www.nysipm.cornell.edu/factsheets/vegetables/potato/late_blight_fs.pdf
1998
Fact Sheet 726.20

VEGETABLE CROPS

Cornell University

Integrated Pest Management

Late Blight of Potatoes and Tomatoes

Phytophthora infestans

William E. Fry
Department of Plant Pathology, NYS College of Agriculture and Life Sciences, Cornell University

Late blight of potatoes and tomatoes, the disease that was responsible for the Irish potato famine in the mid-nineteenth century, is caused by the fungus-like oomycete pathogen *Phytophthora infestans.* It can infect and destroy the leaves, stems, fruits, and tubers of potato and tomato plants. Before the disease appeared in Ireland it caused a devastating epidemic in the early 1840s in the northeastern United States.

P. infestans was probably introduced to the United States from central Mexico, which is its center of origin. After appearing in North America and Europe during the 1840s, the disease spread throughout most of the rest of the world during subsequent decades and had a worldwide distribution by the beginning of the twentieth century.

Severe late blight epidemics occur when *P. infestans* grows and reproduces rapidly on the host crop. Reproduction occurs via sporangia that are produced from infected plant tissues (Fig. 1) and is most rapid during conditions of high moisture and moderate temperatures (60°-80°F). Sporangia disperse to healthy tissues via rain splash or on wind currents. Reproduction is asexual; each sporangium is an exact copy of the one that initiated the parent lesion, and each can initiate a new lesion.

Phytophthora infestans affects several different plant species and has the potential to cause devastating disease almost everywhere potatoes are grown. It is also a serious pathogen on tomatoes in cool, wet climates. In central Mexico it is a parasite or pathogen of many different wild *Solanum* species. In Canada and the United States *P. infestans* has been reported to infect hairy nightshade (*Solanum sarachioides*), bittersweet (*S. dulcamara*) and Petunia (*Petunia hybride*) in addition to potatoes and tomatoes. In South America it has been reported as an important pathogen of pear melon (*S. muricatum*).

Recent Introductions of Exotic Strains

During the early 1990s several exotic strains of *P. infestans* were introduced from Mexico. These strains have increased the severity of late blight on potato and tomato because they are more aggressive than earlier ones in the United States and Canada. They initiate infections more quickly and reproduce more profusely, causing epidemics to occur rapidly. To combat these strains it is necessary to use more resistant potato and tomato cultivars or to use fungicides more intensively. Unfortunately, resistance of potato foliage and stems is not necessarily related to tuber resistance. For example, though the foliage of the cultivars Allegany and Elba is moderately resistant, the tubers are quite susceptible.

Potato cultivars with desirable market qualities and whose foliage and tubers show high levels of resistance are being developed. Traditional methods include using breeding lines or cultivars as sources of resistance; new methods include using wild species as sources of resistance and employing molecular techniques in which genes responsible for resistance are transferred into potato cultivars via genetic engineering techniques.

Though some commercial tomato cultivars are more susceptible than others, few are resistant. Under conditions that are favorable to late blight, epidemics in tomatoes seem to be more rapid than in potatoes. Some sources of resistance have been identified in wild relatives of tomato, and efforts are being made to bring higher levels of resistance into cultivars that are desirable for the market.

Figure 1A. Late blight lesion on potato foliage. White "fuzz" is *Phytophthora infestans* sporulating from the lesion. (photo: R. V. James)

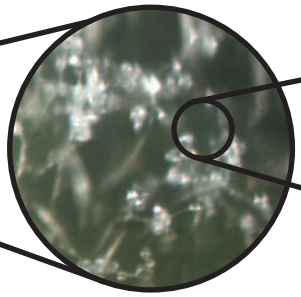

Figure 1B. Lesion as seen through a hand lens or dissecting microscope. Sporangiophores with sporangia are interspersed among leaf hairs. (photo: K. Loeffler)

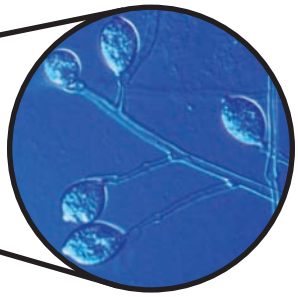

Figure 1C. Micrograph of sporangia (lemon-shaped structures) on sporangiophores. Each sporangium is about 0.030 mm in length.

