Control of Diseases of Potatoes

Eugene S. Schultz

The Irish potato is susceptible to many diseases. The fungi that might attack it range from the slime molds to the smuts and rusts. It is subject to several viruses of the yellows and mosaic groups. Such nonparasitic diseases as black heart, sunscald, freezing injury, and a malnutrition caused by deficiency in magnesium, potash, and boron may cause damage. Several nematode diseases have been found on it.

Unless effective methods of control are practiced, serious diseases, such as late blight, ring rot, and leaf roll, can cause the total loss of a crop.

Late blight of potato is a downy mildew caused by a fungus, *Phytophthora infestans*. The mildew, or flour-like spots, usually on the lower surface of the leaf, distinguishes the disease from other leaf spots on the potato plant. Humid conditions favor it. Despite its name, the first infection often occurs soon after the plants emerge when favorable moisture and temperature prevail. At 70° to 75° F. the fungus grows so fast inside the leaves that within a week after infection it causes dead spots one-half to 1 inch in diameter. The entire plant may be killed within 2 weeks. The brown discoloration of the foliage brings to mind the terms “blight” and “rust.”

Late blight destroyed potato crops so often during the nineteenth century...
that it outranked other potato diseases and was called "the potato disease." In 1845 it was responsible for the potato famine in Ireland.

The late blight fungus produces a mycelium, or moldlike growth, which invades and kills the plant tissue. From the mycelium grow treelike sporangiophores, which bear sporangia, the agents involved in spreading infection. At about 70° and 90 percent relative humidity, sporangia develop in 5 to 7 days after infection. In water at 72° to 77°, sporangia form germ tubes. At lower temperatures, 58° to 63°, they develop swarmspores, or zoospores. The two methods of germination enable the fungus to adapt itself to a rather wide range of temperature. The higher temperature favors mycelial development, rapid invasion, and killing of the plant. The lower temperature favors formation of zoospores and rapid increase in inoculum to spread infection.

The fungus infects the potato tuber as well as the tops. Spores from infected tops are carried by rain to the tubers in the soil. Tubers also are readily infected if they are harvested before the blighted tops are killed. Infected tubers quickly show a brown discoloration, which changes to a purplish color. The fungus usually invades the tuber to about one-fourth to one-half inch below the skin. At about 36° to 40°, the affected tubers persist in a dry rot condition. Higher temperatures favor secondary infection with bacteria, and the blighted tubers develop soft rot.

The organism is carried into the following season in infected tubers. When such tubers are planted, the fungus invades the shoots on which form the spores that infect the foliage. Another source of infection is the blighted tubers in the potato refuse or dump pile. Late blight appears on potato tops on waste piles a few weeks before the plants in the fields emerge, so that the potato tops in the fields become infected as soon as the plants emerge and before spraying begins.

The spread of late blight is favored by humid conditions such as may prevail in the New England States, other regions along the Atlantic coast, and the North Central States. With favorable moisture, it also appears in the Middle Western, Southern, and Pacific Coast States. It occurs in long periods of rain, fog, and dew, which favor fruiting, infection, and dissemination of the fungus.

Control measures are sanitation, protection, and the use of resistant varieties.

Sanitation involves careful inspection and removal of blighted tubers from seed potatoes and elimination of piles of waste potatoes. Waste potatoes that have to be dumped should be given a dormancy or sprout-inhibiting treatment—a.25 percent isopropyl ester of trichlorophenoxy acetic acid—before dumping to prevent sprouting.

For many years a combination of copper sulfate and lime—bordeaux mixture—has been used to control late blight. Weekly spraying with it during the growing season effectively, although not absolutely, controls late blight even during epidemics. Copper-lime dusts control late blight if applied at rates of 25 to 30 pounds the acre at weekly intervals.

Some of the more recently developed products like Dithane and Parzate are less toxic to foliage and therefore favor higher yields than when bordeaux mixture is used. Because these products are not generally so effective for control of late blight as bordeaux mixture, some growers change to bordeaux mixture during the latter part of the season when conditions favor an epidemic of late blight.

