Identifying and Distinguishing Seedling and Root Rot Diseases of Sugar Beets

Robert M. Harveson, Panhandle Research and Extension Center, University of Nebraska, Scottsbluff 69361

Corresponding author: Robert M. Harveson. rharveso@unlnotes.unl.edu


Introduction
Sugar beets are susceptible to a number of seedling and root rot diseases that are primary constraints to profitable sugar beet production (5,11,14,15,33,43). The majority of the seedling diseases are caused by soilborne pathogens, but *Phoma betae* is a seedborne fungal pathogen that can cause both seedling and mature root problems during the season (19,20). Since numerous other environmental and cultural factors may also cause symptoms that could be easily confused with symptoms caused by these pathogens, it is important to be able to correctly identify and differentiate between the various diseases and problems in order to most effectively manage them.

All of the seedling pathogens can additionally be involved with root rot diseases throughout the season; however only the most prevalent and important root rot diseases will be addressed. For example, two of the seedling pathogens (*Pythium* and *Phoma*) can cause root rots later in the season (21,23,33,43), but will not be discussed further as their impact is generally minor in comparison with the other more common root-rotting pathogens. The two most important pathogens causing both seedling and root rot diseases are *Rhizoctonia solani* and *Aphanomyces cochlioides* (16,33,43). Three additional root rot diseases not generally considered to cause serious seedling problems will also be discussed, including Fusarium yellows, Fusarium root rot, and rhizomania. The goal of this publication is to help others learn to identify and effectively differentiate the various seedling and root rot diseases common to sugar beet.

**Seedling Diseases**

**Disease: Damping-off, Black Root Complex**

**Primary hosts.** Sugar beet (*Beta vulgaris* L.).


**Symptoms.** *R. solani* causes both pre- and post-emergence damping-off, but is most often observed causing disease on emerged seedlings (16,20,33,41,43). Evidence of infection begins as dark brown lesions below the soil surface and progressing up the hypocotyls, often resulting in wilting and complete collapse of cotyledons and death of plants (Fig. 1). Infection does not occur in soil temperatures below 15°C, but can occur at anytime above 20°C (20).
A. cochliloides does not generally affect seedling emergence or initial stand establishment. The damage it causes is primarily post-emergent and is favored by warm soil temperatures ranging from 20 to 30°C (20,29,30). Symptoms begin near the soil line as water-soaked lesions that progress from gray to black. Stems become characteristically dark, thin, and thread-like with an absence of cotyledonary wilting (Fig. 2), which are the major criteria for distinguishing damage from R. solani infections (7,16,33,43). Plants may recover, but stands may still be impacted later as affected plants are often more susceptible to breakage and death from wind damage due to the weakened, delicate stems.

P. ultimum and P. aphanidermatum are those Pythium species most often associated with seedling problems (20). P. ultimum is considered to be a cool-weather pathogen, responsible for seed rot and pre-emergence damping-off at temperatures below 20°C (16,20). P. aphanidermatum, in contrast, is a warm-weather pathogen favored by soils ranging from 30 to 35°C (16,20,23,42). Post-emergent damping-off symptoms of either Pythium species are indistinguishable from those of R. solani, and consist of wilting, lodging, and death of seedlings (Fig. 1).

Although pre-emergence damping-off due to Phoma betae can occur if infested seeds are planted into cool wet soils (4 to 12°C) (19), the majority of damage occurs after emergence in soils at temperatures of 16 to 20°C (19,20). Symptoms consist of dark brown to black necrosis of hypocotyls (Fig. 3), with the diseased tissue often containing small black pycnidia (200 to 325 µm). Little infection occurs above 25°C (16,19,20). Affected seedlings may often survive and recover to varying degrees, but the pathogen is also capable of persisting in the crowns and causing leaf spots, root rots, and storage rots in harvest piles later in the season (19,20,21).
Host range. *Rhizoctonia solani* and both *Pythium* species have wide host ranges, consisting of numerous crop and weed species (2,9,23). The anastomosis groups (AGs) of *R. solani* associated with sugar beets include AG2-2 and AG4 (2,3,35). Seedling disease has most often been attributed to AG4, but isolates belonging to AG2-2 have also been isolated from affected seedlings (35). *Aphanomyces cochlioides* and *Phoma betae* are primarily limited to infecting plants in the Chenopodiaceae, and the closely related Amaranthaceae (19,29).

