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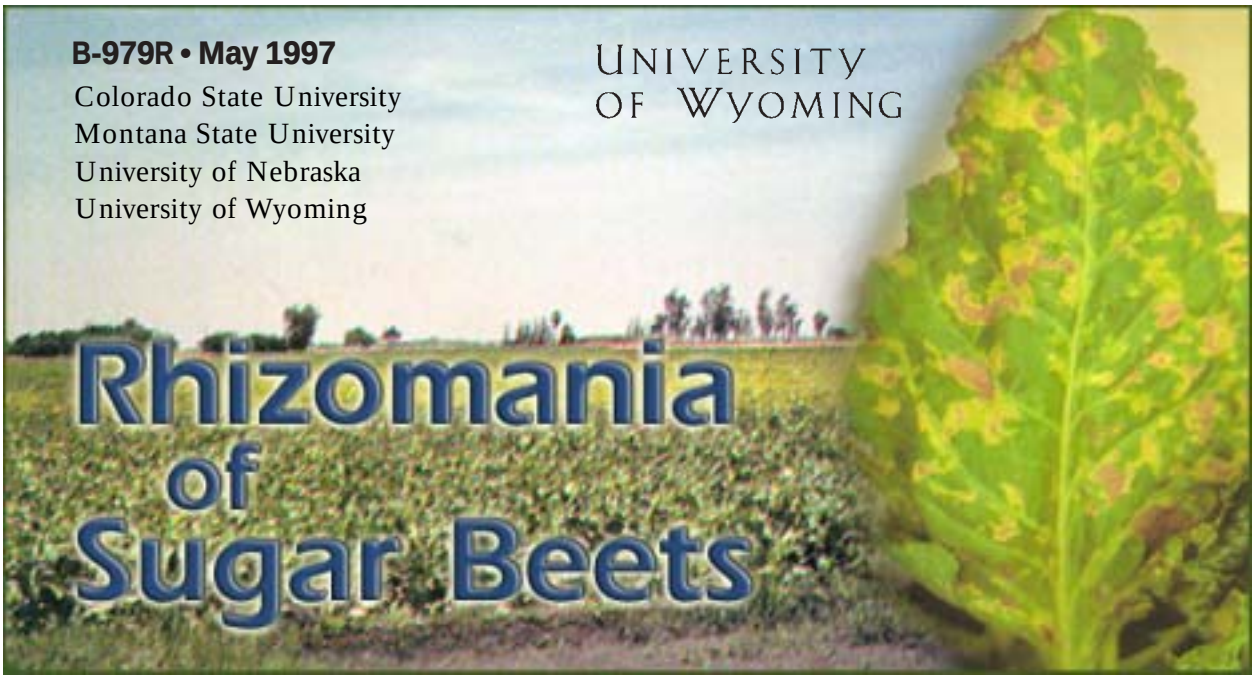

Rhizomania of Sugar Beets

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Quick Facts

- Rhizomania is a disease of sugar beets caused by Beet Necrotic Yellow Vein Virus (BNYVV).
- The soilborne fungus *Polymyxa betae* is the vector of BNYVV.
- BNYVV can persist in soil for years within resting spores formed by the fungus.
- Rhizomania development is favored by warm, wet soil.
- Most control methods involve containing the virus by preventing the movement of infested soil.
- Rhizomania is unrelated to the common root-rot disease of sugar beets caused by *Rhizoctonia*.
- Cultural practices that promote tuber skin set and harvesting methods that minimize tuber skinning and bruising greatly reduce tuber infection.

Introduction

Rhizomania, "crazy root," or "root madness" is one of the most serious diseases of sugar beets. Rhizomania can greatly reduce sugar yield by curbing either the tonnage or sugar content or both the tonnage and sugar content of harvested roots. Further losses to producers in infested areas can result when the movement of agricultural products is restricted by quarantine laws. Rhizomania is not related to the root rot of sugar beets caused by *Rhizoctonia*.

Rhizomania was first described in Italy in 1952. The causal agent was apparently spread by the movement of infested soil. Rhizomania was subsequently confirmed in Japan in 1969 and in the United States (California) in 1983. The causal agent was not known until 1973 when Japanese plant pathologists showed that rhizomania was caused by a virus and that a common soilborne fungus served as the vector or carrier of the virus.

