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Managing fungal diseases of potato¹

Gary A. Secor and Neil C. Gudmestad

Abstract: Potato, *Solanum tuberosum*, is the fourth-ranked food crop used to support a growing world population because of cultivar versatility and high complex carbohydrate content. Potatoes can be stored for long periods for table and processing markets, but are plagued by storage disease problems. Diseases both in the field and during storage can be limiting factors in sustainable and profitable potato production wherever they are grown. In North America, many diseases caused by fungi are important and require a variety of management practices to reduce them to tolerable economic levels. Such diseases include late blight [*Phytophthora infestans*], silver scurf [*Helminthosporium solani*], pink rot [*Phytophthora erythroseptica*], dry rot [*Fusarium sambucinum*], verticillium wilt [*Verticillium dahliae* and *Verticillium albo-atrum*], black scurf [*Rhizoctonia solani*], and early blight [*Alternaria solani*]. These diseases have both a field and storage component, and disease management inputs may be necessary throughout the season for disease control. A continuing combination of cultural practices, planting of resistant cultivars, clean seed, crop rotation, and fungicides (plant medicines) are necessary for disease control. This presentation highlights the basics of each disease and, based on disease epidemiology, the current strategies used for control, and strategies underway for future control, including the development of resistant cultivars.

Key words: resistance, storage, integrated crop management.

Résumé : La polyvalence de ses cultivars et sa forte teneur en glucides permettent à la pomme de terre de se classer au quatrième rang parmi les cultures vivrières capables de nourrir une population mondiale en croissance. La pomme de terre peut être conservée longtemps pour les marchés de la table et de la transformation, mais elle est sujette à des maladies d'entreposage. Les maladies, tant au champ qu'en entrepôt, peuvent constituer des barrières à la production durable et rentable de la pomme de terre partout où on la cultive. En Amérique du Nord, plusieurs maladies fongiques sont importantes et nécessitent l'utilisation d'un éventail de pratiques pour les maintenir à des niveaux économiquement acceptables. Ces maladies incluent le mildiou [*Phytophthora infestans*], la gale argentée [*Helminthosporium solani*], la pourriture rose [*Phytophthora erythroseptica*], la pourriture sèche fusarienne [*Fusarium sambucinum*], la verticilliose [*Verticillium dahliae* et *Verticillium albo-atrum*], la rhizoctonie [*Rhizoctonia solani*] et l'alternariose [*Alternaria solani*]. Ces maladies ont des impacts au champ et à l'entrepôt, et des actions peuvent être nécessaires tout au long de la saison pour les gérer et les enrayer. Une combinaison permanente de pratiques culturelles avec la plantation de cultivars résistants, de la semence saine, la rotation des cultures et des fongicides (les médicaments des plantes) est nécessaire pour limiter les maladies. Cette présentation met en relief, pour chaque maladie, l'essentiel des connaissances et, basées sur son épidémiologie, les stratégies de lutte actuelles et celles préparées pour demain, y compris le développement de cultivars résistants.

Mots clés : résistance, entreposage, gestion intégrée des cultures.

Introduction

One of the standard recommendations for integrated disease control and management in any crop, including potatoes, is "use a cultivar resistant to (name the disease), if available." This is probably the best management practice because of immediate availability, low cost, and no risk implementation. However, for many crops, including potatoes, disease-resistant cultivars are not widely available or are not suitable for the intended end use, i.e., chipping or bak-

ing. If we look at the top 20 potato cultivars grown in North America and their respective disease resistance profiles (Table 1), it is easy to see that there is a paucity of disease resistance present, and when disease resistance is present, it is usually classified as moderate. This is striking considering that these 20 cultivars represent 81% of the potato acreage grown in the United States in 1997, and one cultivar, Russet Burbank, represents over 50% of the U.S. potato acreage, approximately 560 000 ha, and has limited disease resistance characteristics.

It is not, however, a case of potato breeding programs failing to find and implement disease resistance genes into named cultivars, but rather their consumer and commercial acceptance. Most, if not all, breeding programs use a combination of greenhouse and field observations to screen for disease resistance. Cultivar characteristics such as appearance, taste, or processability generally override the lack of disease resistance and consequently many cultivars have no disease resistance. Many diseases do not occur in all areas, resulting in no opportunity to screen or no pressure to do

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Table 1. Fungal disease resistance profiles of the top 20^a U.S. potato varieties.