Geographic distribution. All pathogens are found worldwide wherever sugar beet is grown.

Pathogen isolation. All pathogens are easily isolated from diseased seedlings and grown in pure culture. Several selective media are available for *Aphanomyces* (30,31) and *Pythium* spp. (23,25); however the pathogens can also be rapidly identified from diseased plant tissue in water cultures or low-nutrient media such as water agar or ½-strength potato dextrose agar (PDA). After 24 to 48 h, the characteristic vegetative mycelium along with spores or fruiting bodies can be recognized under low magnification (×50-100), even though multiple pathogens may be simultaneously colonizing seedlings (Fig. 4) (20). The plant pathology program at Scottsbluff, NE routinely uses ½-strength PDA amended with streptomycin sulfate (0.5g/liter). Best results are seen after washing seedlings in water before proceeding. Surface sterilizing diseased seedlings has often been shown to prohibit or reduce the likelihood of pathogen isolation at this stage because of the limited amount of tissue being disinfested.

Pathogen identification. *A. cochlioides* growth in water cultures consists only of unbranched, cylindrical tubes (6-8 × 400-1000 µm) (29,43). These hyphal tubes serve as zoosporangia, and zoospore initials emerge from the tips of the hyphae arranged in a single row. They collect as spherical, encysted spores (usually 7 to 10 µm in diameter) at the tips of the sporangia (Fig. 5) and later (1 to 3 h) emerge as secondary zoospores from the primary zoospore cysts and swim in the surrounding water (20,29,30,45). This entire process will occur within 12 to 14 h at room temperature, and is diagnostic for *A. cochlioides*. 

![Fig. 4. Infected seedling in water culture. Note *Aphanomyces* encysted zoospores (clusters) and lobate sporangia of *Pythium* sp.](image-url)
Within the cortex of young seedlings or in culture, *A. cochlioides* produces abundant nonseptate, but morphologically distinctive mycelium (Fig. 6) (29,30). Macroscopically this distinctive hyphal growth on ½ PDA appears curly at the edge of colonies (Fig. 7, left) and grows more slowly (20 to 22 mm/day) at room temperature than *R. solani* and *Pythium* spp. On the *Aphanomyces*-selective medium, metalaxyl-benomyl-vancomycin agar (MBV) (31), the hyphal growth is not curly, but grows flat and beneath the agar surface with little or no aerial mycelium.

In water culture, colony growth of *R. solani* primarily floats and spreads widely across the surface (20). No spores are formed, but the mycelium is distinctly septate, with cross-walls located just above and on the hyphal branches (2,41) (Fig. 8). A similar rapidly spreading pattern of growth is observed in culture (Fig. 7, right), and most pathogenic isolates grow rapidly (27 to 30 mm/day) on all substrates.
In water cultures, *P. ultimum* grows from seedling tissue and produces abundant branched, nonseptate mycelium 1.7 to 6.5 \( \mu m \) in diameter (20,23,42), primarily below the surface of the water, as opposed to that of *R. solani*. After 24 or 48 hr, spherical terminal sporangia (12 to 28 \( \mu m \) in diameter) (Fig. 9A) and barrel-shaped intercalary sporangia (17-23 × 28 \( \mu m \)) are produced. This species is characterized by mononclinous antheridia, most originating immediately below terminal oogonia, and thick walled, aplerotic oospores, 14.7 to 18.3 \( Fm \) in diameter (Fig. 9B) (42).

In water culture, tissue infected by *P. aphanidermatum* yields nonseptate mycelium 2.8 to 7.3 \( \mu m \) in diameter, and later comparatively large, inflated filamentous or lobed zoosporangia of varying lengths (50 to 1000 \( \mu m \)) (Fig. 10) (20,42). Reniform zoospores (7.5 × 12 \( \mu m \)) are formed within vesicles attached to the lobed sporangia. Zoospores exit the vesicle (Fig. 10 inset), swim for a period, and then encyst and germinate by the production of germ tubes. *P. aphanidermatum* is additionally characterized by terminal, smooth oogonia (20 to 25 \( \mu m \)), mostly intercalary antheridia (Fig. 11), and aplerotic oospores (Fig. 12) 17 to 20 \( \mu m \) in diameter (23,42).