Because lime in bordeaux mixture increases transpiration to a harmful degree and because one-half the amount of lime used formerly in bordeaux mixture is satisfactory, many growers use a recommended formula such as 10-5-100—10 pounds of copper sulfate and 5 pounds of lime in 100 gallons of water.
Search for resistant varieties began about the middle of the nineteenth century. Varying degrees of resistance appeared in different varieties, but no cultivated varieties manifested immunity from late blight. Sebago, Menominee, and Calrose, though not immune, are somewhat blight-resistant. The tubers in some varieties, such as Sebago, are more blight-resistant than the foliage.

Among certain wild species, such as *Solanum demissum*, some lines or strains are immune. Some lines of that species also are blight-susceptible. Empire, Ashworth, Chenango, Essex, Placid, Virgil, and Kennebec are varieties in which *S. demissum* was one of the parents and are immune to some races of late blight.

The development of blight-resistant varieties is a complicated procedure—actually, the plant breeder has to develop resistance to several biological races that vary in ability to cause disease. A blight-immune variety may be immune to one race but susceptible to other races. Within the past decade, however, efforts to develop blight-immune varieties show that varieties can harbor immunity to more than one race of the fungus of late blight.

Common scab, caused by *Streptomyces scabies*, is recognized by slightly raised spots or lesions of rough, corky tissue on the tuber. The lesions may be so numerous as to involve the entire surface. They may involve distinct russetting or deep scab. Scab lesions spoil the looks of the tuber and cause waste in peeling and reduction in grade.

The fungus lives in the soil and on infected tubers. It occurs in nearly every potato-growing region. Soil acidity, moisture, temperature, and aeration influence the development of scab. A soil reaction below pH 5.2 is unfavorable for most of the common scab races, although some strains are said to cause infection below pH 5. In general, the fungus is favored at pH 5.2 to 7. Common scab develops at a wide range of temperatures, 50° to 85°, but it thrives best at about 70°. Relatively high soil moisture tends to check the disease in some localities, but in other districts high moisture may favor scab. Lack of aeration inhibits development of the organism.

The several races of the fungus vary in type and color of mycelium, color in the medium, and pathogenicity. Apparently the races result by mutation and some races mutate more than others.

Treatment of seed tubers with disinfectants kills the scab fungus on the tuber, but it fails to control the disease if the treated tubers are planted in scab-infested soil. The use of 300 to 500 pounds of sulfur an acre reduces the severity of scab in some soils. The use of ammonium sulfate in fertilizer that increases soil acidity may inhibit the disease somewhat. None of the treatments has been entirely adequate for scab control, however.

The most promising method involves the development of scab-resistant varieties. Studies in Europe have disclosed that Jubel, Hindenburg, Ostragis, and Arnica are scab-resistant. In this country, scab resistance was noted in Russet Rural and Russet Burbank. Although the scab-resistant European varieties are not adapted to growing conditions in America, they do serve as resistant parents. From them the resistant Cayuga, Menominee, Ontario, and Seneca were derived. Investigations on scab resistance by the Department of Agriculture and State experiment stations are designed to develop varieties that are superior to them in scab resistance, quality, adaptability, and yield.

Vegetable wilt, caused by *Verticillium albo-atrum*, induces wilting of the tops and vascular discoloration of the stems, tubers, and roots—symptoms similar to those associated with other wilt-inciting fungi. Since *V. albo-atrum* is favored by lower temperatures than are wilt-inciting Fusaria,
it often occurs in the cool seed-potato areas.

Verticillium wilt is recognized by flaccid, drooping leaves. Often only one stalk in the hill or a part of a stalk may be wilted. Later all the plants in the hill are involved and finally are killed.

Cross sections near the stem or stolon end of the tubers expose dark-colored vascular elements, the sap-conducting elements. The verticillium wilt fungus does not cause a disintegration or rot of the invaded tissues. Sometimes other organisms may enter the wilt-invaded parts, however.

Verticillium wilt on potato was reported in Europe and America early in the twentieth century. It occurs in the seed-potato areas in the New England, North Central, and Northwestern States.

The wilt fungus is harbored in the tubers and persists in the soil. If conditions are favorable, wilt-free soil can be infested by wilt-infected seed potatoes. Attempts at getting wilt-free seed tubers by cutting off the discolored stem end of infected tubers have met with failure because fungus hyphae may penetrate beyond the discolored section of the tuber.

Roguing wilted tops has reduced the amount of wilt of seed potatoes if the tops in the three hills on each side of the wilted hill also were removed. In some regions the wilt fungus persisted in the soil after a 2-year rotation, but infestation was practically eliminated after a 3- to 4-year rotation.

