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The Standard Guide to Late Blight of Potatoes: Causes, Symptoms, and Solutions

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The late blight disease of potatoes is the most devastating disease of potatoes in the world. It is most destructive, however, in areas with frequent cool, moist weather. Zones of high late blight severity include the northern United States and the east coast of Canada, Western Europe, central and southern China, south-eastern Brazil, and the tropical highlands.

- Late blight is also very destructive to tomatoes and some other members of the family Solanaceae. Late blight may kill the foliage and stems of potato and tomato plants at any time during the growing season.
- It also attacks potato tubers and tomato fruits in the field, which rot either in the field or while in storage.
- Late blight may cause total destruction of all plants in a field within a week or two when weather is cool and wet. Even when losses in the field are small, potatoes may become infected during harvest and may rot in storage. The historical aspects of late blight of potatoes in relation to the Irish famine and the establishment of *Phytophthora infestans* as the cause of late blight are presented

Symptoms

- Symptoms appear at first as water-soaked spots, usually at the edges of the lower leaves.
- In moist weather the spots enlarge rapidly and form brown, blighted areas with indefinite borders.
- A zone of white, downy mildewy growth 3 to 5 millimetres wide appears at the border of the lesions on the undersides of the leaves. Soon entire leaves are infected, die, and become limp. Under continuously wet conditions, all tender, aboveground parts of the plants blight and rot away, giving off a characteristic odor.
- Entire potato plants and plants in entire fields may become blighted and die in a few days or a few weeks.
- In dry weather the activities of the pathogen are slowed or stopped. Existing lesions stop enlarging, turn black, curl, and wither, and no oomycete appears on the underside of the leaves. When the weather becomes moist again the oomycete resumes its activities and the disease once again develops rapidly.
- Affected tubers at first show purplish or brownish blotches consisting of water-soaked, dark, somewhat reddish brown tissue that extends 5 to 15 millimetres into the flesh of the tuber. Later the affected areas become firm and dry and somewhat sunken. Such lesions

may be small or may involve almost the entire surface of the tuber without spreading deeper into the tuber interior.

- The rot, however, continues to develop after the tubers are harvested. Infected tubers may be subsequently covered with sporangiophores and spores of the pathogen or become invaded by secondary fungi and bacteria, causing soft rots and giving the rotting potatoes a putrid, offensive odor.
- Tomato leaves, stems, and fruit are also attacked. Entire tomato fields may be destroyed. Fruitmay rot rapidly in the field or in storage.

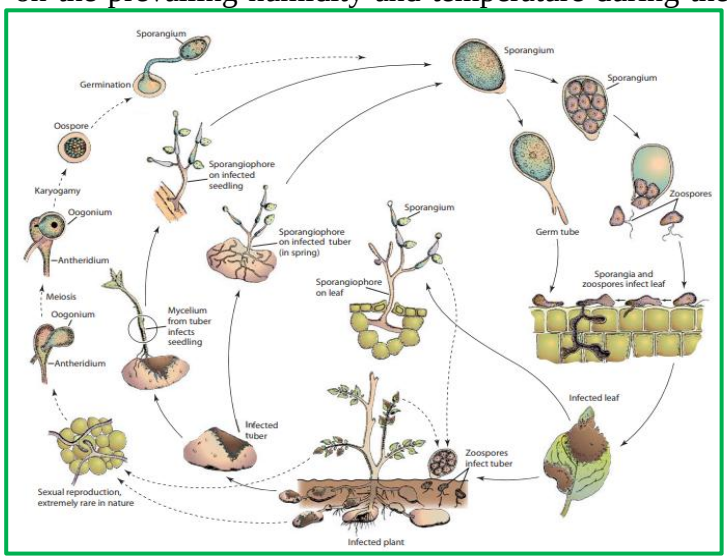
Pathogen - (*Phytophthora Infestans*)

- The mycelium produces branched sporangiophores that produce lemon-shaped sporangia at their tips.
- At the places where sporangia are produced, sporangiophores form swellings that are characteristic for this oomycete.
- Sporangia germinate almost entirely by releasing three to eight zoospores at temperatures up to 12 or 15°C, whereas above 15°C sporangia may germinate directly by producing a germ tube.
- The oomycete requires two mating types for sexual reproduction. Until the late 1980s, only one mating type was present in countries outside Mexico. Since then, however, both mating types have become widely distributed in most countries and, as a result, new strains of the pathogen have appeared. Some of the new strains are much more aggressive than the old ones and quickly replace them. When the two mating types grow adjacently, the female hypha grows through the young antheridium (= male reproductive cell) and develops into a globose oogonium (= female reproductive cell) above the antheridium.
- The antheridium then fertilizes the oogonium, which develops into a thick-walled and hardy oospore. Oospores germinate by means of a germ tube that produces a sporangium, although at times the germ tube grows directly into the mycelium.

Development of Disease

- The pathogen strains that prevailed until the 1980s belonged to mating type A1 and reproduced in the absence of its compatible mating type A2, i.e., asexually. Therefore, they did not produce oospores and overwintered only as mycelium in infected potato tubers.
- Spread of the compatible mating type A2 from Mexico to the rest of the world has made possible the sexual reproduction of the pathogen, which results in the production of oospores in infected aboveground and belowground potato and tomato tissues. Usually, the more susceptible the potato variety the more oospores the pathogen produces per unit leaf area.
- Oospores may survive in the soil for 3–4 years. Such oospores not only can overwinter in the soil, they also make possible the production of new more virulent strains through genetic recombination of pathogenic characteristics of the mating strains.