Oospores, in some instances, will form in diseased tissues (Fig. 13) and culture media. However, the formation of other reproductive structures needed for identification of both *Pythium* species (sporangia, oogonia, antheridia, and oospores) are more consistently induced through the use of grass blade cultures. Briefly, this entails using grass blades cut into 1-cm pieces, sterilized by boiling in water for 10 min, and incubating in petri dishes with deionized water and agar blocks of desired cultures at room temperature (23,42). Cultures are then viewed at desired intervals to observe formation of various structures.
Both *Pythium* species additionally grow very rapidly (40 to 45 mm/day) on common media such as PDA, and produce branched, coenocytic mycelium (Fig. 14). Initially, the mycelial growth is beneath the agar surface, but after colonies reach edges of plates, growth results in thick cottony aerial mycelium. After several weeks, this dense mycelial growth collapses and cultures tend to die readily unless continually transferred or stored in some manner. Growth on *Pythium*-selective media such as Mircetich medium is inhibitory (25), and similar to that of *A. cochlioides* on MBV (31). Hyphal growth is closely appressed to the agar surface with little aerial mycelial growth.

*Phoma betae* is not as easily identified in water culture as the above-described pathogens, although pycnidia may sometimes be observed on some seedlings (19,20). However, growth of *P. betae* on water agar results in distinctive structures resembling holdfasts or appressoria on the bottom of a plastic Petri dish (20,22). By examination of the inverted dish under a low-power microscope, the presence or absence of the pathogen is very easily confirmed (Fig. 15).

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**Fig. 13.** Oospores of *Pythium* spp. formed in sugar beet root tissue.

**Fig. 14.** Hyphal growth typical of *Pythium* spp., consisting of branched, coenocytic mycelium.

**Fig. 15.** Holdfasts observed on bottom of Petri dish, characteristic of *Phoma* growth on water agar.
Growth of *P. betae* on ½-PDA cultures is easily 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 (40). *P. betae* also produces pycnidia (225 to 325 µm in diameter) in concentric rings in culture (19,20,40). One-celled, hyaline pycnidiospores (2.6-4.3 × 4.1-7.0 µm) exude from mature pycnidia. Pycnidia and spores vary greatly in size due to environmental influences (20,40).

**Pathogen storage.** *Aphanomyces* and *Pythium* are short-lived in culture and are thus most effectively stored as oospores in soil samples. Fresh isolates should be subcultured from the leading edge of colonies and incubated in oatmeal broth (23,37). Oatmeal broth is made by boiling 5 g of rolled oats in 150 to 200 ml water for 5 min. Oats are removed with cheesecloth, and the remaining volume of broth is brought up to 500 ml. Fifty ml of broth is aliquoted into 125 ml flasks, autoclaved, and inoculated with the pathogen after cooling. After 4 to 6 weeks, contents of flasks are ground in a blender, oospores enumerated with a hemacytometer, and put into sterilized soil. These samples serve as stock cultures, and can be diluted as needed to recover isolates, or included as known concentrations in experiments. Samples can be kept at room temperature for several years (R. M. Harveson, unpublished).

For those *Pythium* isolates from which oospores are unable to be produced, cultures may also be stored on PDA-mineral oil slants, or as colonized hemp seeds, wheat leaf pieces, or agar blocks in deionized water (23). *Aphanomyces* cultures may also be maintained on PDA or oat meal agar (OMA) slants at 12°C with periodic hyphal transfers (every 3 to 4 months) (30).

*Phoma* isolates may also be stored by transferring water cultures to sterilized soil. After cultures have dried, they may be stored at 4°C (21,40). *Rhizoctonia* isolates can be grown on various autoclaved seeds, and stored at 4°C. Barley, corn, or sugar beet seeds have been successfully used to store isolates (2,35). Additionally, dried cultures can be cut into pieces and stored in vials at 4°C for at least 4 years (R. M. Harveson, unpublished).

**Pathogenicity tests.** Inoculum production of individual pathogens for pathogenicity testing consists primarily of the above-described storage methods. *Pythium* and *Aphanomyces* are most efficiently tested by infesting soils with oospores and planting susceptible sugar beet cultivars into them (23,29). However, *A. cochlioides* oospores will not germinate in culture, so recovery or quantification must occur through baiting with live plants (8,30). Pathogens can also be used for pathogenicity testing through production (via water, grass blade cultures, or minimal salts solutions) and inoculation with zoospores (23,26,30,42). For optimal disease development, both pathogens must also be incubated within very moist soils and at appropriate soil temperatures.

