

Early blight biology and control in potatoes

by Phillip Wharton and Elisabeth Wood

Introduction

Early blight, caused by *Alternaria solani* (E. & M.) Jones and Grout (*Hyphomycetes, Hyphales*), is a very common disease of potato and is found in most potato growing areas. Although it occurs annually to some degree in most production areas, the timing of its appearance and rate of its progress determine the impact on the potato crop.

The disease occurs over a wide range of climatic conditions and depends in a large part on the frequency of foliage wetting from rainfall, fog, dew, or irrigation, on the nutritional status of foliage, and on cultivar susceptibility. Though losses rarely exceed 20%, if left uncontrolled, losses can be higher and impact stored potatoes. In Idaho and other potato-growing areas, fungicide treatment helps contain losses to less than 5%.

In the Pacific Northwest, foliar infection is the most critical phase of the disease. However, in some regions, particularly the Rocky Mountain production areas, tuber infection can be more important than foliage infection. Despite its name, the disease rarely develops early and usually appears on mature foliage.

Symptoms

Foliar symptoms of early blight first appear as small, irregular to circular, dark-brown spots on the lower (older) leaves. These spots may range in size from a pinpoint to 1/4 inch in diameter (figure 1). As the spots enlarge, they become restricted by leaf veins and take on an angular shape (figure 2).



Figure 1. Early blight lesions first appear as small, irregular to circular dark-brown spots on lower leaves. Spots range in size from a pinpoint to 1/4 inch.



Figure 2. Early blight lesions are characterized by an alternating series of light tan and dark-colored concentric rings surrounded by a narrow band of chlorotic tissue.



Figure 3. Early blight lesions on young leaves may be up to 1/2 inch in size. Unlike late blight lesions, they are angular and become restricted by leaf veins.



Figure 5. On severely infected leaves, the small early blight lesions may coalesce over large areas of the leaf.



Figure 4. Late in the growing season, the upper leaves and stems of infected potato plants may be peppered with early blight lesions.

Early in the growing season, lesions on young, fully expanded, succulent leaves may be larger—up to 1/2 inch in diameter—and may, because of their size, be confused with late blight lesions (figure 3). Leaf lesions are relatively easy to identify in the field because lesion development is characterized by a series of dark concentric rings alternating with bands of light-tan tissue (figure 2). Elongated, superficial, brown or black lesions may also form on stems and petioles (figure 4).

By the end of the growing season, the upper leaves of infected potato plants may be peppered with numerous, small early blight lesions, and, subsequently, lesions may coalesce to cover a large area of the leaf (figure 5). Severely infected leaves eventually wither and die but usually remain attached to the plant. Severe infection of foliage by the early to mid-bulking period can result in smaller tubers, yield loss, and lower tuber dry matter content.



Figure 6. (a) Early blight lesions on tubers are usually circular to irregular and slightly sunken, often surrounded by a raised, purple to dark-brown border. (b) Underlying tissues are leathery to corky in texture.

Tuber symptoms of early blight include circular to irregular lesions that are slightly sunken and often surrounded by a raised, purple to dark-brown border. The underlying tissues are leathery to corky in texture, dry, and usually dark brown (figure 6). These lesions reduce the quality and marketability of fresh market tubers. Tuber infection also presents

a challenge to processors because tuber lesions often require additional peeling to remove the darkened lesions and the underlying tissues.

Disease cycle

The dark-colored spores (figure 7) and mycelia of the pathogen (*Alternaria solani*) survive between growing seasons in infested plant debris and soil, in infected potato tubers, and in overwintering debris of susceptible solanaceous crops and weeds, including hairy nightshade (*Solanum sarrachoides*). Overwintering spores and mycelia of *A. solani* are melanized (darkly pigmented) and can withstand a wide range of environmental conditions, including exposure to sunlight and repeated cycles of drying, freezing, and thawing.

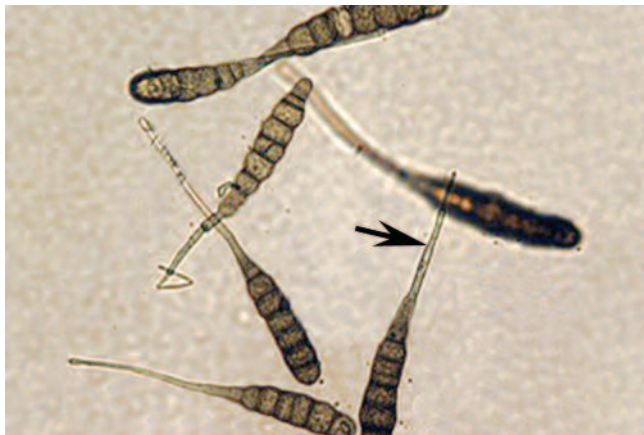


Figure 7. Spores of *Alternaria solani*, causal agent of early blight, are melanized and can withstand a wide range of environmental conditions. Note the transverse and vertical septa and the long “beak” (arrow).