Figure 2. Potato tubers infected with *P. infestans*: external symptoms (left) (photo: T. A. Zitter), typical granular rot of internal tissues (right).

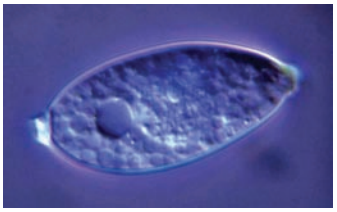

Figure 3. Sporangia of *P. infestans*: intact sporangium (left), sporangium from which a zoospore is germinating (right). (photos; B. G. Turgeon)

DISEASE CYCLE

Figure 16. Tomato plants completely defoliated by late blight.

Figure 15. Tomato leaflet with late blight lesions. (photo: C. Smart)

In most of the United States and Canada, *Phytophthora infestans* requires a living host to survive between seasons. Usually it lives in infected potato tubers (Fig. 2). which can survive in storage or the soil (to become volunteers) after harvest or anywhere potatoes might be discarded. Tubers that have been discarded at any stage of crop production or handling (harvest, storage, shipping, spring cleanup, or planting) are known as “culls.” Culls may survive if they are not destroyed (frozen, crushed, composted, or buried at least 2 feet beneath the soil surface). Infected tubers that are planted or cull tubers that survive the winter may be sources of the pathogen that initiate epidemics the following season.

P. infestans is usually dispersed aerially one to several miles from the overwintering site to living potato or tomato foliage via sporangia (Figs. 1,3 left), which can survive exposure to dry, sunny conditions for up to an hour and even longer under cloudy conditions. Sporangia can germinate within a few hours after landing on potato or tomato foliage if free moisture (e.g., dew, rainfall, sprinkler irrigation, fog) is present. Germination takes place either indirectly via zoospores (Fig. 3 right) or directly via a germ tube that penetrates into foliage, stems, or fruit to initiate infections. Infections are visible as small lesions after three to four days. Necrotic areas on some lesions are only 1 to 2 mm in diameter (Fig. 4) Lesions enlarge as the pathogen grows through the tissues, and the pathogen can sporulate from older lesions when the environment is favorable (leaf wetness for more than 10 to 12 hours at moderate temperatures [60°-70°F]) (Fig. 5). Sporulation may occur on lesions that are only four to six days old. Under dry conditions no sporulation occurs and the lesion has a brown dead center, surrounded by host tissue that has collapsed and appears either water soaked, gray-green, or yellowed (Fig. 6). Both tomato and potato fruits are susceptible (Figs. 7, 8). Their stems may be infected (Figs. 9, 10), and stem lesions are capable of producing sporangia for a longer time than can lesions on leaves.

Disease development (growth and reproduction of the pathogen) is favored by moderate temperatures (60°-80°F and wet conditions. It can develop in very warm daytime temperatures (ca. 95°F) if conditions are extremely wet and night

Figure 14. Piles of potatoes discarded owing to infections by *P. infestans,* followed by severe soft rot.

Figure 13. Field of potatoes completely destroyed by late blight. (photo: S. B. Goodwin)

Figure 4. Potato leaflet with many very young (small) late blight lesions.

Figure 5. Potato leaflets with older (six to eight days old), sporulating lesions.

Figure 6. Potato leaflet with older lesion that is not sporulating.

Figure 7. Tomato fruits infected with *P. infestans.* (photo: T. A. Zitter)

temperatures are moderate (60°-75°F). Epidemics can be rapid and devastating because of the high reproductive potential of this pathogen. Individual lesions can produce 100,000 to 300,000 sporangia per day. Each sporangium is capable of initiating a new infection that will become visible within three to four days and produce sporangia within another day or two under optimal conditions. Thus rapid reproduction of the pathogen and destruction of leaflets can defoliate potatoes or tomatoes and completely destroy healthy fields in a short time (Figs. 11-13). Such epidemics result from many sequential cycles of infections: every lesion produces many sporangia, each of which can be dispersed to a new leaflet to initiate a new infection, which in turn can produce many sporangia, and so on.

Tubers may be infected by *P. infestans* whenever sporangia and tubers come into contact, from early in the tuberization process until harvest. Infections most commonly occur when sporangia are washed from lesions on stems and foliage to the soil and then through the soil to tubers. Infections can occur on developing or mature tubers, but contact between tubers and sporangia is more likely when the tubers are enlarging; tuber enlargement creates cracks in the soil and gives sporangia ready access. Tubers become infected most often when soils are cool and wet (near field capacity); soil temperatures higher than 65°F seem to suppress infections. Because sporangia can survive days or weeks in soil, tubers can become infected for a period of time after infections in the foliage are no longer producing sporangia.