Because of a lack of spore production, inoculum of *Rhizoctonia* cannot be easily quantified (2,30,32,43). Therefore it is generally increased on substrates such as seeds or cornmeal and sand, and then quantified as grams of infested material or number of seeds for inoculation purposes (2). The colonized substrate can be incorporated into test soils or placed in contact with sugar beet plants (32). Incubation conditions should also coincide with warm, moist conditions. *Phoma* cultures likewise may be grown on autoclaved seeds for inoculation purposes, or conidial suspensions from sporulating pycnidia may also be utilized (21).

**Root Rot Diseases**

**Disease: Rhizoctonia Root Rot, Root and Crown Rot**

**Primary host.** Sugar beet (*Beta vulgaris* L.).


**Symptoms.** Two different phases of root disease have been reported, one being infections beginning in the crown, accompanied by petiole blackening and death (crown rot) (Fig. 16), often associated with soil thrown up into crowns during early cultivations (32,38). The other phase involves earlier infections occurring on taproots as a tip rot (root rot), and progressing toward the root.
crown (Fig. 17) (14,15,16,33). Root symptoms begin as circular to oval, localized, dark lesions that coalesce to form larger rotted areas of the root as disease progresses (Fig. 18). The extent of rotted tissue often is restricted to external layers of the root (Fig. 19) and does not generally penetrate into the interior until very advanced stages of disease (11,15,16,33). Both phases involve the same type of foliar symptoms, including sudden, permanent wilting and complete collapse and death of leaves and petioles (11,15,16,32,33,38) (Fig. 20).
**Host range.** The host range for *R. solani* is very wide (see above), but AG 2-2 is the one most often associated with root rot infections of mature roots (33,35). Members of this AG have also been reported causing disease in dry-edible beans (3).

**Geographic distribution.** *R. solani* is found worldwide wherever sugar beets are grown.

**Pathogen isolation.** Small root or crown pieces on the margins between healthy and rotted tissues can be surface-sterilized and plated onto common media (water agar or PDA). Observe cultures and watch for distinctive, spreading hyphae with right-angle branching.

**Pathogen identification.** See pathogen identification procedures for *R. solani* seedling disease above.

**Pathogen storage.** See pathogen storage procedures for *R. solani* seedling disease above.

**Pathogenicity tests.** Testing procedures for Rhizoctonia root rot will be the same as for the seedling disease.

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**Disease. Aphanomyces Root Rot**

**Primary host.** Sugar beet (*Beta vulgaris* L.).

**Pathogen.** *Aphanomyces cochlioides* Drechs.

**Symptoms.** Foliar symptoms consist of stunted, yellowed leaves with non-vigorous growth (Fig. 21) (7,11,14,16,29,33,43,45). Wilting may also occur during the day, particularly on warm days, but plants often recover at night. However, permanent wilting is not common, in contrast with Rhizoctonia root rot. Leaves may also take on a scorched appearance and become brittle. Root symptoms begin as yellowish-brown, water-soaked lesions on taproots (Fig. 22) that later become dark and necrotic as they dry (7,14,15,16,33,43). These lesions can occur anywhere on the taproot, but often occur toward the distal end as a tip rot (Fig. 23). If environmental conditions become more favorable for plant growth, plants may recover to produce a relatively healthy crop. However, many roots may still exhibit varying degrees of root distortion and/or scarring (Figs. 24 and 25) (7,14,16,33,43,45). Severe disease can completely destroy taproots leaving little except for crowns (Fig. 26), yet often maintain deceptively healthy-looking tops. Economic loss may also occur in several different ways. Field loss occurs when severely diseased roots are easily dislodged from soil and thrown into furrows during the defoliation process and are not subsequently retrieved by the harvester (Fig. 27) (14). Furthermore, infected roots also continue to rot in storage piles after harvest, which interferes with optimal extraction of sucrose.
Host range. Economic problems from *A. cochlioides* are primarily limited to plants in the Chenopodiaceae and Amaranthaceae; however, several other plant families (mostly weed species) have been reported from greenhouse studies as hosts, including Aioaceae, Caryophyllaceae, Hydrophyllaceae, Linaceae, Paperavaceae, and Portulacaceae (29).

Geographic distribution. *A. cochlioides* is found worldwide wherever sugar beets are grown.