Symptoms

Symptom expression varies greatly with some infected plants occasionally appearing healthy. Classical root symptoms following early infection include a mass of fine, hairy secondary roots, mostly dead, that give the

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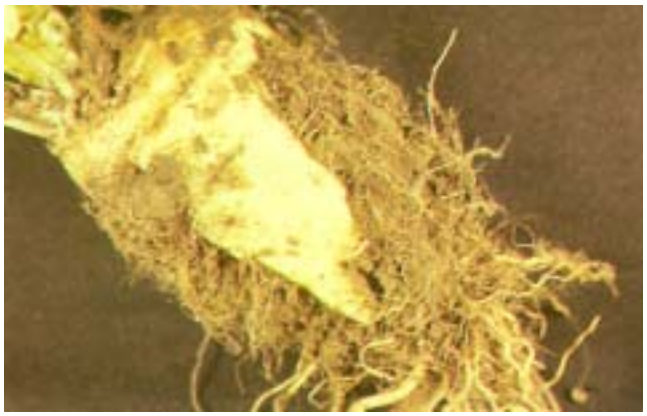

Figure 1. Lateral root proliferation resulting in the "bearded" or "hairy" root symptom associated with rhizomania. (Courtesy E. D. Kerr).

Figure 2. Crown enlargement and root constriction resulting in "wine-glass" shaped roots affected by rhizomania. Darkening and discoloration of vascular tissue is evident. (Courtesy R. T. Lewellen).

taproot a beard-like appearance (Figure 1). With slightly later infection, the storage root is often rotted and constricted, becoming much broader near the crown and thus resembling the shape of a wine glass (Figure 2). Infected roots occasionally rot. Very late infections may result in no obvious symptoms.

Leaves may become flabby and wilt without discoloration. Sometimes smaller leaves proliferate at the crown. A general chlorosis (yellowing) of foliage commonly occurs. Because infected roots are inefficient in water and nutrient uptake, general foliar symptoms are similar to water stress or nitrogen deficiency (Figure 3). Rarely, veinal yellowing with associated dead areas of leaf tissue can be seen (Figure 4). This symptom occurs when infection becomes systemic. At this point the virus can usually be recovered from the leaf tissue; otherwise, it normally cannot.

Diseased plants usually occur in patches or areas of a field as opposed to being scattered individually (Figure 3). Because the fungus vector thrives in moist areas, disease severity is usually greatest in depressions or compacted, poorly drained portions of a field that tend to collect water and remain wet. Reduced water uptake by infected roots increases the tendency for soil around diseased plants to remain waterlogged, thus promoting additional rhizomania development and root decay caused by other fungi.

Disease cycle

BNYVV is the causal agent of rhizomania. The soilborne fungus *Polymyxa betae* serves as a vector of BNYVV by carrying the virus to healthy roots. The association of BNYVV with the fungus is an unusual biological relationship that results in rhizomania development when a susceptible host is present and conditions are favorable for infection. Sugar beets serve as hosts to both the fungus and the virus. Although some weeds, primarily in the goosefoot family, also serve as host, their role in rhizomania development is not clear.

Data from field surveys in Wyoming and Nebraska show that *P. betae* is relatively common and usually causes little damage to sugar beets. Because BNYVV is spread by *P. betae*, conditions that favor the infection of sugar beets by the fungus also favor rhizomania development. Therefore, a greater understanding of rhizomania development and control can be achieved by knowing the life cycle of the virus vector. (Figure 5)

The fungus forms two types of spores during its life cycle – resting spores and motile zoospores. Clusters of tiny, thick-walled resting spores called cystorsori enable the fungus to survive in soil for 15 years or longer in the absence of a suitable host (figure 6). The virus can also persist in these resting spores for at least 15 years. When soil conditions become favorable for infection, the germination of the resting spore is triggered by the pres-

Figure 3. Symptoms of rhizomania generally occur in patches or areas of infested fields (background plants) surrounded by healthy-appearing plants (foreground plants). Above-ground symptoms may appear as a general chlorosis (yellowing) of foliage typical of nitrogen deficiency. (Courtesy J. Gerik).