Variety	% planted acres ^b	Resistance(s) ^c
'Russet Burbank'	50.5	Fus. (mod.)
'Russet Norkotah'	12.0	None
'Shepody'	8.1	Rhizoc. (mod.)
'Ranger Russet'	4.0	Vert. (mod.), Fus. (mod.)
'Norland'	3.5	None
'Russet Nugget'	2.9	Early blight (mod.), Vert. (mod.)
'Snowden'	2.1	None
'FL 1533'	1.9	None
'Goldrush'	1.7	Vert. (mod.)
'Superior'	1.3	None
'Centennial Russet'	0.9	Fus., Vert., Rhizoc., early blight (all mod.)
'Atlantic'	0.9	Late blight, Vert. (tol.)
'Red LaSoda'	0.8	Early blight (mod.)
'NorValley'	0.6	None
'Norchip'	0.5	None
'Red Pontiac'	0.5	None
'Sangre'	0.5	Tuber early blight (mod.)
'Norwis'	0.4	None
'Ontario'	0.2	Late blight?, Fus.?
'LaRouge'	0.2	None

^aCrop Production, Nov. 1997; National Agricultural Statistics Service, USDA.^bRepresents 81% of total fall production.^cFus., *Fusarium*; Rhizoc., *Rhizoctonia*; Vert., *Verticillium*; mod., moderately tolerant; tol., tolerant.**Table 2.** Fungal disease resistance in 130 North American potato varieties.

	Dry rot	<i>Verticillium</i>	<i>Rhizoctonia</i>	Late blight ^a	Pinkrot	Silver scurf	Early blight
No. of varieties	14	39	9	36	0	0	21 ^b
% of varieties	11	30	7	28	0	0	16

^aResistance to race 0 or 1 (US-1 genotype).^bOne reported tuber resistance.

so. A survey of North American cultivars with disease resistance (Table 2) reveals that over 25% of 130 cultivars released have resistance to one or more fungal diseases. It is unknown whether these resistances are due to field observations or carefully controlled scientific trials. The most success has been incorporation of *Verticillium* resistance. Late blight [*Phytophthora infestans*] resistance has been incorporated into many cultivars despite relatively low pressure from this disease until the 1990s. This resistance is undoubtedly to the 150-year-old US-1 isolate present in the United States until recently. It is also evident that resistance to many fungal diseases, notably pink rot [*Phytophthora erythroseptica*] and silver scurf [*Helminthosporium solani*], is absent.

Potato breeding programs in Europe have different disease problems to contend with than in North America, and therefore different emphasis is placed on disease resistance breeding. European cultivars are rated for resistance or susceptibility to late blight, gangrene [*Phoma exigua* var. *foveata*], common scab [*Streptomyces scabies*], dry rot [*Fusarium sambucinum*], skin spot [*Polyscytalum pustulans*], and powdery scab [*Spongospora subterranea*]. Of these, late blight is considered the most important and receives the most attention.

Late blight

Late blight has in recent years become a destructive disease throughout the entire potato growing areas of North America. Because no resistant cultivars are widely grown, management of the disease involves multiple strategies at every stage of the potato growing cycle. Management strategies are necessary to prevent the buildup of overwintering inoculum (destruction of cull piles, preventing volunteers, eliminating infected seed), elimination of alternate hosts, and rigorous application of protectant fungicides. Disease management depends on effective fungicide application, including timing, rate, interval, and coverage. Fungicides can be applied by aircraft (airplane and helicopter), ground (conventional high pressure and air assist), and irrigation. Because this disease is dependent on weather conditions, disease prediction models have been developed to forecast disease occurrence and fungicide application recommendations. Use of these models has given very effective disease control over the past 5 years. We issue late forecasting results and other pertinent disease information three times a week from June 1 to September 1, and can be accessed by a toll-free number, computer, and radio broadcasts, all widely used by the potato industry. We have found that despite ex-

Table 3. Race composition of *P. infestans* genotypes in North America from 1994 to 1997.