Pathogen isolation. If root lesions are actively growing (Fig. 22), successful isolations may be made on various media, including MBV (30,31) or PDA. However, the pathogen will not grow from old lesions or those from dried, scabby-scarred roots (Figs. 24 and 25), as oospores are formed in tissues and will not germinate in vitro. Shavings from the scabby lesions can readily be used in the greenhouse to bait the pathogen out on sugar beet seedlings (8). The shavings are placed into soils with the seeds and then isolation is performed as described earlier from emerged, diseased seedlings.
**Pathogen identification.** See pathogen identification procedures for *A. cochlioides* seedling disease above.

**Pathogen storage.** See pathogen storage procedures for *A. cochlioides* seedling disease above.

**Pathogenicity tests.** Testing procedures for Aphanomyces root rot will be the same as for the seedling disease.

**Diseases. Fusarium Yellows and Fusarium Root Rot**

**Primary host.** Sugar beet (*Beta vulgaris* L.).

**Pathogens.** *Fusarium oxysporum* Schlect. f. sp. betae Snyd. & Hans. and *F. oxysporum* f. sp. radici-betae.

**Symptoms.** Foliar symptoms of both Fusarium yellows and Fusarium root rot diseases are similar, including interveinal yellowing (Fig. 28), general chlorosis, wilting (Fig. 29), and scorched, brittle leaves (Fig. 29 inset) (11,12,13,33,43). The leaf scorching may be confused with that caused by *A. cochlioides*. However, both *Fusarium* pathogens additionally cause internal necrosis of vascular elements (Figs. 30 A and B), which is characteristic for both diseases, effectively eliminating the possibility of either Rhizoctonia or Aphanomyces root rots (14,15,33,43). The primary difference between the two diseases is the dark external cortical rot of the distal tip of the taproot associated with Fusarium root rot (Fig. 31) (5,12,13,24,33,43). Fusarium yellows symptoms are limited to foliar wilting, interveinal yellowing, and vascular discoloration, but no rotting of the taproot (5,12,13,33).

![Fig. 28. Interveinal yellowing of leaves, characteristic of Fusarium yellows or Fusarium root rot.](image)

![Fig. 29. Wilting, scorched, and brittle leaves due to Fusarium yellows or Fusarium root rot.](image)

![Fig. 30. Necrosis of vascular elements (limited damage on left, severe on right) diagnostic for Fusarium yellows or Fusarium root rot.](image)

![Fig. 31. Severe external, cortical root rot characteristic for Fusarium root rot.](image)

**Host range.** Many isolates are limited to sugar beets, but some have been demonstrated to cause disease in other plant species within the Chenopodiaceae and Amaranthaceae, such as spinach (*Spinacia oleracea*) and red root pigweed (*Amaranthus retroflexus*) (12,24,39).
**Geographic distribution.** Fusarium yellows is a common problem throughout the production areas of the western United States (33,39,43). It has also recently been found in the North Dakota and Minnesota growing regions (Red River Valley) (17), and can cause stalk blights of seed crops in Oregon (39). Before 2006, the known distribution of Fusarium root rot had been limited to sugar beet production fields in Texas (12,13,24). However, isolates causing root rot in sugar beets have recently been identified from Colorado and Montana (6).

**Pathogen isolation.** Both Fusarium pathogens are readily isolated from host tissue, plant debris, or soil (12,13,24,44). Isolation from internal root tissue is easily accomplished with surface disinestations using 10% ETOH and 1 to 5% NaOCl, followed by plating on numerous different media. Several variations of peptone PCNB agar are useful for soil isolations (27). Komada's medium (18) is also selective for *F. oxysporum* from soil or plant tissue, but these media do not differentiate between the pathogenic isolates and the ubiquitous saprophytes.

**Pathogen identification.** The two pathogens are similar morphologically. The Fusarium yellows pathogen produces sickle-shaped macroconidia measuring 3.5-5.5 × 21-35 µm, and straight to slightly curved microconidia (2.5-4.5 × 6-15 µm) (39). Globose chlamydospores can be either intercalary or terminal measuring 7 to 11 µm (39). The Fusarium root rot pathogen generally produces sparse numbers of macroconidia, but does produce microconidia (3-5 × 8-10 µm) in false heads and chlamydospores (4-7.5 × 20-30 µm) (24).

