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The Search for Integrated Management of Common Scab

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Although common scab (CS) was first described in the literature more than 100 years ago, it continues to be a significant world-wide problem, especially in potato production. Growers report serious CS in fields not formerly affected by the disease, and on cultivars that were thought to be resistant. Worse, most popular potato cultivars are quite susceptible to CS. Frustratingly, CS severity is variable (and unpredictable) from year to year and location to location. Traditional control measures are insufficient and often fail, including reducing soil pH and keeping plants well-irrigated from the time of tuber initiation through early development. Why is this? What don't we understand about this disease that could help in devising better control strategies? And why don't we have more resistant cultivars?

The disease

CS is characterized by superficial, raised, or deeply pitted corky lesions on potato tubers and other root and tuber crops (see Figure, left panels). Lesions detract from the marketability of the crop. Shallow or raised lesions can be removed by peeling during processing, but in the case of deep pitted lesions, peeling losses can be substantial and can result in rejection of the shipment. Since potato processing operations prefer to leave the skin intact, even superficial or raised lesions can present a problem. If CS lesions cover more than 5% of the potato tuber surface, potatoes fail to make U.S. No. 1 grade (USDA 1991).

Researchers have reported that some European scab-causing species produce different symptoms, and also differentially affect potato cultivars. *S. reticuliscabiei* causes netted scab, which affects all underground parts of potato plants including roots, though only on a few potato cultivars are affected (Bouchek-Mechiche et al. 2000; Pasco et al. 2005). Different scab pathogens are apparently responsible for sweet potato scab (Clark et al. 1998).

Host range

Streptomyces is a broad host range pathogen. Numerous root and tuber crops are hosts for the disease, including potato, carrot, beet, radish, and even peanut (Goyer and Beaulieu 1997; Loria et al. 2006). CS on potato has the greatest economic impact world-wide.

The pathogen

CS is caused by gram-positive soil bacteria (actinomycetes) in the genus *Streptomyces*. *Streptomyces* is a huge genus with many soil-inhabiting species, most of which do not cause plant disease. Most *Streptomyces* species in soil are saprophytes and decomposers. They are best known as the source of many medically important antibiotics including streptomycin, erythromycin, vancomycin, nystatin, and microstatin (Chater 2006). Only a few species and about 1% of the *Streptomyces* population typically found in any soil sample are potential plant pathogens.

At least eleven CS-causing species have been delineated, based on 16S ribosomal DNA sequences, whole genome sequence similarity and biochemical characteristics (Bouchek-Mechiche et al. 2006; Loria et al. 2006; Wanner 2009). *S. scabies* (synonym *S. scabiei*) is the best known of these and occurs world-wide. *S. europaeiscabiei* and *S. stelliscabiei* are morphologically similar to *S. scabies*, though genetically distinct. They were originally described from Europe and are also found in North America. A fourth morphologically similar species described from Egypt is similar or identical to *S. bottropensis*, and has been found in North America. Other scab-causing species include *S. turgidiscabies*

(in Japan, Korea, and Scandinavia), *S. acidiscabies* (eastern North America, Japan and Korea), *S. aureofaciens* (Finland), *S. reticuliscabiei* (netted scab in Europe), *S. ipomoea*e (sweet potato scab) and three new species described from Korea. All of these species also contain non-pathogenic members.

Mechanism of pathogenicity

What makes a few *Streptomyces* plant pathogens, while most are not? The only currently known pathogenicity factor (that is, something absolutely required for disease) is a toxin called thaxtomin, first discovered by Russell King's group in Canada around 1988 (Lawrence et al. 1990). Genes for biosynthesis of thaxtomin were cloned by Rose Loria's group at Cornell a few years later. These genes are found on a special part of the *Streptomyces* genome that has the hallmarks of a metabolic "island", or group of genes that are expressed together to convey a particular characteristic on a bacterium. Such islands commonly harbor genes for a whole process, such as synthesis of an antibiotic, degradation of a chemical, or pathogenicity. They can easily move from one bacterial species to another, transferring that characteristic, in a process called HGT, horizontal gene transfer (because the characteristic is inherited from neighboring bacteria, rather than being inherited vertically down the generations from parents). HGT is common in bacteria, and it explains how antibiotic resistance is moving around in people and in farm animals, for example. Movement, or HGT, of pathogenicity genes between *Streptomyces* species in the soil was first proposed by Loria and Japanese colleagues as the probable origin of a new pathogenic *Streptomyces* species in Japan (Loria et al. 2006). HGT of a chromosomal region containing thaxtomin biosynthesis genes is the current model for creation of pathogenic *Streptomyces* species, and provides an explanation for the appearance of CS on cultivars or in areas where it was previously not a problem - pathogenicity regions may be spread by HGT to non-pathogenic streptomycetes to create new scab-causing species or strains. The presence of pathogenicity genes is necessary but not sufficient for development of CS. Just as humans harbor all sorts of potential pathogenic bacteria, there are pathogenic strains of *Streptomyces* present in most soils, and yet, we observe tremendous variation in the occurrence and severity of CS disease.