Some varieties—Menominee, Saranac, and Sequoia—are more resistant than others.

Blackleg, caused by *Erwinia atroseptica*, is recognized by an inky-black lesion on the base of the stem, which is a primary distinguishing character from similar soft rot caused by bacteria.

The blackleg bacteria invade tops and tubers. Infection may enter the stalks from the infected seed piece. Diseased tops manifest slight to severe dwarfing and somewhat rigid and rolled leaves that in the later stages yellow, wilt, and die.

In very wet weather, the inky-black lesions at the base of the stalk may spread to most of the plant. The bacteria enter the new tubers through the stolons of a blackleg stalk and invade the vascular elements, as well as other tissues of the tuber. Affected tubers show soft rot, involving the entire tuber. Under less favorable conditions the decay is arrested so that only the tissues in the center of the tubers are disintegrated.

The blackleg organism is spread from infected to healthy tubers with the seed-cutting knife and the picker planter as well as by contact of freshly cut, healthy seed pieces with diseased seed pieces. The seed-corn maggot and other insects carry the blackleg bacteria and infect the injured plant tissue of the potato.

In very moist weather, bacteria in the soil invade freshly cut or poorly healed seed pieces—a possible explanation for the more general appearance of blackleg in wet than in dry seasons in some places.

Because cuts, bruises, and other injuries permit the entrance of rot-inciting organisms, tubers should be handled carefully to avoid bruising. Storage should be provided with favorable temperatures and humidity for healing over (or suberizing) injured tissues. Removing from the seed potatoes all tubers showing rot and storing cut seed potatoes immediately after cutting at about 70° F. and 80 percent humidity to favor adequate healing of the cut surfaces have been effective control measures in some places.

We know of no varieties that are immune to blackleg, but some contract blackleg less easily than others. In 1947 Carl Stapp, a bacteriologist in Germany, reported that Carnea, Flava, Johanna, Priska, Robusta, Sickingen, and Starkeragis were more resistant to blackleg than the 56 other German varieties he included in his test.
CONTROL OF DISEASES OF POTATOES

RING ROT, caused by Corynebacterium sepedonicum, is a bacterial disease. It is recognized by wilt of the foliage and rot of the vascular ring of the tubers. Chlorosis, or yellowing, and marginal browning and wilting of the leaves are symptoms. In the tubers the disease is detected by a light-yellow discoloration of the vascular elements, which break down and exude a cheesy bacterial and cellular ooze when a tuber is squeezed. When other microorganisms enter, the lesions enlarge, and soft rot of the tuber results. Cankers or fissures often appear on the surface of infected tubers in the later stages.

Bacterial ring rot was found in Maine in 1932, a year after it was reported in eastern Canada. By 1940 it was detected in most of the potato States. In some areas it caused severe losses.

Ring rot apparently is not harbored in the soil, but infected tubers overwintering in the soil may develop infected volunteer plants, which may serve as sources of infection. The disease is spread from infected to healthy tubers by the seed-cutting knife, planting machines, grading equipment, and contaminated hands, gloves, bags, baskets, barrels, and bins that have come in contact with diseased potatoes.

Control involves prevention, sanitation, and the use of resistant varieties.

Control of ring rot is primarily the responsibility of seed-potato growers, who should propagate and maintain their own seed potatoes. Potatoes that are free from ring rot can be kept so if no infected seed potatoes are introduced from other sources. If a grower must bring in seed potatoes from other farms and localities, however, he will be safe if he plants them in separate fields for a few years or until he is absolutely certain that they are free from ring rot.

Seed-potato certifying agencies in most States set up a rigid zero tolerance for ring rot in certified seed potatoes in 1940. Largely as a result of those standards, the spread and extent of ring rot has been reduced greatly, and growers of seed potatoes have access to an adequate supply of healthy potatoes even if they cannot grow their own seed stock.

Sanitation involves selling for table stock the crop that harbors ring rot and disinfecting all equipment, machinery, containers, and bins that in any way were in contact with the diseased potatoes. Bins, crates, barrels, and bags can be treated with copper sulfate (25 pounds to 100 gallons of water). Machinery can be treated with formaldehyde (1 part in 10). Ring-rot-free seed potatoes, such are produced by foundation seed-potato growers or on State foundation seed-potato farms, should be planted. By following this clean-up program many potato growers have eradicated ring rot from their farms.