**Pathogen storage.** Long-term storage of *F. oxysporum* isolates is best accomplished by lyophilization of single-spored cultures in 50% glycerol or on colonized carnation leaf pieces in sterile skim milk (1,28,44). Cultures may also be stored for shorter periods of time on colonized dried filter paper cut into pieces and kept at 4°C (12). Cultures may also be stored as chlamydospores in sterile soil, but some isolates may mutate or lose viability over time (44).

**Pathogenicity tests.** Pathogenic isolates of *Fusarium oxysporum* are indistinguishable morphologically from saprophytic ones; therefore pathogenicity determinations are particularly important for these pathogens. Mass production of isolates may be accomplished with liquid culture, solid agar media, or various substrates such as grain chaff, seeds, or cornmeal and sand (1,28,44). Inoculations may then be made, but should correspond to the age of plants, or environmental conditions when natural infections are usually observed. Inoculum should also be applied into the natural infection court, either through soil or placed next to hosts (12,13,24,44). Infections generally appear when soil temperatures approach 25 to 28°C, thus these conditions would be optimal for pathogenicity testing.

Morphological differences between the two *Fusarium* pathogens are minimal, but they can be distinguished based on several different criteria. In addition to symptom expression in sugar beets (root rot vs. yellows symptoms) (12,13,24), Fusarium root rot isolates from Texas were also demonstrated to be distinct genetically from yellows isolates by analyses utilizing RAPD PCR, isozymes from at least 3 enzymes, and vegetative compatibility groupings (VCGs) (4,12,24). Unfortunately, these types of tests are often impractical to conduct, as they would require more sophisticated equipment and advanced training to effectively differentiate between pathogens. Therefore, pathogenicity testing, although difficult to standardize, is one of the more common methods for characterizing *F. oxysporum* isolates.

**Disease: Rhizomania**

**Primarily host.** Sugar beet (*Beta vulgaris* L.).

**Pathogen.** Beet necrotic yellow vein virus (BNYVV).

**Symptoms.** Foliar symptoms of rhizomania in the field consist of wilting (Fig. 32) and varying degrees of yellowing or chlorosis with an erect, upright posture (Fig. 32 inset) (11,14,15,33,36). The chlorosis may sometimes be confused with nitrogen deficiency, but no interveinal chlorosis or leaf scorching (typical of Aphanomyces root rot, and Fusarium yellows or Fusarium root rot) are observed (15,16,33,36). Systemic infection results in foliar symptoms consisting of yellow vein clearing (Fig. 33), which may later turn necrotic (16,33,36). This rarely-seen symptom is the source for the name of the pathogen.
(BNYVV). Root symptoms begin as a light brown discoloration of the central stele within the taproot. Classical root symptoms following early infection include small, stunted taproots with masses of secondary roots, giving the roots a "bearded" appearance (Fig. 34) (11,14,15,33,36). This is the origin of the name "rhizomania," meaning "crazy root." Later infections often cause roots to be constricted, resulting in a wine-glass shaped appearance (Fig. 35) (34,36).

The virus is transmitted by the soilborne protozoan Polymyxa betae, which survives in soil or root debris as thick-walled survival structures (Fig. 36) (cystosori), which also house the virus. Under conditions of high soil moisture, the cystosori liberate zoospores that carry the virus into plants as they infect roots (34,36). Without the vector, the viral disease is a non-issue.

**Host range.** The pathogen is restricted to plants in the Chenopodiaceae, Aizoaceae, and Amaranthaceae (34,36).
**Geographic distribution.** The pathogen is now widespread throughout all sugar beet growing regions in the world.

**Pathogen isolation.** As obligate pathogens, neither the virus nor the vector can be truly isolated or grown in pure culture. However, the virus can be transmitted mechanically to induce local lesions on hosts (36), proving the existence of the pathogen without serological or genetic diagnostic methods. Both the vector and virus can also be transmitted to new plants by coating untreated seeds with powdered, dried sugar beet roots infested with viruliferous *Polymyxa betae* (10).

**Pathogen identification.** The presence of the vector is readily confirmed by observing the thick-walled cystosori within roots (10,36) in wet mounts under the microscope (Fig. 36). The viral pathogen cannot be formally confirmed without the use of other more complex tests such as electron microscopy, ELISA, dot blots, or PCR assays.

**Pathogen storage.** Viral isolates can be stored as viruliferous cystosori within dried roots, as described previously.

**Pathogenicity tests.** Isolates can be tested either as mechanical inoculations for local lesion development or infestation of new seeds with viruliferous *P. betae*, as previously described above.

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**Literature Cited**


