

# Rhizomania on Sugar Beet

## Importance, identification, control

Oliver T. Neher and John J. Gallian

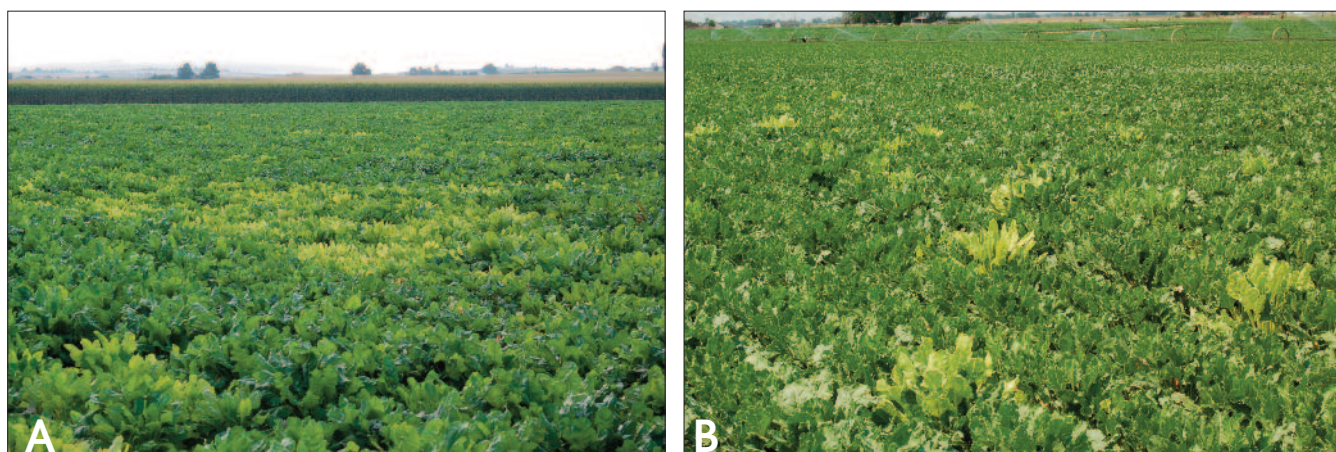


Figure 1. Symptomatic plants can occur in patches (A) or be distributed throughout a field (B). Photos by Oliver T. Neher

### Introduction

Rhizomania, caused by beet necrotic yellow vein virus (BNYVV), is considered the most serious disease of sugar beet worldwide. It can cause major losses in root yield and percentage sucrose as well as increase sucrose losses in storage. It was first reported in northern Italy in 1952 and has since spread to nearly all sugar beet growing areas including Japan, Europe, and the United States. Rhizomania was observed in California in 1984, followed by Texas during 1987. Between 1992 and 1994, rhizomania was detected in Colorado, Idaho, Nebraska, Wyoming, and the Red River Valley of Minnesota and North Dakota. Rhizomania was subsequently found in Washington and Oregon during 2002.

Rhizomania was first detected in Idaho near Rupert in 1992. The first year, 27 fields (670 acres) out of 354 fields sampled (14,740 acres) were diagnosed with rhizomania. Since then, rhizomania has spread throughout Idaho and many growers have experienced some economic loss.

With the introduction of resistant varieties containing the *Rz1* gene, the disease is now considered manageable.

### Symptoms

Symptomatic plants can occur in patches or be distributed throughout a field (figure 1). They are often found under conditions that favor high soil moisture, including areas with poor soil drainage and fields receiving frequent irrigations. Rhizomania, Greek for root madness or crazy root, gets its name from the excessive proliferation of

### Contents

Introduction.....	1
Symptoms.....	1
Causal organisms .....	3
Epidemiology and disease cycle .....	3
Control.....	4



**Figure 2.** Excessive proliferation of lateral rootlets is associated with rhizomania. Photo by Oliver T. Neher



**Figure 5.** Leaf symptoms consist of slight yellowing to a bright, fluorescent yellow leaf color; narrow erect growth habit; and leaf proliferation. Photo by Oliver T. Neher



**Figure 3.** Infection with beet necrotic yellow vein virus can result in a wineglass-shaped root. Photo by Oliver T. Neher



**Figure 4.** A range of foliar and root symptoms is associated with rhizomania. (Second beet from left is healthy.) Photo by Oliver T. Neher

lateral rootlets (figure 2) that sometimes appear on roots of plants affected by beet necrotic yellow vein virus. In addition to a bearded appearance caused by the prolific lateral rootlets, roots might be stunted and have a wineglass shape (figure 3).