Tubers infected by late blight are especially susceptible to soft rot. If some tubers in a crop are infected, store the crop in cool, dry conditions. If infected potatoes are stored at high relative humidity and moderate temperatures, soft rot can be severe, destroying infected tubers first but subsequently destroying previously healthy ones (Fig. 14).

Infections can probably also occur during harvest and subsequent handling. Although late blight inoculations during storage were previously considered highly unlikely, such occurrences have been documented recently.

Tomato leaflets (Fig. 15) can be destroyed at least as rapidly as potato leaflets, leading to complete defoliation (Fig. 16) in a short time.

Figure 8. Green tomato fruits infected with *P. infestans*. The pathogen is sporulating from the infected fruits. (photo: T. A. Zitter)

Figure 9. Potato stem infected with *P. infestans.*

Figure 10. Infected tomato plants with lesions on stems and foliage. (photo: M. McGrath)

Figure 12. Field of potatoes with severe late blight.

Figure 11. Field of healthy potato plants.

Control

Use of integrated management practices is necessary for successful suppression of potato or tomato late blight. In the absence of sexual reproduction *P. infestans* requires a living host to survive between seasons. Therefore, sanitation (elimination or exclusion of infected plant parts from a farm) is important in the overall management strategy. Ideally, no infected potatoes should be present in the vicinity of the crop. Volunteer plants that might be infected should be destroyed. Cull potatoes should be frozen, crushed, fed to livestock, or buried under at least 2 feel of soil. Only tubers that are free of *P. infestans* should be planted. The "Certified" grade for seed potatoes allows up to 1 percent late blight infection. Growers should request information from the seed potato producer as to whether late blight was observed during field or harvest inspections.

After planting, additional precautions will reduce the chances of successful inoculations and can suppress development and reproduction of the pathogen. Using resistant cultivars (Figure 17) will reduce the chances of infection and slow the pathogen growth rate if some infections develop. Early in the season, the lowest labeled rate of protectant fungicide will provide protection and thus prevent a rapid epidemic. Fungicide should be applied either at an appropriate regular interval for the production area or adjusted on the basis of weather. Several forecasting systems that identify favorable weather conditions are available (e.g., Blitecast, Tomcast) and can be used to adjust the intensity of scouting as well as the frequency of fungicide applications. Hilling of potatoes increases the amount of soil between tubers and the soil surface and thus helps protect tubers from sporangia that land on the soil surface.

Scouting. Regular inspections of growing crops are important to the overall management of late blight. Because topography and crop growth can influence the microclimate encountered by the pathogen, late blight may be detectable earlier in some areas than in others. It is likely to appear first in wet areas (low spots in the field, areas adjacent to woods and hedgerows, dense crops, or areas adjacent to other features that might shade crops), especially when the macroclimate has been less than optimal for pathogen development.

Protectant fungicides are often needed from mid- to late season when plants are growing actively and have a dense canopy. Applications should be repeated regularly to replace fungicide that has been washed or abraded away and to protect foliage produced since the last application. It is during this time that the more effective fungicides are needed. (Consult current Cooperative Extension recommendations for specific information.) Applications should be more frequent during weather that is favorable to late blight (wet with moderate temperatures) than in unfavorable weather (dry foliage and very cool [<50°F] or very hot temperatures).

Applying fungicides. Protectant fungicides are most effective if applied more frequently at low labeled dosages than less frequently at high dosages. This is partly because more frequent applications ensure better coverage. Coverage can be poor when applications are made using very low volumes of water (less than 15 to 20 gallons/acre). Some application systems such as electrostatic sprayers can achieve good coverage with very low volumes of water (5 gallons/acre), but most systems that produce large droplets and small volumes will achieve poorer coverage than those that use smaller droplets and larger volumes of water.