Figure 4. Veinal yellowing and dead (necrotic) lesions associated with infection of sugar beet by beet necrotic yellow vein virus (BNYVV). Although this symptom is usually diagnostic of rhizomania, it is rarely observed in the field. (Courtesy R. T. Lewellen).

ence of a host-plant root. As resting spores germinate, motile zoospores are released that actively swim to the root surface where new infections occur.

The infection of roots by zoospores results in the formation of a fungus body or plasmodium inside the root. The plasmodium is able to quickly produce additional zoospores that are released and attracted to new roots. This rapidly repeating infection cycle requires approximately 48 hours for completion at 25 degrees Celsius (77 degrees Fahrenheit) and enables a rapid increase of the fungus in soil when soil conditions are favorable for infection. The plasmodium also forms resting spores that infest soil as root tissues degrade, enabling the fungus to persist in soil until conditions once again become favorable for the infection of a suitable host. Both spore types can be viruliferous or carriers of BNYVV.

Soil temperature and moisture greatly affect the development of rhizomania. Root infection is favored by relatively high soil temperatures with an optimum of 23 to 27 degrees Celsius (73 to 81 degrees Fahrenheit). Infection is sharply reduced by cooler temperatures with a minimum temperature of approximately 15 degrees Celsius (59 degrees Fahrenheit) required for the germination of resting spores and the infection of roots. Warm soil temperatures in the spring result in earlier infection and more severe damage from rhizomania. Zoospores require free moisture to move to roots and cause infection. Therefore, soil moisture at or near saturation for a prolonged period is necessary for infection and disease development. Short periods of rain in spring and early summer and the use of irrigation favors fungus activity and increased rhizomania severity provided the soil temperature is favorable. Soil pH in the range of six to eight plays a minor role in disease development. Zoospore activity appears to be more frequent in coarse-textured soils.

Disease Diagnosis

Because symptoms of expression vary greatly, the diagnosis of rhizomania cannot be based solely on visual inspection. Instead, routine diagnostics are done by a serological ELISA test or enzyme-linked immunosorbent assay. Plant tissue is needed for the test, which requires approximately 36 hours to complete. A positive ELISA test indicates that BNYVV was detected and rhizomania was present. A negative test merely indicates that BNYVV, if present, was not detected. Marginally (weak) positive ELISA tests may occur when viruses serologically related to BNYVV are present