Although no variety is immune to ring rot, some varieties contract the disease less easily than others. Teton, introduced in 1946, is an example.

VIRUS DISEASES of potatoes have been reported in every country. Leaf roll and latent mosaic apparently occur most widely.

The nature and cause of virus diseases have been under investigation for more than 100 years. Many investigators now regard them as filterable proteins that multiply in living cells.

The virus diseases of potatoes can be classified as mosaics and yellows. Distinguishing characters of the mosaic group are mottling and the ability to be transmitted in the sap. A mark of the yellows group is chlorosis; it cannot be transmitted in sap. The mosaic group includes several viruses, some of which have several strains or races that differ in pathogenicity. The yellows group includes the viruses that cause leaf roll, witches'-broom, and aster yellows.

As with most of the fungus diseases, the potato virus diseases are designated by common names that indicate the response or reaction of the plant to the virus. The reactions or symptoms are affected by temperature, host, and the
strain or race of the virus. Mottling usually is absent at higher temperatures. Roll of the leaves is favored by relatively high temperatures. Some varieties manifest the effects of a virus; another variety might act as a symptomless carrier of the same virus. One race of a virus can cause a reaction in the same variety that differs from the reaction induced by another race. Most of the potato viruses are transmitted by sucking insects (aphids and leafhoppers) but some are spread also by chewing insects. Some viruses are spread by mere contact of diseased plants with healthy plants and by mechanical agents, such as the seed-potato cutting knife and planting machines.

Although several mosaic diseases incited by as many different viruses affect the potato, I shall describe only three—latent mosaic, virus A, and vein banding mosaic, which occur in many potato regions.

Latent mosaic, caused by virus X, is so called because many varieties of potatoes are symptomless carriers. Synonyms of the latent mosaic virus are potato virus X, potato-acronecrosis virus, simple mosaic virus, potato virus 16, *Solanum* virus 1, and *Annulus dubius*.

Latent mosaic virus is harbored by potato varieties more generally than any of the other potato mosaic viruses. Almost every plant in most of the older varieties harbors the virus. Many varieties manifest the milder strains of this mosaic by slightly light-green and rugose leaves when the weather is cool.

Latent mosaic virus has several races or strains that induce a range of reactions, varying from weak or no apparent symptoms to severe necrosis. A plant harboring one race may be protected against infection from other races of this virus. More than one race may be harbored by a plant, suggesting mutation or simultaneous infection by more than one race.

Other plants in the nightshade family that are susceptible to latent mosaic are tomato, tobacco, pepper, jimsonweed, petunia, physalis, henbane, woody nightshade, and black nightshade. Jimsonweed is a good one for detecting different races of latent mosaic virus. It reacts to the races by manifesting no symptoms for the weaker races to severe necrosis for the more virulent races. Globe amaranth, or bachelor’s button, which manifests red local lesions, also is a good host for detecting latent mosaic virus.

Latent mosaic is easily transmitted mechanically. Contact of diseased with healthy plants and the cutting knife spread the disease. Some sucking insects, such as aphids, fail to transmit latent mosaic, but grasshoppers are reported to transmit it.

Reductions in yield of 10 to 30 percent are reported for the common races of latent mosaic. Virulent races can cut yields more than 50 percent. Because most of the old varieties harbor latent mosaic, it is possible that yields from them are 10 or 15 percent lower than the yields of healthy stock.

Virus A often induces slightly light-green and rugose leaves, symptoms that are similar to those induced by virus X, the cause of latent mosaic. The symptoms are favored by cool temperatures and are masked at high temperatures. Some varieties merely show light-green foliage. Others develop dead spots in the foliage.

Although tobacco and some other species in the nightshade family are susceptible to virus A, the symptoms often are so like those induced by virus X that diagnosis is difficult.

Virus A is transmitted by aphids. It can also be transmitted in the sap. It is transmitted mechanically less easily than virus X. Virus A apparently involves several races, which vary in degree of reaction they induce on the host plants.

Virus A reduces yields by 15 to 25 percent in some varieties, apparently by causing plants to mature too early. It does not affect the set of tubers.

Since most of the old varieties gen-
erally harbor virus X, they carry a composite infection after contracting the aphid-transmitted virus A. On many varieties the two viruses induce more pronounced symptoms than those of one of them, such as distinct mottling and crinkling. This composite infection is recognized as mild mosaic, a term first used to designate mildly affected plants, as distinguished from severely dwarfed, curled, rolled, and necrotic plants.