- During infection, a number of potato defence-related genes are induced (activated) by the pathogen, including genes coding for b-1,3-glucanase, known to be induced in many host–pathogen systems, genes coding for enzymes involved in detoxification, and several other types of genes involved in plant defence against pathogens.
- The mycelium from infected tubers or from germinating oospores and zoospores spreads into shoots produced from infected or healthy tubers, causing discoloration and collapse of the cells.
- When the mycelium reaches the aerial parts of plants, it produces sporangiophores, which emerge through the stomata of the stems and leaves and produce sporangia.
- The sporangia, when ripe, become detached and are carried off by the wind or are dispersed by rain; if they land on wet potato leaves or stems, they germinate and cause new infections.
- The germ tube penetrates directly or enters through a stoma, and the mycelium grows profusely between the cells, sending long, curled haustoria into the cells. Older infected cells die while the mycelium continues to spread into fresh tissue.
- A few days after infection, new sporangiophores emerge from the stomata of the leaves and produce numerous sporangia, which are spread by the wind and infect new plants. In cool, moist weather, new sporangia may form within four days from infection; thus, a large number of asexual generations and new infections may be produced in one growing season. Wherever the two mating types A1 and A2 are present together in the same plant tissue, fertilization may take place and oospores may be produced.
- The frequency of oospore formation and their role in the development of the disease within a growing season are not yet known. In any case, as the disease develops, established lesions enlarge and new ones develop, often killing the foliage and reducing potato tuber yields.
- The second phase of the disease, the infection of tubers, varies between potato varieties and pathogen isolates. It begins in the field when, during wet weather, sporangia are washed down from the leaves and are carried into the soil.
- Emerging zoospores germinate and penetrate the tubers through lenticels or through wounds. In the tuber the mycelium grows mostly between the cells and sends haustoria into the cells. Tubers contaminated at harvest with living sporangia present on the soil or on diseased foliage may also become infected.
- Most of the blighted tubers rot in the ground or during storage. The development of late blight epidemics depends greatly on the prevailing humidity and temperature during the different stages of the life cycle of the oomycete.
- The oomycete grows and sporulates most abundantly at a relative humidity near 100% and at temperatures between 15 and 25°C. Temperatures above 30°C slow or stop the growth of the oomycete in the field but do not kill it, and the oomycete can start to sporulate again when the temperature becomes favourable, provided, of course, that the relative humidity is sufficiently high.



Control

- Late blight of potatoes can be controlled successfully by a combination of sanitary measures, resistant varieties, and well-timed chemical sprays.
- Only disease-free potatoes should be used for seed. Potato dumps or cull piles should be burned before planting time in the spring or sprayed with strong herbicides to kill all sprouts or green growth.
- All volunteer potato plants in the area, whether in the potato field or in other fields, should be destroyed, as any volunteer potato plant can be a source of late blight infection.
- The recent introduction of the A2 mating type and the potential for mating and production of hardy oospores that can survive the winter in the soil may drastically change our ability to control late blight by the means just described.
- Only the most resistant potato varieties available should be planted. Unfortunately, most popular commercial potato varieties are more or less susceptible to late blight.
- The blight oomycete comprises a number of races, which differ from one another in the potato varieties that they can attack. Several potato varieties resist one or more races of the late blight oomycete. Some of them are resistant to vine infection but not to tuber infection.
- Many varieties possess so-called field resistance, which is a partial resistance of varying degrees but is effective against all races of the blight oomycete. However, it is not sufficient to rely on varietal resistance to control late blight, as, in favourable weather, late blight can severely affect these varieties unless they are sprayed with a good protective fungicide. Even resistant varieties should be sprayed regularly with fungicides to eliminate, as much as possible, the possibility of becoming suddenly attacked by races of the oomycete to which they are not resistant. However, it is always advisable to use resistant varieties, even when sprays with fungicides are considered the main control strategy, because resistant varieties delay the onset of the disease or reduce its rate of development so that fewer sprays on a resistant variety may be needed to obtain a satisfactory level of control of the disease.
- Various computerized light forecasting systems (e.g. Blightcast) have been developed and are used. Several broad-spectrum and systemic fungicides are used for late blight control.
- The new strains of the oomycete produced as recombinants of fertilization of the two mating types are resistant to some of the systemic (metalaxyl) and, therefore, sprays with such materials are ineffective against such strains.
- Protective spraying of foliage usually affects a considerable reduction in tuber infection. However, when partially blighted leaves and stems are surviving at harvest time, it is necessary to remove the aboveground parts of potato plants or destroy them by chemical sprays (herbicides) or mechanical means to prevent the tubers from becoming infected.
- Experimental but not yet practical control of the disease has been obtained by the pre-treatment of tomato plants with the chemical dl-3-amino-butyric acid or pre-inoculation with tobacco necrosis virus, both of which induce systemic-acquired resistance (SAR) in the tomatoes, protecting them from late blight infection.
- Haustoria formation and growth of hyphae in SARinduced leaves against *P. infestans* appear inhibited, different, and damaged.
- Certain pathogenesis-related proteins accumulate in the leaves of treated plants and only in plant wall papillae and in the cell walls of the oomycete pathogen. Whether these changes play a significant role in resistance to the disease is not clear.