Isolate	Mating type	Genotype	Year isolated	Physiological race
51.1	A1	US-1	1995	R0,(10),(11)
52.1	A1	US-1	1995	R0,3,10,11
70.2	A1	US-1	1995	R0,11
77	A1	US-1	1995	R0,11
99.2	A1	US-1	1995	R0,11
279	A1	US-1	1997	R0
203.2	A1	US-11	1996	R0,1,3,5,7
203.3	A1	US-11	1996	R0,1,5,7
214 ^a	A1	US-11	1997	R0,1,5,7
234.4	A1	US-11	1997	R0,1,5,7
234.6	A1	US-11	1997	R0,1,(5),7
250.1	A1	US-11	1997	R0,1,5,7
55.2	A1	US-6	1995	R0,10,11
273	A2	US-7	1994	R0,1,5,7
216	A2	US-7	1997	R0,1,2,3,4,5,6,7,10,11
48.1	A2	US-8	1995	R0,1,2,3,4,5,6,7,10,11
92.1	A2	US-8	1995	R0,1,2,3,4,5,6,7,10,11
105	A2	US-8	1995	R0,1,5,7
126	A2	US-8	1995	R0,1,2,3,4,5,6,7,9,10,11
129.2	A2	US-8	1995	R0,1,2,3,4,5,6,7,(8),10,11
48.2	A2	US-8	1995	R0,1,2,3,4,5,6,7,10,11
142	A2	US-8	1995	R0,1,2,3,(4),5,6,7,10,11
150	A2	US-8	1996	R0,1,2,3,4,5,6,7,10,11
156	A2	US-8	1996	R0,1,2,3,4,5,6,7,9,10,11
174.2	A2	US-8	1996	R0,1,2,3,4,5,6,7,10,11
181	A2	US-8	1996	R0,1,(2),3,4,5,6,7,(8),(9),10,11
186	A2	US-8	1996	R0,1,2,3,4,5,6,(7),10,11
188	A2	US-8	1996	R0,(1),(2),3,4,(5),6,7,(9),10,11
191	A2	US-8	1996	R0,1,2,3,4,5,6,7,9,(10),11
204	A2	US-8	1997	R0,1,2,3,4,5,6,7,10,11
209.1	A2	US-8	1997	R0,1,2,3,4,5,6,7,10,11
211	A2	US-8	1997	R0,1,2,3,4,5,6,7,10,11
212	A2	US-8	1997	R0,1,2,3,4,5,6,7,9,10,11
215	A2	US-8	1997	R0,1,2,3,4,5,6,7,10,11
217	A2	US-8	1997	R0,1,2,3,4,5,6,7,10,11
219	A2	US-8	1997	R0,1,2,3,4,5,6,7,10,11
220	A2	US-8	1997	R0,1,2,3,4,(5),(6),7,10,11
227	A2	US-8	1997	R0,1,2,3,4,5,6,7,10,11
230	A2	US-8	1997	R0,1,2,3,(4),5,6,7,10,11
243	A2	US-8	1997	R0,1,2,3,4,5,6,7,10,11
283	A2	US-8	1997	R0,1,2,3,4,5,6,7,10,11
218.1	A2	US-8	1997	R0,1,2,3,4,5,6,7,10,11
218.2	A2	US-8	1997	R0,1,2,3,4,5,6,7,10,11
282	A1	nd ^b	1997	R0,1,2,3,4,5,6,7,10,11
50.1	A1	nd ^c	1995	R0,1,2,3,4,5,6,7,10,11
74.2	A1	nd ^c	1995	R0,1,2,3,4,5,6,7,9,10,11
81.1	A1	nd ^c	1995	R0,1,2,3,4,5,6,7,10,11
19.1	A2	nd ^d	1994	R0,1,2,3,4,5,6,7,9,10,11
1.1	A1/A2	nd ^e	1994	R0,1,2,3,4,5,6,7,10,11
118.2	A2/A1	nd ^f	1995	R0,1,2,3,4,5,6,7,10,11

Note: Parentheses denote at least half of the trials were positive, but not 75%.

^aIsolated from tomato.