Symptom expression and disease severity depend on the point in time (or developmental stage of the sugar beets) when the infection with BNYYV occurs. Infection earlier in the growing season will lead to stunted plants showing a wide range of foliar and root symptoms (figure 4). Plants infected later in the season might exhibit minimal foliar or root symptoms or none at all.

The timing of infection also plays an important role in harvest losses. Plants affected early in the season will show reduced root yield and a reduction in overall quality, mainly in percentage sucrose, while plants infected later in the season may suffer only from a reduction in root quality.

### Foliar symptoms

Leaf symptoms consist of slight yellowing to a bright, fluorescent yellow leaf color; erect growth habit; leaf proliferation (figure 5); and, under severe pressure, stunting. In mild infections, little or no stunting of the foliage is observed, but leaf color will be a slightly noticeable, uniform lime green. Leaves are usually uniformly chlorotic, and infected plants (called blinkers, figure 6) are easily recognized within a field. By late summer to early fall, symptoms of BNYYV can be confused with nitrogen deficiencies in the maturing crop: older leaves are often chlorotic, while younger leaves are green. Necrotic yellow veins (figure 7), the characteristic foliar symptom from which BNYYV derived its name, is rarely observed in Idaho.



**Figure 6.** Individual plants (blinkers) showing foliar symptoms of rhizomania. Photo by Oliver T. Neher



**Figure 7.** Necrotic yellow veins are the characteristic foliar symptom of beet necrotic yellow vein virus. Photo by Oliver T. Neher



**Figure 8.** Vascular discoloration is caused by infection with beet necrotic yellow vein virus. Photo by Oliver T. Neher

## Root symptoms

The most common symptom is the proliferation of lateral roots, giving the root a bearded appearance. Like foliar symptoms, however, root symptoms strongly depend on the timing of infection. Early infection can lead to a loss of the root's growing point, which will cause stunted, wineglass-shaped roots (figure 3) with excessive lateral rootlet production. In longitudinal sections of the root, the vascular tissue can show a light brown discoloration (figure 8) and will have a disorganized or marbled appearance in the vicinity of infected rootlets. This is in contrast to the white, parallel vascular bundles in normal, healthy tissue. With mild infections, roots may show only slight lateral rootlet proliferation on the taproot or lateral roots and the vascular discoloration may not be obvious.

## Diagnosis

It is important to remember that plants showing foliar symptoms may not have severe root symptoms, whereas symptomless plants might have severely infected roots. Therefore it is important to collect and observe multiple plants throughout the field. Serological tests (ELISA = enzyme-linked immunosorbent assay) specific for BNYVV can help to identify infected plants early in the season, but the test loses accuracy later in the season. When submitting plants for serological testing, it is important to have sufficient lateral rootlets associated with the plant samples.

## Causal organisms

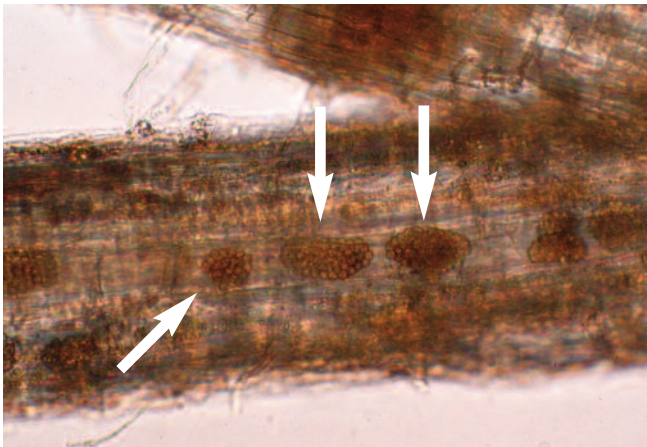
The symptoms of rhizomania are caused by an infection with beet necrotic yellow vein virus (BNYVV), which belongs to the genus *Benyvirus*. BNYVV is transmitted only by the fungal vector *Polymyxa betae*, a soilborne, fungus-like organism ubiquitous in soil.