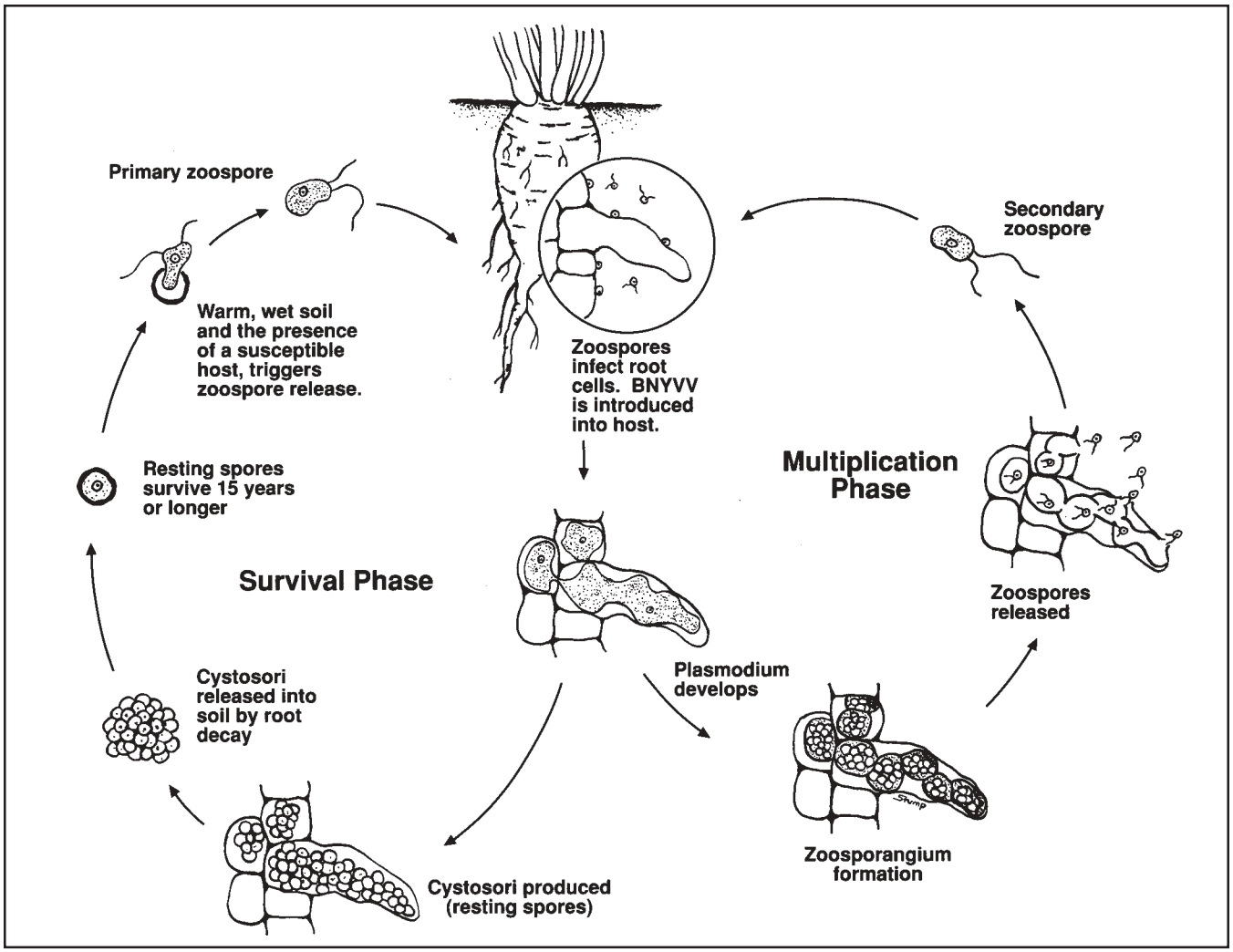

Figure 5. Life cycle of Polymyxa betae *in sugar beet. Rhizomania develops when zoospores carrying beet necrotic yellow vein virus (BNYVV) introduce the virus into root cells. Once introduced, both virus replication and fungal development occur, resulting in the production of BNYVV-contaminated secondary zoospores and resting spores. The virus can persist for 15 years or longer within contaminated resting spores. When* P. betae *is not contaminated with BNYVV, infection of sugar beet by the fungus usually results in little damage. (Prepared by W. L. Stump and G. D. Franc).*

in the tissue being tested. Also, false negative ELISA tests can commonly occur due to a number of factors including the methods of sample collection and the stage of disease development. Therefore, all test results must be carefully interpreted. To maximize the likelihood of an accurate test, sugar beet samples collected for testing should include new fibrous root growth occurring immediately after rainfall or irrigation, and samples should arrive at the laboratory within one day after collection. Currently, few laboratories in the United States test for the presence of BNYVV.

Procedures for the rapid detection of BNYVV directly from the soil have not been perfected. A bioassay for the detection of viruliferous *P. betae* in field soil samples has been developed for greenhouses. Susceptible sugar beets are planted in the soil sample being tested and allowed to grow under conditions ideal for rhizomania development. After approximately eight weeks, plants growing in the soil sample are harvested and tested for BNYVV by ELISA. A positive ELISA test from a properly conducted bioassay sample indicates that the pathogen was present in the soil sample. Soil samples collected for analysis should not represent

Figure 6. Clusters if thick-walled resting spores (cystosori) formed by ***Polymyxa betae*** *in sugar beet root cells. (Courtesy E. D. Kerr).*

more than 40 acres and should consist of 2 quarts of soil composited from at least 20 subsamples of soil-tube cores taken from the upper 6 inches of the soil profile. The laboratory performing the bioassay should be contacted to determine when soil samples should be collected. Quarantine laws may specifically state the maximum field size and other sampling restrictions that must be met before fields qualify as rhizomania free.

Control measures

Surveys to locate infested fields will aid in the control of rhizomania. Because rhizomania can readily spread through the movement of infested soil, fields not previously used for sugar beet production are also at risk and may be infested. The results of a properly conducted soil bioassay or the direct testing of suspicious beets by ELISA will permit growers to identify affected fields and allow them to make more effective management decisions related to cropping practices and containment.

The continuous planting or close rotation of sugar beets increases the risk of loss due to rhizomania. Early planting when soil temperatures are cooler and the use of production practices that result in the rapid establishment of the plant canopy will reduce the risk of loss. Early planting should be done at slightly greater plant densities to compensate for increased seedling losses in cooler soils.