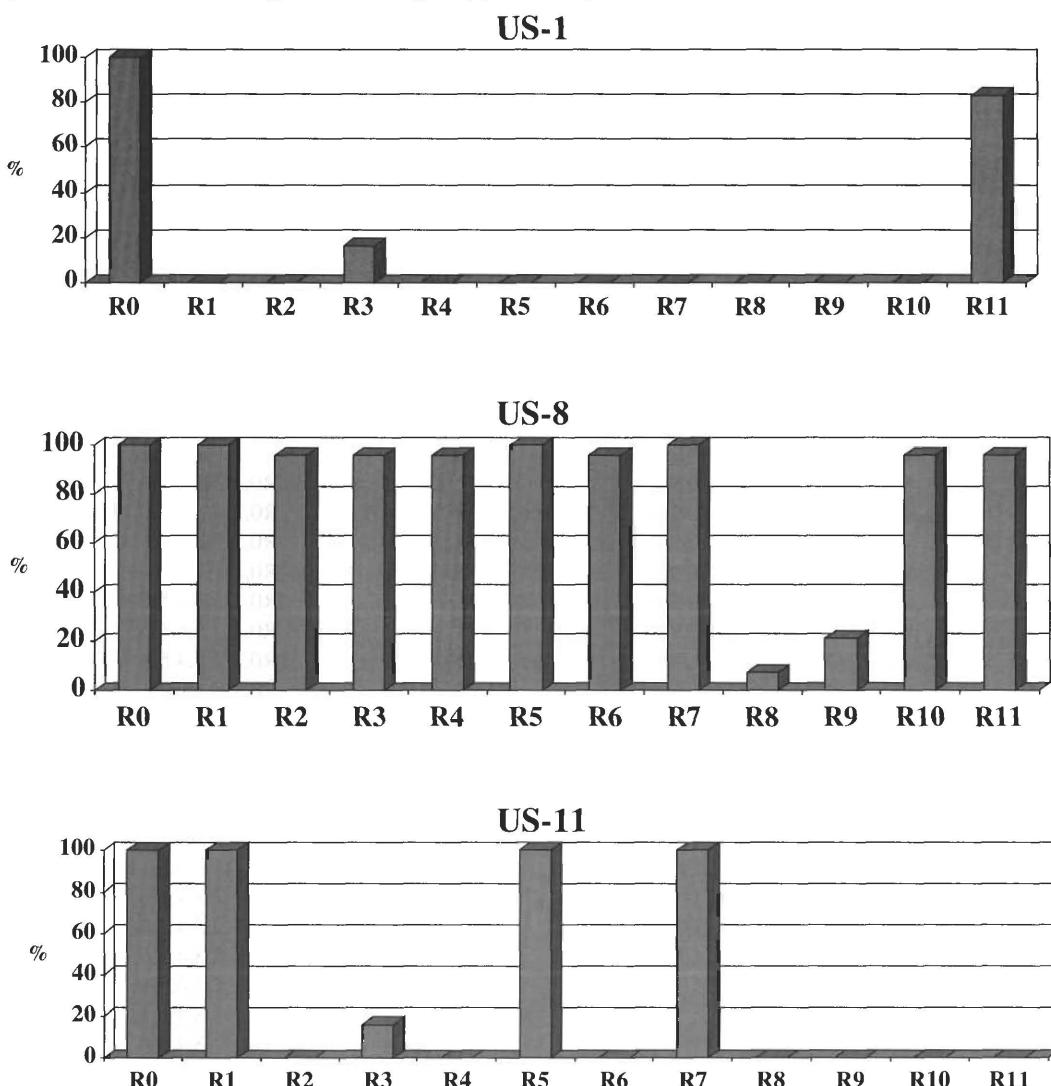
^bThe *Gpi* banding pattern did not resemble any known genotype.

^cThe *Gpi* banding pattern was identical with that of a US-8.

^dThe *Gpi* banding pattern was identical with that of a US-1.

^eOospores were formed with both A1 and A2 mating types. More profuse oospores were formed with A2.

^fOospores were formed with both A1 and A2 mating types. More profuse oospores were formed with A1.

Fig. 1. Frequency of individual virulence genes in each genotype of *P. infestans*.

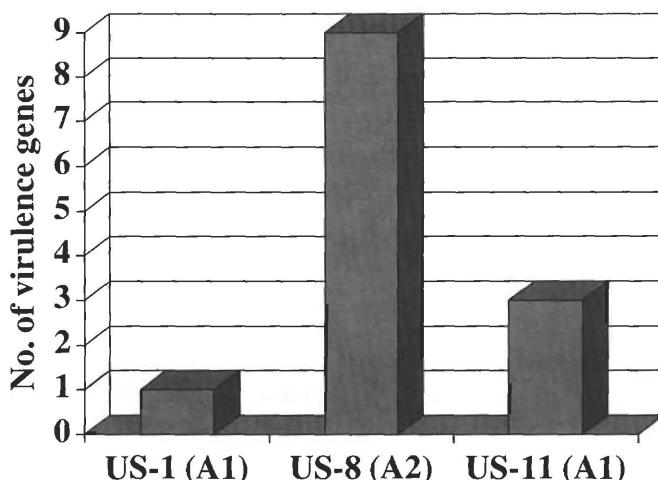
cellent fungicide application, we have more tuber infection than expected from the low foliar infection. We recommend fungicides to be applied after the primary vine kill and before harvest to reduce tuber infection. More research needs to be done in the areas of understanding and reducing tuber infection. All of these strategies need to be used in order to develop an overall management plan for late blight control.

