the defense response system.

It is interesting to note that the composition of *S. scabies* genotypes on scab lesions varies depending on the resistance level of the host. Resistant clones produce a higher proportion of nonpathogenic isolates than susceptible clones (Wanner 2007). There are several possible explanations for this observation. The resistant potato clones may limit the growth of pathogenic *S. scabies* genotypes or provide a favorable environment for nonpathogenic ones, which then outcompete the pathogenic types. On the other hand, susceptible potato clones may provide a better environment for the establishment or growth of the pathogenic genotypes.

Genetics of Resistance

To date, major genes for scab resistance have not been identified or mapped. However, based on a study of transmission of scab resistance from diploid clones to tetraploids via sexual polyploidization, Murphy et al. (1995) suggested that scab resistance is simply inherited. The strongest source of resistance was a Phureja group clone. Alam (1972) also suggested that a major dominant gene is responsible for resistance in populations of hybrids between *S. tuberosum* dihaploids and Phureja Group clones. He noted that a recessive second gene is also required for the dominant gene to confer resistance. Braun (2013) identified a single major quantitative trait locus for scab resistance in a diploid F2 population derived from a cross between *S. tuberosum* and *S. chacoense*. Pasco et al. (2005) suggested that qualitative resistance to netted scab caused by *S. reticuliscabiei* exists in some European cultivars.

Most literature treats scab resistance as a quantitative trait. These studies are often based on tetraploid populations, in which the detection of major genes is difficult. The pathogen species may also influence the outcomes of genetic studies. Pasco et al. (2005) suggested that resistance to some isolates is quantitative, while resistance to others is qualitative. Broad-sense heritability (h^2) is the ratio of genotypic variance (σ^2_g) to phenotypic variance (σ^2_{ph}). The genotypic variance is comprised of the sum of the dominance (σ^2_D), additive (σ^2_A), and epistatic (σ^2_I) variances. In short, it is the variation due to genetic differences among individuals. The phenotypic variance (σ^2_{ph}) is comprised of the sum of the variance due to the genotypic variance (σ^2_g), experimental error (environmental variance) (σ^2_e), and genotype by environment interaction (σ^2_{gxe}), where genotype performance varies from environment to environment. Narrow-sense heritability is a measure of additive variance. Bradshaw et al. (2008) reported a moderate heritability estimate (0.66) in a tetraploid family. In another study of a segregating tetraploid population, broad sense heritability for incidence of pitted scab in a field with low pathogen pressure was low (0.34), but it was higher with more pressure (0.64) (Zorrilla et al. 2014). The number of pitted lesions per tuber was also high (0.77) in this study in the field with high disease pressure. Similarly, in another study of tetraploid breeding lines, Navarro et al. (2015) reported a broad sense heritability estimate of 0.83 in trials dedicated to common scab screening, with high disease pressure. In contrast, in trials on conventional fields, heritability was much lower (0.53). Estimates of broad-sense and narrow sense heritabilities were high in a set of tetraploid varieties, with values of 0.89 and 0.86, respectively (Haynes et al. 1997). In a collection of advanced genotypes from public potato breeding programs in the United States, Haynes et al. (2010) estimated broad-sense heritability for CS to range from 0 to 0.78 for percent surface area, 0.49 to 0.90 for lesion type, and 0.30 to 0.80 for proportion of scabby tubers. In contrast, Haynes et al. (2009) reported a low broad sense heritability estimate (0.18) for CS resistance in a diploid *Solanum phureja-S. stenotomum* potato population. The large differences in reported heritability values across studies may be due to a number of factors, including differences in screening methodologies, genetic differences between the populations, and differences in breeding strategies that resulted in the populations.

Breeding for Resistance

Several studies have been committed to developing germplasm with enhanced resistance to CS (Bjor and Roer 1980; Caligari and Wastie 1985; Bradshaw et al. 2008; Haynes et al. 2009). As discussed above, a two gene model for resistance to CS has been proposed in studies of diploids. In contrast, complexities of tetraploid inheritance have not allowed similar models to be proposed in studies using cultivars and advanced breeding clones (Dionne and Lawrence 1961; Goth et al. 1993; Haynes et al. 2010; Dees and Wanner 2012). Future breeding efforts may be based on the development of diploid cultivars (Jansky et al. 2016), allowing diploid resistant germplasm to be used as parents in breeding programs. If it is

necessary to transfer resistance from the diploid to the tetraploid level, then sexual polyploidization appears to be effective (Murphy et al. 1995).

Due to the intensity of labor required for screening for CS, the use of marker-assisted selection (MAS) would likely lead to significant gains in selection for resistance. One method to initiate marker development is quantitative trait loci (QTL) analysis. Bradshaw et al. (2008) identified two QTLs for CS in a *S. tuberosum* tetraploid population. As discussed above in a diploid population derived from a cross between a susceptible *S. tuberosum* dihaploid clone and a resistant *S. chacoense* clone, Braun (2013) identified a significant QTL on chromosome 11 for both percent surface area covered by scab lesions and lesion type.

Conclusions

Potato common scab has long been a concern for growers, scientists, breeders, and industry. A number of studies have addressed the causal agents of the disease (*Streptomyces* sp.), a main virulence factor (thaxtomin A) and genes for biosynthesis of the phytotoxin, management practices to mitigate the risk of common scab, and breeding techniques utilizing a range of phenotype procedures. Although these studies have significantly contributed to a more comprehensive understanding of the disease, there are several opportunities for research that may ultimately lead to a reduced risk of failure for growers to meet the USDA grading requirements.

One avenue to pursue is a method of quantification that utilizes a high-throughput phenotyping method that captures all sides of the potato tuber rating both surface area and lesion severity. Such a method would allow for increased replication in the field and greenhouse studies, likely increasing heritability of the trait by reducing phenotypic variance and error and increasing the probability of discovering native traits that result in resistance to the disease. The benefits would also include an even more robust correlation analysis between field and greenhouse assays. Moreover, this quantification method would allow for high-throughput analyses that evaluate grower practice methods and applications that may reduce disease incidence and severity.

Germplasm and population offspring evaluation are also important to identify and improve resistance in varieties available to growers. Programs dedicated to evaluating management methods are important for providing information that help growers manage the disease.

Further studies that elucidate population structure at critical time points and change over time of pathogenic *Streptomyces* species, level of influence from the wider microbial community, and distribution would also be valuable for understanding the context of experimental design to evaluate management practices and germplasm. Such studies from the scientific community would ultimately have an impact on the steps toward success in reducing the incidence and severity of this disease for potato growers and industry stakeholders.

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