In spring, spores (conidia) serve as primary inoculum to initiate disease (figure 8). Plants grown in fields or adjacent to fields where potatoes were infected with early blight the previous season are most prone to infection because large quantities of overwintering inoculum are likely to be present from the previous crop. The initial inoculum readily moves within and between fields, carried by air currents, windblown soil particles, splashing rain, and irrigation water.

Spores of *A. solani* (figure 7) are produced on potato plants and plant debris at temperatures between 41° and 86°F (the optimum is 68°F). Alternating wet and dry periods with temperatures in this range favor spore production. Few spores are produced on plant tissue that is continuously wet or dry.

The dissemination of inoculum follows a diurnal pattern in which the number of airborne spores increases as leaves that are wet with dew or other sources of nighttime moisture dry off, relative humidity decreases, and wind speeds increase. The number of airborne spores generally peaks in midmorning and declines in late afternoon and at night.

Spores landing on leaves of susceptible plants germinate and may penetrate tissues directly through the epidermis, through stomata, and/or through wounds such as those caused by sand abrasion, mechanical injury, or insect feeding. Free moisture (from rain, irrigation, fog, or dew) and favorable temperatures (68° – 86°F) are required for spore germination and infection of plant tissues. Lesions begin to form 2 to 3 days after initial infection.

Many cycles of early blight spore production and lesion formation occur within a single growing season once primary infections are initiated. Secondary spread of the pathogen begins when spores are produced on foliar lesions and carried to neighboring leaves and plants. Early blight is largely a disease of older plant tissues and is more prevalent on senescing tissues on plants that have been subjected to stresses induced by injury, poor nutrition, insect damage, or other causes.

Early in the growing season, the disease develops first on fully expanded leaves near the soil surface and progresses slowly on juvenile tissues near the growing point. The rate of disease spread increases after flowering and can be quite rapid later in the season during the bulking period and during periods of plant stress. Early blight lesions are often found on most leaves of unprotected plants late in the growing season.

In potato tubers, germinated spores penetrate the epidermis through lenticels and mechanical injuries to the skin. Tubers often become contaminated with *A. solani* spores during harvest. These spores may have accumulated on the soil surface or been dislodged from desiccated vines during harvest. Infection is most common on immature tubers and those of white- and red-skinned cultivars since they are highly susceptible to abrasion and skinning during harvest. Coarse-textured soil and wet harvest conditions also favor infection.

In storage, individual lesions may continue to develop, but secondary spread does not occur. Infected tubers may shrivel through excessive water