A late blight resistant potato cultivar is desirable for many reasons. It slows disease progress, extends the spray interval, and reduces fungicide usage and production costs, increasing profits to the producer. North American cultivars listed as resistant to late blight, such as 'Kennebec' or 'Bisson', are resistant to the old US-1 genotype, which predominated until about 1995. To our knowledge, no cultivars have been released with resistance to the new genotypes present in North America, which are US-6, US-7, US-8, US-11, and US-17. These new genotypes encompass both the A1 and A2 mating types and are capable of infecting potato, tomato, and hairy nightshade (*Solanum sarachoides*), although host preferences exist among genotypes. Most potato breeding programs have active late blight resistance programs in place, either new or expanded since

1995. These programs utilize both vertical and horizontal genes for resistance; 11 major R genes have been identified for late blight resistance. Genes for late blight resistance are being used from worldwide sources, including Europe, South America, and Mexico. Both cultivars and wild species are being used as sources of resistance. The International Potato Center (CIP) has spearheaded a global initiative for late blight resistance and has an active program that breeds for late blight resistance. Germplasm has been identified and released (6, 7, 9, 11) with resistance to late blight. It is estimated that a cultivar with resistance to the new late blight genotypes could be released within the next 5 years.

Potato breeding efforts for late blight resistance have been intensified in recent years, and almost all released cultivars must have resistance to late blight in order to be an accepted cultivar. Resistance may be foliar, tuber, or both; resistance in one does not necessarily mean resistance in the other. If we look at potato cultivars with late blight resistance released from the Netherlands as an example, the majority of cultivars released have tuber resistance and only a few have foliar resistance or both. Of 118 cultivars re-

Fig. 2. The median number of virulence genes present in the US-1, US-8, and US-11 genotypes tested.



leased, five have foliar resistance, 75 have tuber resistance, 15 have both, and 23 have neither. It is interesting to speculate on the relative inheritance of tuber versus foliar resistance. Researchers for our breeding program at North Dakota State University and that at Michigan State have recognized the importance of tuber resistance and screen both foliage and tubers for resistance (21). Because 11 genes for resistance to late blight have been identified (R genes), physiological races of *P. infestans* occur with various combinations of R genes. Presumably an almost infinite combination of R genes can occur in *P. infestans* (actually 11!, or 39 916 800 combinations). Identification of R gene composition of late blight isolates used to screen potato germplasm and cultivars for late blight resistance would be extremely important. The R gene composition of *P. infestans* has been investigated in Europe (1), but similar work has only begun in North America. Recently Pasche et al. (23) looked at R gene composition of several U.S. genotypes. Her results show a broad range of R genes present between and within genotypes (Table 3, Figs. 1 and 2). This serves to point out the necessity of determining R gene composition of *P. infestans* isolates used in breeding programs.

Molecular and biotechnology approaches for incorporating resistance are being used in addition to traditional breeding approaches (31). The use of wild *Solanum* species (10), marker-assisted breeding (20), and protoplast fusion are all being used to enhance late blight breeding strategies. Helgeson and his colleagues (12) have regenerated protoplast fusion lines of cultivars of *Solanum tuberosum* and *Solanum bulbocastanum*, a wild species with resistance to *P. infestans*. Repeated field trials under severe conditions at multiple sites have shown that some of fusion clones have high levels of resistance to late blight. The tubers lack commercial acceptability and need further backcrossing before release of an acceptable cultivar. Several large agricultural corporations are using biotechnology to develop late blight resistant potato cultivars. Naturemark Inc. is modifying existing cultivars using transgenic technology for late blight resistance, similar to their successful program for developing cultivars resistant to the Colorado potato beetle. Zeneca

has antifungal protein genes that can ostensibly be used to make transgenic plants with resistance to late blight. Other corporations such as Novartis, American Cyanamid, and AgrEvo have active programs for the development of new late blight technology.

Silver scurf

Silver scurf disease of potato is caused by the fungus *Helminthosporium solani*. The disease has gone from obscurity to destructive the past few years because of a combination of factors, including resistance to thiabendazole fungicides, improved storages with higher humidities, lowered defect tolerances, and increased awareness. It is a blemish disease of tubers, causing a metallic discoloration of the periderm in irregular patterns. It does not cause yield losses at harvest, but does cause weight loss of stored potatoes due to increased water loss, resulting in excess shrink and flabbiness. It affects quality of all market classes of potatoes. It is a cosmetic disease of fresh table potatoes, resulting in reduced consumer acceptance and rejection, particularly after prolonged storage. Round white processing potatoes used for potato chips are more difficult to peel because the dehydrated and diseased periderm is difficult to peel, and remaining peel causes an undesirable edge on the chips after cooking. The disease does not affect any other part of the potato plant except the tubers, and both the teliomorph and alternate hosts are unknown.