Early season water management is especially critical for reducing loss from rhizomania. One should manage soil moisture to minimize or eliminate the need to irrigate during the first six weeks after seed germination. Because disease development is favored by high soil moisture, avoid over irrigation and any other practices that result in standing water or excessively wet soil. Proper fertility and irrigation practices for the variety grown must always be followed to reduce plant stress and further reduce the risk of disease development. If possible, runoff water from infested fields should be contained to prevent the movement of viruliferous spores to downstream sites. Efforts to reduce infection by lowering soil pH are not practical in most situations because of the large pH change required (a target pH of 5.6 or less).

Deep tillage to improve drainage will also help to reduce disease risk. However, avoid unnecessary tillage operations that spread infested soil within a field. Compaction associated with tillage operations also decreases drainage and increases disease risk. Minimize soil erosion in an infested field because it is highly probable that conditions that permit wind erosion will also spread and redistribute resting spores. Wind erosion can be reduced by maintaining surface residue from the previous crop, maintaining soil surface ridging and roughness, or by growing a cover crop.

Once a field becomes infected, crop rotation will not appreciably reduce disease risk because of the long-term survival of viruliferous cystosori. However, some soil fumigants such as Telone II may kill enough cystosori to reduce disease development to acceptable levels. Fumigation treatments are very expensive, and research is being done to determine their efficiency and the conditions under which they should be used. Expenses associated with fumigant application may be justified because significant sugar beet acreage is routinely treated with Telone II for nematode control. The use of soil-applied fungicides has not been effective for rhizomania control in infested fields.

Currently available tolerant or resistant varieties perform satisfactorily in the presence of rhizomania in some production areas, especially when used in combination with soil fumigation. However, these varieties must be tested in each production area to evaluate their performance un-

der local environmental conditions and production practices. They must also be evaluated for performance after exposure to local diseases, insects, and weed pests. Research on the development of resistant varieties is progressing rapidly with some having dual resistance to both rhizomania and curly top virus.

Because effective, practical control methods are not currently available, most efforts to control rhizomania spread are placed on containment or on limiting the movement of infested soil into uninfested fields and production areas. The sharing of farm equipment, migrant labor, and the movement of cattle or other livestock among farms are examples of how infested soil may be moved. The practice of returning bare dirt to fields greatly increases the risk of spreading BNYVV and its vector as well as other soilborne disease agents.

Infested fields should be isolated as much as is practical to prevent the movement of the tiny amounts of soil that are sufficient to spread the pathogen. Traffic in infested fields should be limited only to that which is absolutely necessary. Signs warning against entry may also reduce traffic and the subsequent spread of infested soil. Although it may be impossible to prevent that spread, rigid sanitation practices may delay its movement into uninfested fields.

If entry into an infested field is unavoidable, rubber boots or disposable footwear should be worn to permit the easy cleaning and removal of any soil adhering at the field site. Also, soil on tractors, machinery, and highway vehicles should be removed. This is necessary because resting spores are extremely difficult to kill with chemical disinfectants, especially when associated with soil. Therefore, infested soil removed from footwear and equipment is likely to remain so and will serve as a potential source of contamination.

Boots should be cleaned immediately to remove adhering soil after leaving the field, sanitized with a general disinfecting agent as a precaution to minimize the spread of pathogens, and placed in a disinfected, sealed container before re-entering a vehicle. If disposable footwear is used, it must be disposed of properly to prevent contamination of the vehicle. Soil is most effectively removed from equipment with soapy, hot water applied under pressure. Bleach is relatively ineffective against resting spores because the thick walls provide protection and because it is quickly inactivated by soil and organic matter. Bleach is also corrosive to metal and damaging to clothing. Commercial products for disinfecting machinery and footwear and for use in wheel dips have not been marketed in the United States for the control of *P. betae*.

Source of Information

Duffus, James E. 1986. “Rhizomania (Beet Necrotic Yellow Vein)” Pages 29-30, In: *Compendium of Beet Diseases and Insects*. APS Press, St. Paul, Minnesota. (Available from The American Phytopathological Society, 3340 Pilot Knob Road, St. Paul, Minnesota 55121-2097.)

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