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EC1897

Sugar Beet Seedling Diseases



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Knowing the factors and pathogens that influence sugar beet disease can help producers manage crops more efficiently.

Introduction

Sugar beets are susceptible to a number of seedling diseases that can occur as seed rots, preemergence and postemergence damping-off, or postemergence infection of roots or hypocotyls. Many factors may influence the incidence and severity of disease including environmental issues, pathogen inoculum potential, and host susceptibility.

Pathogens that most commonly cause seedling disease problems in sugar beets in Nebraska include:

- · Rhizoctonia solani Kuhn,
- Aphanomyces cochlioides Drechs.,
- Pythium ultimum Trow,
- · Pythium aphanidermatum (Edson) Fitzp., and
- Phoma betae (A. B. Frank).

Because numerous other environmental and cultural factors may also cause symptoms easily confused with those caused by the pathogens, you must correctly

identify and differentiate among the various diseases in order to most effectively manage them.

Because the various disease symptoms have similar traits, it's sometimes necessary to use other diagnostic methods such as growth characteristics in culture and favoring temperatures (*Table 1*) to distinguish among pathogens.

Furthermore, it's vital to recognize and differentiate seedling disease symptoms because all pathogens additionally can cause later root rot disease throughout the season. Root rots are more commonly encountered in Nebraska than seedling diseases, and may continue to cause problems in the storage piles after harvest, further reducing yield and profit. Recognizing seedling disease symptoms early will help you anticipate root diseases later so that prompt action may be taken.

The two most important and consistently occurring pathogens causing both seedling and root rot diseases in Nebraska are *Rhizoctonia solani* and *Aphanomyces cochlioides*. Although symptoms presented in this publication were induced artificially under greenhouse conditions, they are consistent with what would be observed in the field.

Seedling Disease Terminology

Damping-off — a disease that results in death or decay of seeds or seedlings at germination in soil (preemergent); most evident in young emerged seedlings that suddenly wilt or die from a rotting at base of stem (postemergent).

Cotyledon — the seed leaf; one present in monocots (corn, wheat), and two in dicots (beets, beans).

Hypocotyl — the portion of the developing seedling just below the point at which the cotyledons are attached (upper stem).

Holdfast — a special structure by which a fungus becomes attached to its host.

Sclerotium (**pl. sclerotia**) — hard, sterile, vegetative body of a fungus made up of compact masses of thick-walled, hyphal cells that resist adverse environmental conditions and may remain dormant in soil for long periods of time.

Hypha (**pl. hyphae, adj. hyphal**) — basic vegetative unit of structure and function for most fungi; primarily microscopic, tubular-shaped filaments that constitute the body (mycelium) of the fungus.

Mycelium — mass of tubular, interwoven hyphae that make up the vegetative body of most fungi.

Septate (**n. septation, pl. septa**) — having more or less regularly occurring cross-walls; hyphae being divided by partitions or septa.

Coenocytic — nonseptate; hyphae having no partitions or septa.

Zoospore — an asexually produced fungus spore bearing a whip-like tail and capable of independent movement in water.

Oospore — thick-walled sexually-produced spore in the oomycete fungal class that serves as an overwintering mechanism in soil.

Sporangium (**pl. sporangia**) – a spore case for fungi; commonly a saclike fungal structure that is filled with an indefinite number of asexual spores (sporangiospores). Zoospores are contained in zoosporangia.

Table 1. Comparison of characteristics for distinguishing among seedling disease pathogens.

Pathogen	Colony color	Growth in culture (inches per day)	High moisture requirement
Aphanomyces	White	0.5-0.75	Yes
Phoma	Dark gray to black	0.25-0.50	No
Pythium ultimum	White	1.5-2.0	Yes
Pythium aphanidermatum	White	1.5-2.0	Yes
Rhizoctonia	Light gray to tan	1.0-1.25	No
	Initial symptoms	Temperatures favoring infection	Preemergence damping-off
Aphanomyces	Black thread-like hypocotyls; no wilting of leaves	68-85°F	No
Phoma	Black hypocotyls, with wilting of leaves	62-68°F	Yes
Pythium ultimum	Wilting and complete collapse of all leaves	Below 68°F	Yes
Pythium aphanidermatum	Wilting and complete collapse of all leaves	85-95°F	Yes
Rhizoctonia	Wilting and complete collapse of all leaves	Above 70°F	Rarely