Treating a crop exposed to inoculum. Fungicides that have systemic activity (penetrate into plant tissues) are necessary if a crop has been exposed to sporangia within the last 24 hours. Even if the first infections occurred more than 24 hours earlier, if lesions are visible in the crop and a systemic has not been applied, an effective systemic will probably provide some benefit that is not possible from a protectant. Protectant fungicides (those that are not systemic and cannot penetrate tissue) are ineffective against the pathogen once it has penetrated the cuticle (sometimes within two hours of germination). Thus applications of a protectant fungicide will have no visible effect on disease suppression until six to nine days after application because it takes that long for lesions to be easily visible. Unfortunately, even systemic fungicides (at least those available in 1998) do not suppress all infections and will have little effect on infections that are more than 24 to 48 hours old. Effects from systemic fungicides may be visible within three to four days.

Treating "hotspots." A hotspot is a group of infected plants located amid relatively healthy ones. If very little disease is present in the crop and there are only a few hotspots, the latter should be destroyed as quickly as possible by flaming, disking, and burying the infected foliage or killing the plants with a rapidly acting herbicide. Plants immediately surrounding the hotspot should also be destroyed because they are very likely infected even though the infections are not yet visible. If fungicides are being used, the remainder of the field should

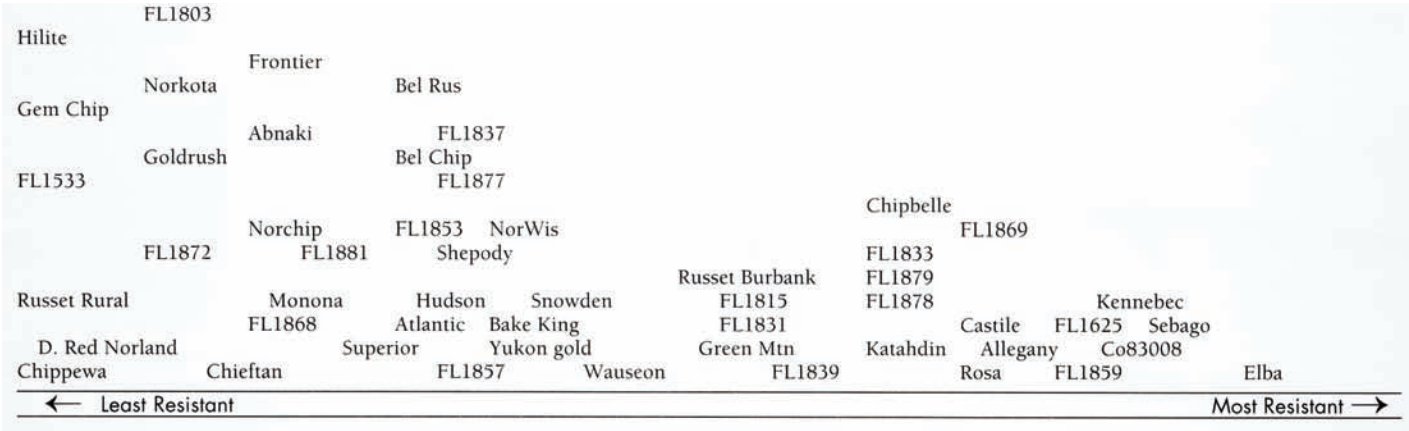

Figure 17. Relative resistances of potato cultivars. Cultivars that are least resistant are shown on the left-hand side and most resistant ones are shown on the right-hand side. This ranking is derived from a series of experiments in different locations (see Inglis et al. 1996.) This ranking considers foliage reactions only and not tuber reactions (which may be different from foliage reactions).

be treated with a fungicide that has some systemic activity, and subsequently, applications of a protectant fungicide should be applied on a tight (frequent) schedule.

Treating established infections. Once 5 to 10 percent of the foliage is infected it is usually not possible to halt the development or progress of the disease. Currently available (1998) systemic fungicides are inadequate to halt an epidemic at this stage. Only weather that is very dry and hot, both day and night, might temporarily stop the epidemic. Stem infections are very resistant to drying, however, and will sporulate when sufficient moisture is available. Growers can attempt to salvage apparently uninfected tomato fruit but should be aware that some fruit infections will not become visible for several days. Foliage in such fields should be promptly destroyed to prevent spread to nearby fields or farms.

Forecasting. Forecasting schemes and fungicide registrations are constantly changing. Consult Cooperative Extension for the latest information on fungicide registrations, efficacy, and forecasting information.

Using fungicides at planting. At the time of this writing, it appears that some systemic oomycete-specific fungicides can protect healthy seed tubers during the seed cutting process. If there is a chance that some seed tubers are infected, use of an effective fungicide will lower the chances that late blight will develop in the subsequent crop. Consult Cooperative Extension recommendations for the most current information.