Silver scurf is primarily a seed-borne disease. After planting of infected seed pieces, sporulation can be seen on the seed pieces one week after planting and can be recovered from soil surrounding the seed piece two weeks post-planting. Infection of new progeny tubers can occur as early as nine weeks after planting. It is unknown how infection moves from the seed to the new tubers; the spores are not motile, and movement to new tubers via infected stolons has not been documented. Low levels of conidia may persist in the soil from one season to the next, but prolonged soil survival has not been demonstrated. Crop rotation is an important management practice to prevent infection by this short-term soil-borne inoculum.

Silver scurf spreads in storage, resulting in an increase in both disease incidence and severity. Spores (conidia) are detected early and throughout the time potatoes are in storage. Spore traps placed in commercial potato storages have detected up to 25 000/day (26). Sporulation occurs in processing storages held at 10°C (50°F) and in seed storages held at 4°C (38°F). Sporulation is reduced by cooler temperatures. Spores are dislodged and released into the storage atmosphere when tubers are moved or handled for grading or shipping, and move throughout the piled potatoes via the air handling system. These conidia are infectious and move to healthy tubers or uninjected parts of the same tuber and cause new silver scurf lesions. Old lesions enlarge and sporulate at the edges. Germination of *H. solani* spores is reduced from 80% to <5% after exposure to humidities of <95% regardless of temperature (10, 15, and 20°C). Seed tubers can become infected when seed is removed from the seed house prior to cutting; *H. solani* spores are dislodged into the air and infect the unprotected seed tubers.

Virtually all *H. solani* isolates collected from North America are resistant to thiabendazole (Mertect). Post-harvest application of Mertect for fusarium dry rot control formerly provided good control of the silver scurf as a nontarget organism, but is no longer effective because of resistance. Seed treatment fungicides are effective for limiting silver scurf at harvest. Recommended seed treatments include mancozeb, TOPS-MZ, and Maxim. Maxim has shown suppression of silver scurf even into the storage season. Dithane ST is registered for silver scurf control going into storage, but use is limited to seed potatoes only; potatoes for food or feed cannot be treated. Fungicide application on seed potatoes as they are coming out of storage prior to shipping may be ideal for disease management, since seed tubers are freshly exposed to the spores, and this may prevent widespread infection of seed prior to planting.

Research has shown that silver scurf increases in the field with early planting and late harvest dates. The longer potatoes are in the ground after vine kill, the more silver scurf they have. The least amount of silver scurf is found when the interval between vine-kill and harvest is 5 days. There is no resistance to silver scurf in existing cultivars in North America or Europe, so resistance cannot be used for disease management. However, 11 wild species have been identified with good resistance to silver scurf, especially in *Solanum demissum*, and this is currently being introgressed into domesticated potatoes by breeding programs for future varieties (27). The use of clean seed, free of silver scurf, is not a feasible control strategy now because virtually all seed is infected with *H. solani*. Minitubers free of *H. solani* can be produced in greenhouses, but are rapidly recontaminated when exposed to spores released from infected tubers in storage. Consequently, even early generations of seed are infected with silver scurf. In addition, new infections are hard to see on unwashed seed, and it is difficult to grade and remove affected seed tubers. Separate storage bins, and air systems, of early generation seed lots would prevent contamination of new seed lots.

Sanitation is recommended annually to get rid of spores that remain in the storage from one season to the next and are circulated to the new crop when the storage fans are turned on. Removal of potato residues and washing with soap and water will get rid of the majority of the over-seasoning spores. Because spore germination is inhibited by humidities less than 90%, maintaining a dry storage environment of 90% for the first month after potatoes are placed in storage is recommended to delay silver scurf spread. Humidities can be increased after this time to prevent excessive shrink and pressure bruise. Separate storages with separate air handling systems for early generations of seed potatoes will prevent contamination of otherwise clean seed lots.

Preliminary research has shown that treatment of the storage atmosphere with activated chlorine dioxide (ClO_2) may reduce the infection and spread of silver scurf in storage and limit disease. Trials conducted in commercial potato storages have shown that adding chlorine dioxide via the humidification system reduces the number of silver scurf spores as well as the incidence and severity of silver scurf in storage after 16 weeks. Work in this area is continuing. We have also identified a mycoparasite of *H. solani*,

tentatively identified as *Cephalosporium* sp., that reduces sporulation and may be useful as a biocontrol agent to reduce spread of disease in storage, but more work is necessary to demonstrate effectiveness. Other work has shown that postharvest application of some salts and organic acids, notably potassium sorbate, can reduce silver scurf and *H. solani* of treated potatoes during storage (22). These simple salts are already used in food preservation and further work may establish their usefulness in silver scurf management.