Symptoms and Favorable Environmental Conditions

R. solani

R. solani causes both pre- and postemergence damping-off, but is most often observed causing disease on seedlings after emergence. Infection begins as dark brown lesions below the soil surface and progresses up the hypocotyls, resulting in wilting and complete collapse of cotyledons and plant death (*Figure 1*). Infection does not occur in soil temperatures below 15°C (60°F), but can occur anytime above 20°C (68°F).



Figure 1. Wilting symptoms and death of sugar beet seedlings due to *R. solani*.

A. cochlioides

A. cochlioides damage is observed primarily after emergence and is favored by warm soil temperatures ranging from 20° - 30°C (68° - 85°F), but does not generally affect initial stand establishment.

Infections begin near the soil line as water-soaked lesions that progress from gray to black (*Figure 2*, inset). Stems become characteristically dark, thin and thread-like with an absence of cotyledonary wilting (*Figure 2*), which are the major symptoms for distinguishing it from *R. solani* infections.



Figure 2. Initial infection at soil line (inset), and advanced symptoms (thin, thread-like hypocotyl with lack of wilting of cotyledons) characteristic of sugar beet infection by *A. cochlioides*.



Figure 3. Wilting symptoms and death of sugar beet seedlings due to *Pythium* spp.

Plants often recover, but stands may still be impacted later; infected plants are often more susceptible to breakage and death from wind damage due to the weakened, delicate stems.

Pythium

There are hundreds of *Pythium* species known to cause disease in various crops, but those species most often associated with seedling problems in sugar beets include *P. ultimum* and *P. aphanidermatum*.

P. ultimum is considered to be a cool-weather pathogen, responsible for seed rot and preemergence damping-off. Although it can grow in the range of 5° - 35°C (40° - 95°F), with optimal soil temperatures ranging between 24° - 30°C (72° - 85°F), it primarily causes seedling disease below 20°C (68°F). In contrast, *P. aphanidermatum* is a hotweather pathogen preferring soils ranging from 30° - 35°C (85° - 95°F) for infection. Postemergence damping-off symptoms of either *Pythium* species are indistinguishable from those of *R. solani* and consist of wilting, stem lodging, and often death of seedlings (*Figures 1* and 3).

Phoma

Although preemergence damping-off due to *Phoma betae* can occur if infested seeds are planted into cool wet soils [4° - 12°C (38° - 52°F)], the majority of damage occurs after emergence in soil temperatures of 16° - 20°C (62° - 68°F). Symptoms consist of wilting and darkbrown-to-black necrosis on hypocotyls (*Figure 4*). Little infection occurs above 25°C (78°F).

Affected seedlings may often survive and recover to varying degrees, but the pathogen may also persist in the crowns, causing leaf spots and root rots. It is a major



Figure 4. Wilting and black hypocotyl symptoms on sugar beet seedlings characteristic of infection by *Phoma betae*.

contributor to storage rots in harvest piles after the growing season.

Pathogen Survival and Disease Cycle

R. solani

R. solani survives as resistant, thickened overwintering structures (sclerotia) or hyphae in crop residues and soil. The sclerotia germinate during the spring (hyphae also grow through soil) and infect sugar beet crowns, petioles and roots. Even if you find that the pathogen is known to be present in your fields, its disease severity and incidence will still depend on soil populations and environmental conditions.

The conditions required for *R. solani* disease development are not as rigorous as those required by *Pythium* or *Aphanomyces*, both of which require watersaturated soils. Therefore, *R. solani* is problematic under a much wider range of environmental conditions and geographic locations. It is the most widespread, consistently occurring pathogen of sugar beets in Nebraska. *R. solani* additionally has a broad host range that includes many species of weeds and other crops that may be grown in rotation with sugar beets.

A. cochlioides

A. cochlioides produces two types of spores during the infection process. Oospores are sexually produced spores that allow the pathogen to overwinter. These oospores are circular, thick-walled structures that can survive for long periods in soils under adverse conditions.