Despite a shared mechanism of pathogenicity based on expression of thaxtomin, differences in disease occurrence are well-known, and the severity of CS is reportedly increasing in many potato-growing regions. We now know there is genetic variation in *Streptomyces* isolates from all over North America, and that some species are characteristic of geographic regions (Wanner 2009). It is difficult to say whether different species are significant for CS because there are also differences in what is known by plant pathologists as virulence, or the disease-causing potential, in isolates belonging to a single species. Laboratory and greenhouse studies show that different isolates do cause more or less severe symptoms on individual potato cultivars (Wanner and Haynes 2009).

Does this model for spread of pathogenicity mean that new pathogenic *Streptomyces* species are evolving continuously? There is molecular evidence that the origin of at least one or two new species of plant pathogenic *Streptomyces* was HGT, and it is certainly possible for new pathogenic species to arise in the future. The propensity of bacteria to "share" genetic material can complicate disease management, as *Streptomyces* species that are already well-adapted to a climate or soil type could pick up pathogenicity genes to create new CS-causing species. However, hundreds of *Streptomyces* species live together in soil, and yet we find only a dozen pathogenic species. Phylogenetic (family tree) evidence indicates that the main pathogenic species are closely related. A few other *Streptomyces* species are associated with potatoes, and these have not become pathogens, despite their proximity to pathogenic species on potato skins and in CS lesions. We presently don't understand the biological barriers to HGT.

An intriguing observation is that only certain potato tissues (underground stems and their derivatives, stolons and tubers) are susceptible to CS. Also, these tissues are only susceptible during a certain stage in early development. Green tissues typically do not become scabby. Mature potato tubers also do not develop CS, and it does not start or spread in storages. All of these observations lead to the obvious conclusion that the plant does or makes something that inhibits bacterial pathogenicity, and finding out what that is may lead to better ways to control CS.

Field symptoms

Potato CS lesions are quite variable with three types of lesions that are sometimes referred to as different diseases: russet (superficial corky tissue), common (raised; often warty), and pitted (shallow to deep holes or pits) scab. Growers frequently ask me if all of these can be caused by the same pathogen. The answer is yes, a single pathogenic species and strain can cause the whole range of CS symptoms. The type of lesion and the area affected by CS are partially determined by the potato genotype. Several other factors appear to be important in CS severity, including how much of the pathogen is present, exactly when in potato development bacteria are present, and factors we don't yet understand.

CS is not typically thought to cause yield losses; however, this may be under-reported, as growers don't associate above-ground foliar symptoms with below-ground damage to underground stems and stolons caused by *Streptomyces*. In

greenhouse tests, I find CS lesions on underground stems and stolons (see Figure, right panel). These can be so severe that they interrupt conductive tissues, resulting in foliar symptoms similar to some virus diseases (see Figure, right panel). Growers would typically not see these plants (they die early), attributing uneven plant stand to other causes. If they saw such plants, they would remove them, assuming they were due to a virus. Yield losses due to CS have been reported in Europe (Hiltunen et al. 2005).

Control of CS

Management of CS is a discouraging business. Inconsistent and/or inadequate results have been obtained with nearly all methods of controlling CS. This may not be so surprising considering that the evaluation of test results is complicated by the inherent variability of CS disease pressure. Also, apparently successful reports are typically based on 1 to 2 years of field experience, which is not a reliable test period, considering the variability in disease severity that is seen in different years. And the final confounding factor in assessing the few positive reports of control of CS is the tendency to report only "successes" in the literature. Table 1 summarizes the effectiveness of management practices that have been employed to control CS.

Table 1. Management practices to control common scab and their effectiveness

Scab management practices	**Effectiveness/limitation**
Lower soil pH to < 5.2	limits rotation crops; can fail
Soil moisture near field capacity for 4-5 weeks beginning at tuber initiation	often fails
Soil amendments: mulches and plant residues; organic amendments (liquid swine manure) potential elicitors, e.g. chitosan (crab shells), BTH (ActiGuard); green clay	Results variable and inconsistent; often fail
Chemical fumigation (PCNB, chloropicrin)	Can work for a season; expensive, environmentally unfriendly
Clean seed potatoes	Not sufficient, as *Streptomyces* is soil-borne
Seed treatments with mancozeb or metiram	Results erratic
Sulfur fertilizers	Reported to reduce scab severity in some locations
****Resistant plant cultivars**	**most reliable, cost effective control method currently available**
Biocontrol, using knowledge of soil microbial community interactions	**to be tested**

