IANR NebGuide
University of Nebraska Lincoln
Published by University of Nebraska–Lincoln Extension, Institute of Agriculture and Natural Resources
Know how. Know now.

G1842

Rhizomania of Sugar Beet

Robert M. Harveson, Extension Plant Pathologist

The virus disease rhizomania causes significant damage to sugar beets worldwide. Learn the symptoms, pathogen and disease cycle, and management techniques to help control it.

Introduction

Rhizomania, a virus disease caused by *beet necrotic yellow vein virus* (BNYVV), is one of the most economically limiting diseases of sugar beets wherever it occurs worldwide. It was first reported in Italy in 1959 and is now widespread throughout Europe. In the United States, it was first reported in sugar beets in California in 1984. It was first identified in Nebraska in 1992 and now has been identified in every major sugar beet production region in the country.

Symptoms

Foliar symptoms of rhizomania in the field consist of wilting *(Figure 1)* and varying degrees of yellowing or chlorosis with an erect, upright posture *(Figure 2).* Sometimes the chlorosis may be confused with nitrogen deficiency, but no interveinal chlorosis or leaf scorching (typical of Aphanomyces root rot and Fusarium yellows or root rot) is observed. Systemic infection results in foliar symptoms consisting of yellow vein banding *(Figure 3)* which may later turn necrotic *(Figure 4).* This rarely seen symptom is the source of the pathogen's name — beet necrotic yellow vein virus. Root symptoms begin as a light brown discoloration of the central stele within the taproot. Classical root symptoms following early infection include small, severely stunted taproots with masses of secondary roots, giving the roots a "bearded" appearance *(Figure 5).* This symptom is the origin of the name "rhizomania," meaning "crazy root." Infections occurring later in the season often cause roots to be constricted, resulting in a wineglass appearance *(Figure 6).*

Pathogen and Disease Cycle

Beet necrotic yellow vein virus is the type member of the viral genus *Benyvirus*. Its divided genome consists of four or five rigid rod particles (about 20 nm in diameter) with three to five molecules of single-stranded RNA. The different virus particles encode for different pathogen functions, including symptom expression, vector transmission, and movement within plants.

The virus is transmitted by the soil-borne plasmodiophorid-like fungus, *Polymyxa betae*, which is an obligate parasite infecting members of the *Chenopodiaceae*. The vector survives

Figure 1. Wilting symptoms in a field characteristic of rhizomania.

Figure 2. Yellowing and erect growth of plants affected by rhizomania in the field.

Figure 3. Yellow veinbanding symptoms characteristic of systemic rhizomania infection.

Figure 4. Vein banding symptoms becoming necrotic – source of virus name (beet necrotic yellow vein virus or BNYVV).

Figure 5. Bearding symptoms with very reduced taproot size, characteristic of early root infections. Source of the disease name "rhizomania" (root madness).

Figure 6. "Wineglass" symptoms characteristic of later root infections.

in soil or root debris *(Figure 7)* as thick-walled, long-lived structures called cystosori. The presence of the vector can be readily confirmed by observing the thick-walled cystosori within roots in wet mounts under the microscope *(Figure 8)*. Under conditions of high soil moisture and warm temperatures, the cystosori liberate viruliferous zoospores that inject the virus into plants as they infect roots. Without the vector, the viral disease does not occur. Beet necrotic yellow vein virus has a restricted host range which includes members of the families *Chenopodiaceae*, *Aizoaceae*, and *Amaranthaceae*. The virus can also be mechanically transmitted.

Management

- Using of resistant cultivars is the most effective and least expensive tool for managing this disease. Unfortunately, some recent evidence suggests that some populations of the pathogen may be overcoming the resistance in cultivars. This is evidenced by the appearance of so called "blinkers," which are plants of a resistant variety that show both foliar *(Figure 9)* and root *(Figure 10)* symptoms characteristic of the disease.

- A combination of cultural practices aimed at interfering with the vector's life cycle will also help minimize yield reduction. The cultural practices include planting early into cool soils, avoiding overuse of irrigation, and crop rotation.

- In general, due to the dependence of the pathogen upon its vector, any factor that prohibits infection by *P. betae* and its spread within or among fields will directly limit or reduce the incidence and severity of rhizomania.

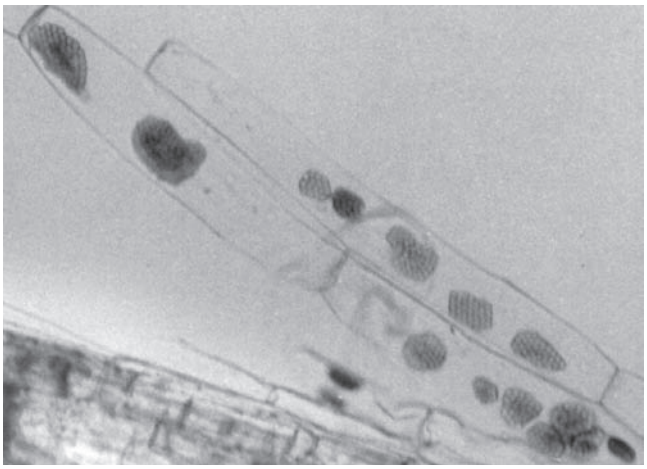

Figure 7. Cystosori of BNYVV vector, *Polymyxa betae*, in epidermal cells of infested small sugar beet feeder root.

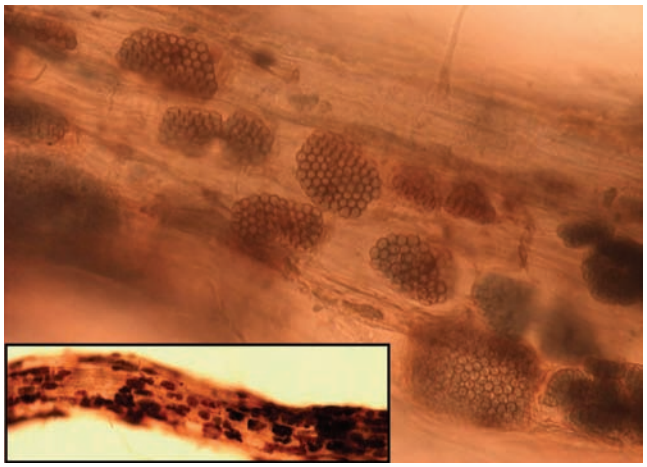

Figure 8. Close-up view of cystosori packed into infested feeder root.

Figure 9. Blinkers — foliar symptoms (bright, fluorescent yellow upright growth) of rhizomania in the field occurring in a tolerant cultivar.

Figure 10. Blinkers — root and foliar symptoms of rhizomania (left) on a tolerant cultivar compared to uninfected plant (right). Note the yellow foliage and mass of secondary rootlets on taproot of infected plant.

This publication has been peer-reviewed.

UNL Extension publications are available online at *http://extension.unl.edu/publications.*

Index: Plant Diseases
Sugar Beet
Issued April 2008