Zoospores are the other type of spore produced by *A. cochlioides*. Zoospores are motile, tadpole-like spores

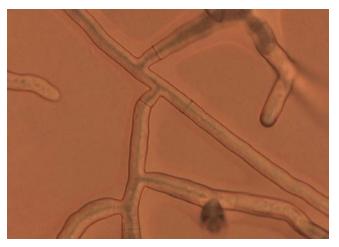


Figure 5. Hyphal growth of *R. solani* in agar culture, consisting of right-angle branching and cross-walls (septations) just above hyphal junctions.

that can swim independently through soils high in water content, and are produced asexually.

A. cochlioides disease initiates when soils become warm and very wet. Under these conditions, the overwintering resting spores (oospores) germinate and can infect plants directly, or through the production of zoospores. Saturated soils help this disease to progress rapidly throughout fields and cause significant losses.

Zoospores also help initiate secondary infections during the growing season, depending on soil environmental conditions. Disease from *A. cochlioides* is limited to plants related to sugar beets.

Pythium

Pythium spp., like *A. cochlioides*, overwinters in soil as thick-walled, sexually-produced oospores. These oospores germinate and infect plants, or may also produce zoospores for multiple infections under high soil moisture conditions. Because *Pythium* species have wide host ranges, they can affect numerous crop and weed species common to western Nebraska.

Phoma

P. betae is primarily considered a seedborne pathogen, and does not survive for more than two years on host residue in soils as conidia or mycelium. Evidence of soilborne inoculum has not been demonstrated. P. betae can also survive on common weeds, and has been found on the roots of common lambsquarters (Chenopodium album L.). Infection in seed fields comes from airborne spores produced by the sexual stage of P. betae (Pleospora bjoerlingii) overwintering on seed stalks from previous crops in the area.



Figure 6. Comparison of slow, curly A. cochlioides growth (left) and expansive, spreading R. solani (right) hyphal growth in agar culture.

Pathogen Identification

R. solani

Colony growth of *R. solani* in a water culture primarily floats and spreads widely across the water surface. No spores are formed, but the mycelium is septate with cross-walls located just above the hyphal branches (*Figure 5*). A similar, rapidly spreading pattern of growth is observed in agar culture (*Figure 6*, right), and most pathogenic isolates grow rapidly (1 - 1.25 in/day) on all substrates.

A. cochlioides

A. cochlioides growth in water culture consists only of unbranched, cylindrical tubes. These hyphal tubes serve as vessels that release zoospores (swimming spores similar to tadpoles) from the tips of the hyphae arranged in a single row. The emerged spores then encyst and collect at the tips of the hyphal tubes (Figure 7). Secondary zoospores are released from the primary zoospore cysts, swim in the surrounding water, and serve as the source of new and repeated infections. This entire process will occur within 12 - 14 hours at room temperature, and is diagnostic for A. cochlioides.

Within the cortex of young seedlings or in water culture, *A. cochlioides* produces abundant nonseptate mycelium (*Figure 8*). This distinctive hyphal growth is easily distinguished morphologically from *R. solani* and *Pythium* spp. (*Figures 5* and 9), and by several other macroscopic characteristics, including presence of hyphae with curly colony edges produced on half-potato dextrose agar (PDA) (*Figure 6*, left), and slower growth overall (0.75 in/day) at room temperature.



Figure 7. Encysted, primary zoospores of *A. cochlioides* clustering at tip of sporangium. Note empty cysts after secondary zoospore release.

Pythium

Both *Pythium* species grow very rapidly, (1.5 - 2 in/day) on common media such as PDA, and produce branched, coenocytic (without cross-walls) mycelium (*Figure 9*). Initially this growth occurs beneath the agar surface, but after colonies reach edges of plates, growth results in thick, cottony, aerial mycelium.

In water or grass blade cultures, *P. ultimum* grows from seedling tissue and produces abundant, branched, nonseptate mycelium, primarily below the water surface, as opposed to that of *R. solani*. After 24 to 48 hours, spherical sporangia (asexual spore-bearing structures) are produced (*Figure 10*). This species may also produce spherical oospores.