The Future

Late blight in the United States may need to be managed very differently in the future than in the past. Before exotic strains were introduced in the early 1990s, the late blight pathogen could only reproduce asexually via sporangia. Sexual reproduction requires two individuals of different mating type (A1 and A2), and before the 19905 all strains were of the same mating type. Both mating types of *P. infestans* (AI and A2) are now present in the United States and Canada, however, and have sometimes come into contact. Thus sexual reproduction is now theoretically possible.

The spores resulting from sexual reproduction are called oospores. Oospores are thick-walled dormant structures (Fig. 18) that can survive in the absence of living plant tissue. The occurrence of oospores could change the epidemiology of the disease because they can survive in soil over winter or summer (if soil temperatures do not exceed 40°C [= 102°F]). If oospores are produced, the soil may become a source of this pathogen, therefore adding an entirely new dimension to the epidemiology of *P. infestans* and the control of late blight. The result will be new "sources" of the pathogen. At the time of this writing (1998), there is no evidence that oospores are contributing to the epidemiology of late blight of potatoes or tomatoes in the United States. Nonetheless, the possibility of sexual reproduction exists and growers and scientists need to be alert to this development.

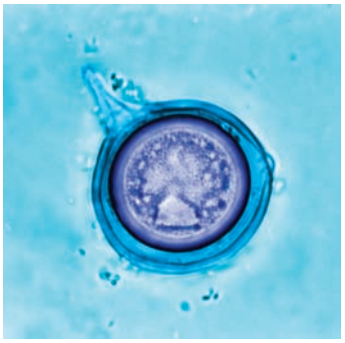

Figure 18. Oospores of *P. infestans*.

Sexual reproduction will also yield recombinant individuals thus providing a supply of "new" genotypes among the progeny. Whereas the majority of recombinant progeny are expected to be less problematic than parental strains, it is possible that some progeny could be more problematic.

Summary

Successful management of late blight relies on an integration of the following tactics: removing sources of the pathogen by eliminating cull potatoes and volunteers and planting only healthy seed tubers; using resistant cultivars when possible and as they become available; scouting locations where late blight might appear first; using a forecasting scheme to gain early warning of weather that is favorable to disease and to adjust frequency of fungicide application or the intensity of scouting and using appropriate protectant or systemic fungicides. After harvest, store potato tubers at cool temperatures under conditions sufficiently dry that there is no free moisture on tuber surfaces. Control tactics are constantly modified as new information and technology become available, so consult the latest Cooperative Extension publications for the best recent specific recommendations.

Useful References

Bouma, E., and H. T. A. M. Schepers, eds. 1997. Proceedings of the workshop on the European network for development of an integrated control strategy of potato late blight. NL -8200 AK Lelystad (the Netherlands), Applied Research for Arable Farming and Field Production of Vegetables.

Cornell University. (annually). Pest Management Recommendations—Commercial Vegetable and Potato Production Ithaca, N.Y.: Cornell Cooperative Extension.

Fry, W E., and S. B. Goodwin. 1997a. Re-emergence of potato and tomato late blight in the United States. Plant Disease, 81:1349-57

Fry, W E., and S. B. Goodwin. 1997b. Resurgence of the Iris! Potato Famine Fungus. Bioscience 47:363-71.

Fry, W. E., and D. Shtienberg. 1990. Integration of host resistance and fungicide to manage potato diseases. Canadian Journal of Plant Pathology 12:111-16.

Inglis, D. A., D. A. Johnson, D. E. Legard, W. E. Fry, and P. B Hamm. 1996. Relative resistances of potato clones in response to new and old populations of *Phytophthora infestans*. Plant Disease 80:575-78.

Ingram, D. S., and P. H. Williams. 1991. *Phytophthora infestans,* the Cause of Late Blight of Potato. London: Academic Press. 273 pages.

Produced by the New York State Integrated Pest Management Program, which is funded through Cornell University, Cornell Cooperative Extension, the NYS Department of Agriculture and Markets, the NYS Department of Environmental Conservation, and USDA-CSREES. Design by Media Services, Cornell University. Layout by Karen English, New York State IPM Program. Cornell Cooperative Extension provides equal program and employment opportunities.

Posted 8/09 at www.nysipm.cornell.edu/factsheets/vegetables/potato/late_blight_fs.pdf.