Vein banding mosaic, caused by virus Y, is distinguished by rugosity, or wrinkled surface of the leaf, and vein clearing in early stages and by necrotic spots and streaks and leaf drop in the later stages. The symptoms vary in different varieties, ranging from slight rugosity to severe dwarfing, curling, and necrosis. Synonyms for vein banding mosaic virus are potato virus Y, Solanum virus 2, and Marmor upsilon.

Vein banding mosaic occurs in leading potato areas of every country. In some places a high percentage of the crop is infected.

Susceptible hosts, besides potato, are tomato, black nightshade, henbane, woody nightshade, petunia, Solanum nodiflorum, Datura innoxia, and tobacco.

The several races of vein banding mosaic vary in virulence. Vein banding mosaic virus is readily transmitted by sap inoculation, grafting, and aphids. It is responsible for a greater reduction in yield than that caused by either virus X or virus A. Infected plants die prematurely; the result is undersized tubers. Usually less than half a crop is obtained from diseased plants.

Because the older potato varieties generally harbor virus X, they harbor two viruses after contracting vein banding mosaic virus (virus Y). This composite infection is known as rugose mosaic, which often causes more severe host reactions than either one of the viruses. On some varieties, however, the vein banding mosaic virus induces as severe reactions as the composite infection.

Leaf roll is recognized by upward cupping or rolling of the leaflets, first on the lower leaves and finally on the entire plant. An infected plant has a leathery texture. Chlorosis, or a generally light-green color, especially in the upper leaves, frequently appears, even before roll of the leaves begins. In colored varieties, reddish discoloration also may appear. The plants are barely half as tall as healthy plants. The tubers are smaller in size and number. In some varieties internal tuber discoloration, known as net necrosis, develops only as a result of current-season infection with the leaf roll virus.

Leaf roll occurs in potato regions in every country. In some areas all potatoes are infected so that growers in such areas do not know their stock is infected. They suppose that leaf roll symptoms are characteristic of a healthy crop.

Synonyms for the leaf roll virus are potato phloem necrosis virus, potato leaf roll virus, potato virus 1, Solanum virus 14, and Corium solani. Apparently the leaf roll virus involves more than one race. Other hosts are tomato, jimsonweed, woody nightshade, black nightshade, and ground-cherry.

Leaf roll has not been transmitted by sap inoculation, but is transmitted by tuber and stalk grafts. Aphids, especially the green peach aphid, Myzus persicae, are vectors. Heavy infestations of aphids, mild temperatures, and absence of diseases and parasites of aphids contribute to the extensive spread of leaf roll.

Leaf roll is responsible for reduction in size of plants and tubers, as well as in set of tubers. Reductions in yield might be as high as 50 percent. In hot and dry weather, leaf roll infected plants produce chiefly culls.

Control of potato virus diseases involves the use of isolated tuber-unit
seed plots or fields and the resistant varieties.

Because insects transmit virus diseases, it is useless to attempt to grow healthy plants near potato fields that harbor the diseases. Healthy potatoes can be grown in fields that are surrounded by woods and located at a distance from other potato fields. The healthiest seed stock should be used for planting the isolated field, and it should be planted as soon as the soil is tillable. Planting the tubers in tuber units—planting seed pieces of the same tubers in consecutive order—facilitates detection and removal of diseased plants. Weekly inspections, beginning when the plants are about 6 inches tall, to detect and remove diseased plants will help control the diseases.

The number of plants that contract diseases increases as the season advances. Some growers therefore practice early harvesting. That means harvesting seed potatoes about 10 weeks after planting. The most effective (but laborious) method is to pull the tops so that the tubers can be harvested later when storage conditions may be more favorable. Killing the tops with a weed killer frequently fails to kill the entire stalk that produces leaves, so that aphid infestation and virus transmission are encouraged unless the tubers are harvested a few days after the tops die.

In tests in northern Maine in 1932 to 1947, two highly susceptible varieties remained free from virus diseases when harvested August 1, even though they had been grown within 500 feet of diseased potatoes. Samples of the varieties, left on the same plots until harvest on September 15, had contracted 90 to 95 percent of mosaic and leaf roll in seasons when aphids were numerous. The tests show that susceptible potatoes can be maintained free from virus diseases in some seed-potato-producing areas, even with meager isolation, if the seed potatoes are harvested early in the season before the dispersal of virus-carrying insects.