Pink rot

Pink rot and leak (collectively called water rot) of potato can be serious diseases of harvested potatoes. Losses as high as 50% can result from these diseases. Pink rot is caused by the fungus *Phytophthora erythroseptica* (*P.e.*), and leak is caused *Pythium ultimum*, but some other species of *Pythium* may cause leak in some areas. Both of these fungi are soil-borne and occur in most if not all soils. The symptoms of the diseases are similar; both cause a rapid watery breakdown of tubers in storage when conditions are suitable. The main difference is that *P.e.* infection can occur in the field prior to harvest through stolons, lenticels, or eyes, whereas *Pythium* infection occurs in wounds and injuries that occur during harvest. Pink rot is favored by wet conditions in the field, and leak occurs when potatoes are harvested at temperatures above 22°C (70°F). Neither disease will spread in storage to any extent.

Management of these diseases includes avoiding excess water, harvest temperatures below 22°C, cooling harvested tubers, and timely application of metalaxyl (Ridomil) at the full label rate when tubers are 2 cm in diameter. The fungicide may be applied by air, ground or irrigation. Bruising, skinning, and higher temperatures all increase disease incidence. Resistance of the pink rot pathogen, *P.e.*, to metalaxyl has been reported in the northeast region of the United States, but widespread resistance has not been found in North Dakota and Minnesota. Practising crop rotation by planting potatoes no more than every 3–4 years and avoiding following them with kidney beans, which act as an alternate host, will help reduce disease.

Most potato varieties grown in North America have not been tested for resistance to pink rot, and no resistant varieties have been identified. However, differences in cultivar susceptibility and resistance have been reported (30). Certain varieties such as 'Shepody' and 'FL 1533' are considered very susceptible to pink rot based on field observations. In general, red-skinned varieties appear to most susceptible to this disease, and 'Russet Burbank' is only moderately susceptible. More screening and testing need to be done to confirm these preliminary data, but there appears to be differences among genotypes in susceptibility and resistance to pink rot.

Dry rot

Dry rot is caused by several species of *Fusarium* that cause a dry rot of potato tubers in storage. The most common species is *Fusarium sambucinum* (synonym = *sulphureum*). Dry rot is a ubiquitous and destructive disease

wherever potatoes are grown. It is both a soil- and seed-borne disease and requires an injury for entry into the host. Soil-borne inoculum can persist for many years in infested fields. Dry rot does not occur in the field because soil-borne *Fusarium* spores must enter through wounds and injuries that occur at harvest. However, the *Fusarium* species that cause dry rot can also manifest themselves as seed decay and in-field wilt. Dry rot develops slowly and does not spread in storage. The disease has been effectively controlled by postharvest applications of thiabendazole (Mertect), but many isolates of *Fusarium* throughout North America have become resistant to Mertect. This results in not only reduced control but also, in some cases, increased incidence and severity of disease (28). Consequently, we must rely on additional management strategies, including avoiding wounds and injuries at harvest, wound healing of stored potatoes to wall-off infection sites, crop rotation, the use of clean seed, and the use of a seed treatment. Mertect may still be used for control of dry rot in fields where resistance has not been found, and in some areas, Dithane ST is approved for postharvest application for seed potatoes only. Some germplasm and varieties have been identified with moderate resistance or tolerance to dry rot (15, 22), but resistance is not a major means of disease management.

Verticillium wilt

Verticillium wilt is a widespread disease of potatoes caused by *Verticillium dahliae* or *Verticillium albo-atrum*. Verticillium wilt is also known as early dying or premature senescence. It causes substantial yield losses and vascular discoloration of the tubers, which makes them undesirable for processing. The fungus survives primarily as micro-sclerotia in plant residues and soil, but can also survive on other weed and crops residues. Infection occurs through root hairs, cultivation wounds, and wounds made by nematodes, especially *Pratylenchus penetrans*. Infected seed is not an important source of infection, but does serve as initial sources of inoculum. The fungus forms large numbers of pepper-flake-sized microsclerotia in infected stems, and since stems are left in the field during harvest, inoculum builds up from one season to the next (29). The number of sclerotia produced varies from cultivar to cultivar and is used as a measure of susceptibility or resistance. The most susceptible variety is 'Kennebec', and fields with many crops of 'Kennebec' were so contaminated and yield losses so severe that production was curtailed. Management of *Verticillium*-caused early death is difficult. One management strategy is to avoid planting susceptible cultivars, such as 'Kennebec'. Another management strategy is crop rotation of at least 3 years between potato crops, but other rotational crops such as cereals and weeds support the fungus from one season to the next, making it not very effective. Removal of microsclerotia-laden vines by physical removal or burning effectively reduces soil inoculum and subsequent disease, but in practice is seldom done. Balanced fertility and water management, especially avoiding early over-watering, reduces the impact of verticillium wilt. The only really effective cultural practice is soil fumigation, which is costly, controversial, and only effective for one to two seasons.