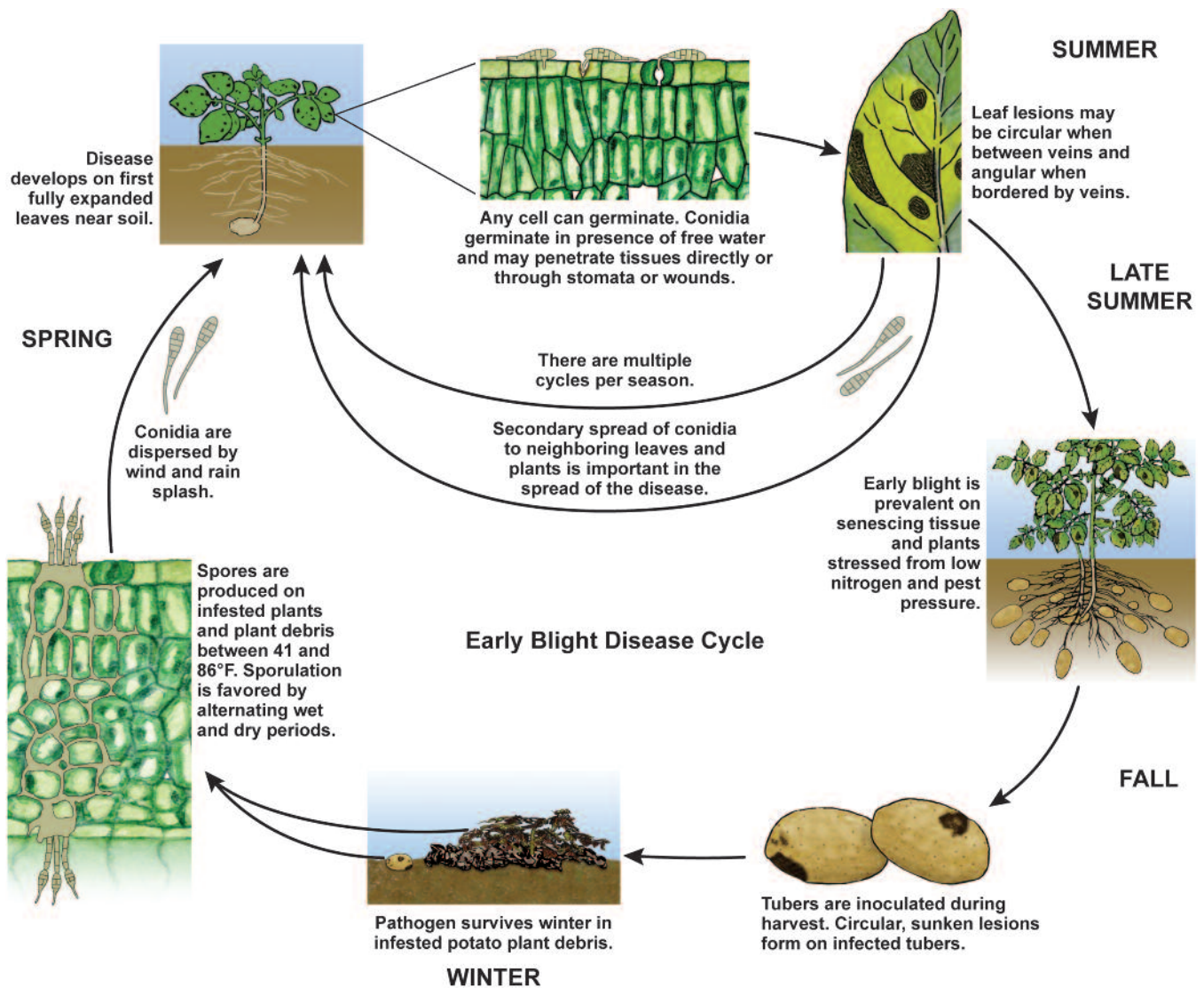


Figure 8. The disease cycle of the early blight pathogen, *Alternaria solani*.

loss, depending on storage conditions and disease severity. Early blight lesions on tubers, unlike late blight lesions, are usually not sites of secondary infection by other decay organisms.

Monitoring and control

Effective management of early blight requires implementation of an integrated disease management approach. The disease is controlled primarily through the use of cultural practices, resistant cultivars, and foliar fungicides.

Cultural control

Cultural practices such as crop rotation, removing and burning infected plant debris, and eradicating weed hosts help reduce the inoculum level for subsequent plantings. Because *A. solani* persists in

plant debris in the field from one growing season to the next, rotation with non-host crops (e.g., small grains, corn, or soybean) reduces the amount of initial inoculum available for disease initiation.

Other cultural control measures include:

- Avoiding irrigation in cool, cloudy weather
- Timing irrigation to allow plants time to dry before nightfall
- Using certified disease-free seed
- Using tillage practices such as fall plowing to bury plant refuse

To minimize tuber infection after harvest, tubers should be stored under conditions that promote rapid suberization because *A. solani* is unable to infect through intact periderm.

Resistant cultivars

Cultivars with good levels of field resistance are available, but no immunity to early blight has been found in commercial potato cultivars or in their wild parents. Highly susceptible cultivars such as Red Norland, Norchip, and Superior should be avoided in locations where early blight is prevalent and disease pressure is high. Field resistance to foliage infection is associated with plant maturity.

Chemical control

The most common and effective method for the control of early blight is the application of foliar fungicides. Protectant fungicides recommended for late blight control (e.g. maneb, mancozeb, chlorothalonil, and triphenyl tin hydroxide) are also effective against early blight when applied at approximately 10- to 14-day intervals.

The strobilurin and carboxamide groups of fungicides are the most commonly used to control early blight in Idaho. These include azoxystrobin and pyraclostrobin in the strobilurin group and boscalid in the carboxamide group.

Resistance to the strobilurin and carboxamide groups of fungicides has been reported in Idaho and other potato growing regions in the U.S. The geographical spread of this resistance is not fully known so applications of both strobilurins and carboxamides such as azoxystrobin and boscalid should always be made in combination with tank-mixtures of protectant fungicides such as chlorothalonil or EBDCs.

Other products that have shown efficacy against early blight include famoxodone, pyrimethanil, fenamidone, and difenoconazole.

Several new products were registered for control of early blight in 2012. These include fluopyram, penthiopyrad, and fluxapyroxad. These fungicides are highly effective against early blight but all belong to the carboxamide group with a similar chemistry to boscalid. This means that they could be susceptible to the development of fungicide

resistance and thus fungicide-resistance management techniques should be applied when using them.

The application of foliar fungicides is not necessary in plants at the vegetative stage, when they are relatively resistant. Accordingly, spraying should commence at the first sign of disease or at row closure (between rows). The frequency of subsequent sprays should be determined according to the genotype and age-related resistance of the cultivar. Protectant fungicides should be applied initially at relatively long intervals and subsequently at shorter intervals as the crop ages.

Early season applications of fungicides before secondary inoculum is produced often have minimal or no effect on the spread of the disease. In Idaho, early blight can be adequately controlled by relatively few fungicide applications if the initial application is properly timed. In Idaho, this is usually row closure. Predictive models to time the first application are commonly used in the U.S. These have been more effective in midwestern U.S. potato growing regions such as Michigan and Wisconsin. The first application for early blight control should be timed at 200 P-days after emergence.

For more information, please visit:
<http://www.idahopotatodiseases.org>

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