BNYVV is known to occur as three strains (A, B, and P), with type A being predominant in the United States, Europe, Japan, and China. Types B and P are found in Germany and France, but also have been reported to occur in Sweden, China, Japan, and Iran.

## Epidemiology and disease cycle

BNYVV depends on the fungal vector *Polymyxa betae* for dispersal and infection. Both the virus and fungal vector are obligate parasites and require a specific, living host to reproduce. The natural host range of *P. betae* is limited and includes mainly plants in the Chenopodiaceae family, primarily species in the same genus as sugar beet (*Beta*), plus a few species in the genus *Chenopodium*. *Silene alba*, or white campion, which is in the Caryophyllaceae family, can also host *P. betae*.

The vector, *P. betae*, survives for more than 10 years in the soil as cysts or groups of cysts called cystosori (figure 9). Dormant cysts germinate in response to root exudates in



**Figure 9.** Cystosori are long-term survival structures of *Polymyxa betae*. Photo by Oliver T. Neher

the vicinity of sugar beet roots or roots of other suitable hosts when soil temperatures reach 59° to 82°F (15°–28°C) under near-saturated soil conditions. Germinated cysts release zoospores that, when in contact with host rootlets, can infect host cells. There *P. betae* multiplies and either releases new zoospores or develops into cystosori. Cystosori are released into the surrounding soil when infected roots die and decompose. With each irrigation, this life cycle is repeated and inoculum density increases.

*P. betae* is favored by high soil moisture, short rotations, and neutral to alkaline soils. Optimum soil temperature for *P. betae* is 77°F.

Only viruliferous zoospores of *P. betae* (zoospores carrying the virus), can lead to rhizomania. Nonviruliferous *P. betae* will still infect sugar beet but will cause very minimal damage.

BNYVV transmitted by *P. betae* zoospores will typically not move systemically throughout the host plant. Rather, the virus stays confined in a few cells surrounding the initial infection site and relies on the fungal vector for its dispersal to other rootlets and throughout the soil.

*P. betae* will move only a few inches per year through the soil without aid, but it is easily spread by infected plant material and with contaminated soil adhering to nonhost root crops or agricultural equipment. Irrigation water, and any other means that can move even small amounts of soil, can disseminate *P. betae*. It is not, however, seed transmitted.

High soil moisture and temperatures that favor infection with *P. betae* also favor infection with other soilborne pathogens such as *Aphanomyces cochlioides* and *Rhizoctonia solani*. Plants already stressed by infection with BNYVV, which causes impaired root function, are more susceptible to infection with secondary pathogens.

## Control

Because there are no varieties with resistance to *P. betae* available, nor are there pesticides registered for the control of *P. betae*, cultural practices are the only methods effective against this pathogen. The severity of rhizomania and the buildup of new viruliferous *P. betae* can be limited by using BNYVV-resistant varieties.

## Resistant varieties

Varieties resistant to BNYVV should be planted in any field with a history of rhizomania, no matter how small the affected area. Available varieties are not immune to rhizomania but have sufficient resistance to achieve good yields when managed properly.

Genetic resistance to rhizomania was initially identified by the Holly Sugar Company. The dominant gene was coined the Holly gene, or *Rz1*. It confers partial resistance against BNYVV. Plants expressing *Rz1* are still susceptible to infections by *P. betae* and BNYVV, but infected plants usually have very mild symptoms, have low virus titers, and perform well. However, under severe disease pressure, the resistance conferred by *Rz1* might be insufficient and plants can express symptoms of rhizomania. To overcome this limitation, other resistance genes were introduced into current varieties. Plants expressing a combination of two resistance genes, *Rz1* and *Rz2*, show lower levels of infection and improved performance.

## Glossary

**Benyvirus.** Genus of plant-infecting viruses with a positive single-stranded RNA genome. Viruses are rod shaped and do not possess an envelope.