Seed-potato certification agencies in different States inspect and certify potatoes that meet their standards. To have potatoes inspected, the grower notifies the agency, which charges a small fee for the service. Certified seed potatoes, though not necessarily disease-free, usually are superior to noncertified potatoes for seed. Certification, more than any other factor, encourages growers to produce healthy seed potatoes.

Potatoes vary in their resistance to virus X. Some varieties contract virus X in more than 50 percent of the plants. Under like conditions other varieties become infected in less than 10 percent of the plants. Still other varieties are immune. A variety that is immune to virus X is immune to all the races of virus X that have been tested so far. The varieties immune to virus X harbor this immunity not only against contact and sap inoculation but also against infection in stalk and tuber grafts. Grafting a scion that is susceptible to virus X onto a stock that is immune to virus X results in formation of aerial tubers on the scion and provides a method for identifying varieties immune to virus X in segregating progenies.

Protective inoculation against races of virus X involves inoculating a susceptible variety with a weak race of the virus for protection against infection from stronger or more virulent races of the virus. As the stronger races of virus X cause greater reductions in yield than the weak races, the potato stocks inoculated with weak races are protected against this reduction.

Some varieties susceptible to virus A contract it less easily than others. Some varieties are immune. Some are symptomless carriers, manifest top and tuber necrosis, and are light green and rugose, or show mottling. Immune varieties are of two types—virus-A, immune to aphid but not to graft infection, and virus-A, immune to both
aphid and graft infection. Varieties included in the second type of virus A immunity manifest this immunity by top and tuber necrosis, a hypersensitive reaction. Examples of varieties of the second type of virus A immunity are Irish Cobbler, Earlainc, and segregating progenies of those varieties. Examples of the first type of virus A immunity are Katahdin and Chippewa, as well as segregating progenies of them.

Observations on the reaction of different varieties to virus Y disclose that many of them develop dead spots on the leaves and streaking of veins, petioles, and stems, which is associated with brittleness and leaf drop. Rugosity and vein clearing appears on some varieties. Other varieties manifest light-green and rugose leaves to so slight a degree that they may be mistaken for healthy plants.

Although no varieties immune to virus Y have been found, some varieties are highly resistant in the field. Katahdin and Chippewa are field resistant to virus Y under average conditions to aphid infestation. Under heavy aphid infestation, however, they contract considerable virus Y infection, an indication that the resistance is affected by the number of aphids.

In reaction to leaf roll, caused by the phloem necrosis virus, some varieties develop distinct foliage chlorosis, roll, and dwarfing. Others manifest those symptoms to a slight degree. Varieties vary in resistance to leaf roll. Under average field conditions Katahdin contracts leaf roll in less than 10 percent of the plants where less resistant varieties contract this disease in more than 50 percent of the plants. Some of the new seedling potatoes are more highly resistant to leaf roll than Katahdin.

Investigations on potato diseases by the United States Department of Agriculture and State experiment stations have contributed much helpful information on identification, means of spread, disease resistance, and control of late blight, scab, ring rot, leaf roll, and viruses A, X, and Y. There is need for additional knowledge on the causal agents as to the origin and development of races; pathological anatomy; disease development in relation to moisture, temperature, soil reaction and nutrition; and the nature of disease resistance.

Problems in control involve devising and evaluating new fungicides and studies on antibiotics designed to find a product that is systemic and persists during the growing season.

A primary objective in disease resistance involves breeding varieties that are immune from as many of the major potato diseases as possible and that possess other desirable characters such as quality, yield, and adaptability.

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For further reading:
Reiner Bonde, E. S. Schultz, and W. P. Raleigh: Rate of Spread and Effect on Yield of Potato Virus Diseases, Maine Agricultural Experiment Station Bulletin 427, 1943.
E. S. Schultz, Reiner Bonde, and W. P. Raleigh: Isolated Tuber-Unit Seed Plots for Control of Potato Virus Diseases and Black Leg in Northern Maine, Maine Agricultural Experiment Station Bulletin 370, 1934; Early Harvesting of Healthy Seed Potatoes for Control of Potato Diseases in Maine, Maine Station Bulletin 427, 1944.