Resistance to verticillium wilt has been studied (2, 5, 18, 19), and traditional breeding programs in Idaho, Wisconsin, and Minnesota have been very successful in finding germplasm with good resistance to *Verticillium* (3, 4, 8, 13, 14, 16, 24, 32). This germplasm has been used by many potato breeding programs in North America to release varieties with good resistance. Resistance has been easy to introgress into red-skinned and russet varieties, but it has been more difficult to transfer this resistance to white-skinned varieties. Molecular techniques are being used to map *Verticillium* resistance genes in diploid potatoes, and Monsanto/Naturemark are evaluating Newleaf clones of major cultivars with *Verticillium* resistance using transgene technology.

Rhizoctonia

Rhizoctonia is caused by a potato seed- and soil-borne fungus called *Rhizoctonia solani*. It causes a stem canker and dieback and distortion of newly emerging underground stems and stolons, resulting in grade, quality, and payout reductions. It also causes wash-resistant black scurf on mature tubers, a blemish disorder resulting in lower consumer preference. The stem canker or dieback stage primarily comes from infected seed inoculum, and the black scurf stage primarily comes from soil-borne inoculum. *Rhizoctonia* survives primarily as hard black sclerota that can persist for long periods in soil, and spores are not important in the disease cycle. *Rhizoctonia solani* is composed of fungal anastomosis groups designated as AG3, AG4, AG5, etc., which tend to be crop specific. The most common group that attacks potatoes is AG3. Disease management requires implementing a number of cultural practices. Soil-borne *Rhizoctonia* is difficult to eliminate from infested fields, but a 3- to 4-year crop rotation helps. Preferred rotational crops include barley and oats, and it is best to avoid sugar beets and dry beans. The use of clean seed is recommended. Avoid seed lots with a high percentage of tubers with sclerota; we recommend that seed have less than 5% coverage with sclerota. Seed with higher levels of *Rhizotonia* contribute to stand and emergence problems. Anything should be done to promote rapid emergence of seed, such as planting in warm soil (greater than 10°C (50°F)), planting shallow, dragging-off hills, etc. The longer sprouts and stems are in the soil the better are the chances that *Rhizoctonia* will infect them. Seed treatment fungicides such as Maxim (fludioxanil) and TOPS-MZ (thiophanate-methyl-mancozeb) are recommended for control of seed-borne inoculum. The black scurf stage of the disease is difficult to control because it is so widely distributed in the soil. Pentachloronitrobenzene has been re-registered as Blocker for control of both seed- and soil-borne inoculum. Resistance to *Rhizoctonia* is not common, and therefore, the use of resistant cultivars for disease management is not a consideration.

Early blight

Early blight is a persistent and consistent disease affecting potatoes late in the growing season as they approach maturity (17). It is often called a disease of senescence, and growers have used the disease as a cheap vine-kill to help

kill the foliar tissues prior to harvest. It almost always affects only the foliar parts of the plant, but can affect tubers, causing a shallow dry rot. This rot is characterized by a bluish or gun-metal gray discoloration surrounding shallow irregular lesions that are about 6 mm deep, brown in color, and corky in consistency. In recent years, early blight tuber infection has been increasing despite the increased use of foliar fungicides for late blight management. The same fungicides are also recommended for early blight control, so this increase is surprising. Some cultivars such as 'FL1625', 'Itasca', and 'NorDonna' seem to be especially susceptible to early blight tuber infection, so one management scheme is to avoid susceptible cultivars. Because tubers become infected when leaf-produced spores enter harvest wounds in the soil, maintaining effective control of early blight on the foliage is important. Foliar management includes using fungicides with good activity against early blight; use of copper-based fungicides, or cymoxanil (Curzate), alone should be avoided. Fungicides should be applied in a timely manner as recommended by a weather-based disease prediction program such as Wisdom. Stressed potato plants are more susceptible to early blight. Stress from low nitrogen, early dying, insect damage, etc. should be avoided. The most important management practice is to avoid injuring tubers at harvest, since early blight spores can only infect through a wound or injury. There are no cultivars resistant to early blight, but there are some cultivars with increased susceptibility to the disease.

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