**Centibars (cb).** Unit of atmospheric pressure equal to 1/100 bar that is used to measure soil water tension. 0 cb represents the state of full soil saturation, whereas soils reading –100 cb (and above) are dry.

**Cystosori.** Long-lived resting spore resistant to environmental factors.

**ELISA.** Enzyme-linked immunosorbent assay.

**Viruliferous.** Carrying a virus.

**Zoospores.** A fungal spore bearing a whip-like appendage and capable of moving in a film of water.

## Soil moisture and temperature

Sugar beets grow best when soil moisture is kept between –60 and –40 centibars (cb). It is not possible to irrigate and continually maintain these levels of soil moisture, but excessive irrigation should be avoided. The disease will be more severe in low spots, areas with poor soil structure and inadequate drainage, and other places where soil moisture is excessive. Soils wetter than –40 cb (–30 to 0 cb) favor infection by *P. betae*, and each irrigation cycle potentially will increase the inoculum.

It is recommended that growers fill the soil profile then wait as long as possible before irrigating again. The best timing for this irrigation practice is after planting and prior to seed germination, when soil temperatures are below 59°F.

## Early planting

The earlier plants become infected, the more severe the damage from rhizomania will be. Early planting at soil temperatures at or below 59°F allows plants to get well established before conditions are favorable for infection with *P. betae*.

## Stand

Good plant populations can help to reduce the severity of rhizomania. Closing the rows early with high plant populations tends to maintain cooler soil temperatures, which can reduce the rate of disease development.

## Cultivation practices

Promoting good soil drainage by avoiding soil compaction, breaking up compaction layers, or installing drainage tiles will help to reduce disease severity and inoculum formation when applied in combination with adequate irrigation.

Applying best management practices to keep the crop adequately fertilized and controlling weeds, insects, and other diseases will help to reduce stress, minimize losses, and promote a healthy crop.

## Crop rotation

Planting resistant varieties in combination with a minimum 4-year rotation will help to reduce the buildup of viruliferous *P. betae* populations. Short rotations will result in greater difficulty in managing rhizomania in subsequent years because inoculum can increase to a level that will overcome plant resistance.

## Further reading

Harveson, R. M., Hansen, L. E., and Hein, G. L., eds. 2009. Compendium of Beet Diseases and Pests. 2d ed. American Phytopathological Society Press, St. Paul, MN

## About the Authors:

**Oliver T. Neher**, former Extension Plant Pathologist, University of Idaho, and **John J. Gallian**, Professor Emeritus of Plant Pathology, University of Idaho

ALWAYS read and follow the instructions printed on the pesticide label. The pesticide recommendations in this UI publication do not substitute for instructions on the label. Pesticide laws and labels change frequently and may have changed since this publication was written. Some pesticides may have been withdrawn or had certain uses prohibited. Use pesticides with care. Do not use a pesticide unless the specific plant, animal, or other application site is specifically listed on the label. Store pesticides in their original containers and keep them out of the reach of children, pets, and livestock.

**Trade Names**—To simplify information, trade names have been used. No endorsement of named products is intended nor is criticism implied of similar products not mentioned.

**Groundwater**—To protect groundwater, when there is a choice of pesticides, the applicator should use the product least likely to leach.

---

Pacific Northwest extension publications are produced cooperatively by the three Pacific Northwest land-grant universities: Washington State University, Oregon State University, and the University of Idaho. Similar crops, climate, and topography create a natural geographic unit that crosses state lines. Since 1949, the PNW program has published more than 600 titles, preventing duplication of effort, broadening the availability of faculty specialists, and substantially reducing costs for the participating states.

Published and distributed in furtherance of the Acts of Congress of May 8 and June 30, 1914, by University of Idaho Extension, the Oregon State University Extension Service, Washington State University Extension, and the U.S. Department of Agriculture cooperating.

The three participating extension services offer educational programs, activities, and materials without regard to race, color, national origin, religion, sex, sexual orientation, age, disability, or status as a disabled veteran or Vietnam-era veteran, as required by state and federal laws. University of Idaho Extension, Oregon State University Extension Service, and Washington State University Extension are Equal Opportunity Employers.