Cultural practices -- CS is often considered a disease of warm, dry, coarse-textured soils with neutral or higher pH, but CS has been reported wherever potatoes are grown, including wet and dry soils, sandy and humus-rich soils, and in soils ranging from pH 4.0 to above 9.0. Traditional management recommendations are to maintain high soil moisture during and after tuber initiation, and to maintain acidic soil pH (Lambert and Loria 1989; Powelson et al. 1993), but reports of CS in carefully irrigated fields are common, illustrating that these strategies are inadequate for controlling CS in the varied environments in which it occurs. Also, precisely the irrigation conditions recommended to inhibit CS enhance several other problematic soil-borne potato diseases, such as black scurf (caused by the fungus *Rhizoctonia solani*) and powdery scab (caused by the protist *Spongospora subterranea*). Since water management is inconsistent for controlling CS, water should be managed to optimize potato productivity, and minimize other diseases.

Soil fumigation -- Questions I am frequently asked are: How much pathogenic *Streptomyces* is found in the soil at different times throughout the growing season? What does this tell us about the risk of a serious CS problem? In collaboration with Barb Christ's group at Pennsylvania State University, we have recently developed a molecular technique that allows us to estimate the amount of pathogenic bacteria in soil and on potato plant surfaces. This has lead us to the surprising result that the same large amounts of pathogen are present in soil around developing tubers that end up with severe CS and in those that show no symptoms. It seems that some regulatory factor in the bacteria (or plant) must control pathogenicity, and it is not simply a function of the number of pathogenic bugs present. We and others are currently working to understand the mechanism of this regulation, as this obviously would provide the knowledge base to formulate new strategies for controlling CS.

We can detect pathogenic *Streptomyces* in nearly all soil samples, nearly all of the time, but in relatively low numbers. The same is true for potato tuber skins; both pathogenic and non-pathogenic *Streptomyces* can be found. The numbers of pathogenic *Streptomyces* are much higher in and around developing potato tubers and in CS lesions. Our data

show that the scab pathogen can multiply very quickly (as much as 100,000-fold in about a week!) under favorable circumstances, such as those that presumably occur on a newly developing tuber. This means that the potential for a CS problem is nearly always present, and it also means that you could introduce a large number of pathogenic bacteria by planting scabby potatoes. Planting severely scabby potatoes is known to increase the incidence of CS in that growing season. Clean seed is always important!

To return to the use of soil fumigation to control CS: Soil fumigants are expensive and environmentally unfriendly. PCNB (pentachloronitrobenzene), also known as Blocker® (Amvac), has been tested with some success. However, studies show that use at higher concentrations (20lbs/A) can reduce tuber size or yield. Pic-plus (chloropicrin) has shown some efficacy in trials in Michigan, Ontario and Florida. Requirement of minimum soil temperatures >45°F for application and 30-day interval post-application planting restrictions limit chloropicrin use in most northerly areas, it is expensive, and the effects seem to last for only a single season (http://www.potatodiseases.org/scab.html).

Crop rotation and cover crops -- Although crop rotation is often listed among control measures for CS, little data exist to support this idea. Reports of legume, cereal or crucifer cover crops limiting CS are not reproducible in multiple years.

Other methods of (chemical) control -- Wilson's group in Australia has shown that the use of synthetic auxins (2,4-D) at sub-lethal doses can reduce severity of potato CS, apparently by reducing thaxtomin toxicity (Tegg et al. 2008). They also were able to use thaxtomin to select resistant somaclonal variants of potato, some of which were more CS-resistant in preliminary field trials (Wilson et al. 2010). Whether this is a strategy with commercial potential remains untested. Sulfur-containing fertilizers, such as ammonium sulfate, are reported to be effective in some areas, perhaps working by lowering soil pH (Pavlista 2005).

Biological control -- Recent research results indicate that *Streptomyces* species closely related to CS-causing species are abundant on the skins of scab-resistant varieties, and are more frequent in less-severe superficial scab lesions than in pitted lesions. Some non-pathogenic species have been investigated for their potential to control CS, with mixed results (Hiltunen et al. 2009); (Wanner et al., unpublished.)

Disease-resistant cultivars

A useful and important component of CS management is the use of resistant cultivars (Powelson et al. 1993; Loria 2001). Relatively good levels of resistance are found in a few cultivars; 'Superior', 'Russet Burbank' and 'Pike' are three popular US cultivars with good CS resistance. Although there is no known immunity to CS in potatoes, we know there is a genetic basis for resistance (Driscoll et al. 2009), and every potato breeding program in the US (and world-wide) is interested in this trait. Although laboratory and greenhouse methods have been developed to screen for CS-resistant potato germplasm, breeding programs traditionally screen by conducting years of replicated field trials in multiple locations to account for environmental and geographic differences in CS disease pressure and incidence. Field trial data are also important for growers seeking to plant the most CS resistant cultivars for their areas.