In water culture, tissue infected by *P. aphaniderma-tum* also yields nonseptate mycelium, but later produces large, inflated lobed zoosporangia of varying lengths



Figure 9. Hyphal growth of *Pythium* spp., consisting of slender, branched tubes.

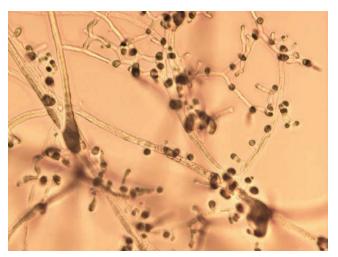


Figure 8. Morphologically distinct hyphal growth of A. cochlioides in agar culture.

(*Figure 11*). Zoospores are formed within and exit the lobed zoosporangia, swim for a period, encyst, and germinate by the production of germ tubes. Oospores, (*Figure 12*) are also produced by *P. aphanidermatum*.

Phoma

P. betae is less distinctive and therefore not as easily identified in water culture as the above-described pathogens. However, growth of P. betae on water agar results in distinctive structures resembling holdfasts or appressoria on the bottom of a plastic Petri dish (Figure 13). Examinating the inverted dish under a low-power microscope easily confirms the presence or absence of the pathogen. Furthermore, growth of P. betae on half-PDA cultures is readily distinguished from the previously described fungi by producing grayish-black colored aerial colonies as seen from above, and black when viewed from the underside of plates.

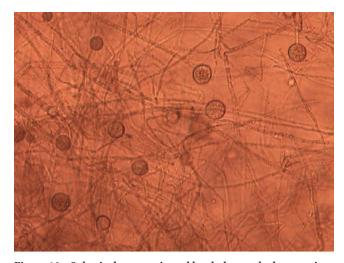


Figure 10. Spherical sporangia and hyphal growth characteristic of some *Pythium* spp.



Figure 11. Irregular, lobate sporangia characteristic of *P. aphanidermatum*.

Management

- No genetic resistance is currently available for any of these seedling diseases. Although root rot tolerance is an available trait for several of the pathogens, resistance is not expressed until later in the season.
- Planting early into cool soils will help avoid or escape disease problems from all pathogens with the exception of *P. ultimum*, which could still occur in cooler soils. If you establish a vigorously-growing stand before soils begin to warm, it will help plants withstand infection later.
- Managing weeds will also help to reduce initial pathogen inoculum for sugar beets, since common weeds are also hosts for several of these pathogens, including lambsquarters, pigweed, and Kochia.

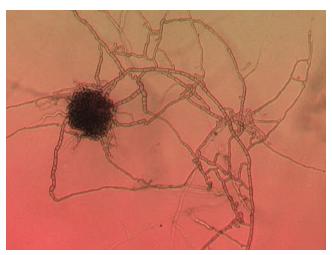


Figure 13. Holdfasts viewed from bottom of Petri dish, characteristic of *Phoma betae* growth on water agar.

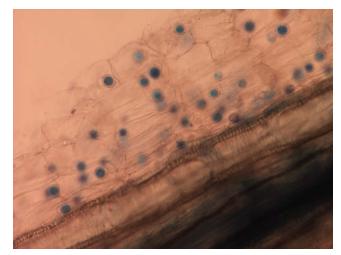


Figure 12. Oospores embedded in sugar beet root tissues. These sexually produced spores also serve as the long-term overwintering means for *A. cochlioides* or *Pythium* spp. in soils.

- Crop rotation is less effective for managing *Rhizoctonia* and *Pythium* than the others, due to the large host ranges for these pathogens. Furthermore, all pathogens except *Phoma* can survive many years in soils due to production of long-lived spore types. *Phoma* is normally introduced into fields as a seedborne pathogen.
- Chemical seed treatments are available for protecting against all pathogens. Unfortunately no one fungicide will inhibit all pathogens. Knowing some of the disease history in your fields and how to distinguish between pathogens will help you make the best treatment choice, if necessary.
- Several biological control options by mycoparasites (fungi that are pathogenic on other fungi) and resident bacteria are currently being tested experimentally but are not yet ready for commercial use.

This publication has been peer reviewed.

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