CS resistance does not seem to follow a typical plant disease resistance model, with a plant resistance gene responding to a pathogen and setting in motion a plant defense response. There is no evidence for a plant defense response to *Streptomyces.* The suggested mechanisms for possible resistance to the pathogen involve detoxification of the toxin thaxtomin, or possibly limitation in uptake or response to the toxin. In fact, plant resistance to *Streptomyces* seems to be better described as tolerance (lack of symptoms in the presence of the pathogen) than resistance (limitation in numbers of the pathogen).

Genetics of CS resistance

Although a few genetic studies that have been published concluded that one or a few genes are responsible for CS resistance in haploid or diploid potato populations, there is nearly continuous variation in CS symptom severity among commercially grown potato cultivars, suggesting that multiple genes are involved. A segregating tetraploid population showed continuous variation in CS resistance, indicating complex genetics (Driscoll et al. 2009).

The only way to gain understanding of the genetics is to have a good way to phenotype potatoes. In the case of CS, this is a major obstacle. Even under the best-controlled circumstances, with a known pathogenic *Streptomyces* strain at a known concentration, and in controlled environmental conditions (greenhouse or growth chambers), a range in symptoms from nearly none to severe CS is seen on tubers from a single potato plant in a single pot. The lack of an easy phenotype significantly complicates data analysis, and thus, progress toward understanding the genetics behind resistance.

Summary: Knowledge gaps limit efforts to devise better strategies for control

Recent research has focused on two areas that may help in controlling CS: (1) developing rational, research-based measures based on understanding the pathogen, its distribution, and under what circumstances it causes disease; and (2) developing reliable disease-resistant cultivars. Factors that have hampered the development of CS-resistant potato cultivars include variable effects of environmental conditions, need for better sources of resistance (within *S. tuberosum)*, genetic variation in pathogen populations, and the variability in CS severity from year to year and location to location. Traditional control measures are insufficient and often fail. Planting cultivars with the best CS resistance in your region is currently the most successful control strategy.

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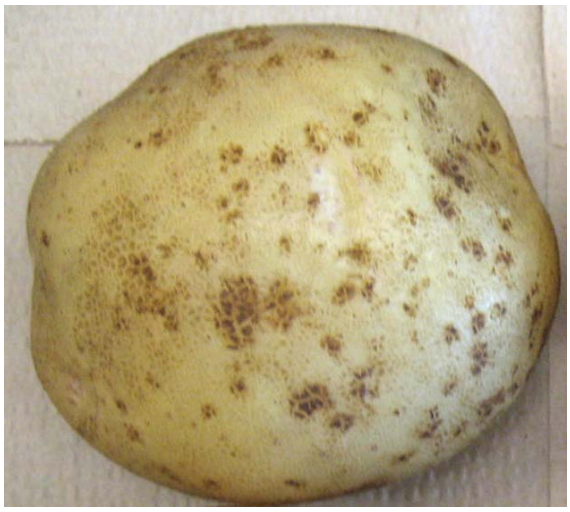

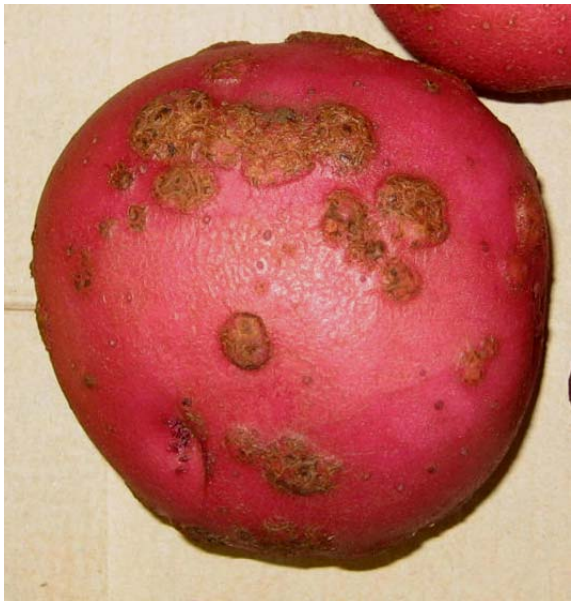

Above right: Symptoms caused by a pathogenic *Streptomyces* strain on a susceptible cultivar. Healthy untreated control plant on left; plant grown with *Streptomyces* at right, showing scab lesions that girdle underground stems, resulting in foliar symptoms.

At left: Typical common scab symptoms. Superficial (top), raised (middle) and pitted (bottom) scab.