

EPPO Datasheet: *Benyvirus necrobetae*

Last updated: 2023-12-01

IDENTITY

Preferred name: *Benyvirus necrobetae*

Taxonomic position: Viruses and viroids: Riboviria: Orthornavirae: Kitrinoviricota: Alsuviricetes: Hepelivirales: Benyviridae

Other scientific names: *BNYVV*, *Beet necrotic yellow vein benyvirus*, *Beet necrotic yellow vein furovirus*, *Beet necrotic yellow vein virus*, *Beet rhizomania virus*

Common names: necrotic yellow vein of beet, rhizomania of beet

[view more common names online...](#)

EPPO Categorization: A2 list

[view more categorizations online...](#)

EU Categorization: PZ Quarantine pest (Annex III)

EPPO Code: BNYVV0



[more photos...](#)

Notes on taxonomy and nomenclature

Rhizomania was first recorded by Canova (1959) in Northern Italy on sugarbeet crops showing poor growth and abnormal root systems, but the cause of the disease was identified several years later in Japan when a virus called beet necrotic yellow vein virus (BNYVV) could be isolated from symptomatic sugarbeet plants (Tamada & Baba, 1973).

BNYVV was initially considered to be a typical member of the Benyvirus group. Later, benyviruses were included in the genus Furovirus, which included viruses with rigid bacilliform virions and a plasmid RNA genome carried by soil microorganisms of the order Plasmodiophoridales. Subsequently, this genus was subdivided into four genera based on genome structure (Torrance & Mayo, 1997): Furovirus, Pecluvirus, Pomovirus, and Benyvirus. BNYVV was included in the genus Benyvirus (Tamada, 2002).

The genus Benyvirus was originally included in the family Tubiviridae, and after redescription was assigned to the monotypic family Benyviridae (Gilmer & Ratti, 2017).

Members of the genus Benyvirus differ from members of other bacilliform virus genera, including the genera Tobamovirus, Tobravirus, and Hordeivirus, in polymerase phylogeny, genome organization, the presence of a polyadenylated 3' end, and genome expression strategy (Hehn *et al.*, 1997).

The genus Benyvirus currently includes the following species: Beet necrotic yellow vein virus, Beet soil-borne mosaic virus, Burdock mottle virus, and Rice stripe necrosis virus (Heidel *et al.*, 1997; Tamada, 1999; Morales *et al.*, 1999; Lee *et al.*, 2001).

Beet soil-borne mosaic benyvirus (BSBMV) is very similar to BNYVV in host range, particle morphology, genome organization and being vectored by *Polymyxa betae*, but these viruses differ serologically (Wisler *et al.*, 1994; Rush *et al.*, 1994; Rush & Heidel, 1995). A cross-protection reaction was observed between BNYVV and BSBMV in beet plants (Mahmood & Rush, 1999). The amino acid sequence of the envelope proteins of BNYVV and BSBMV has 56% identity similarity (Lee *et al.*, 2001), whereas the identity similarity between BNYVV and Burdock mottle virus, is only 38% (Hirano *et al.*, 1999).

BSBMV is widespread in the USA (Rush & Heidel, 1995), but has not yet been identified in other countries (Tamada, 2002). Both BSBMV and BNYVV can be found in the USA in the same area, and in the same plant with rhizomania symptoms (Rush & Heidel, 1995).

BNYVV is close in biological properties (identical host range and vector) to the pomoviruses - Beet soil-borne virus (BSBV) and Beet virus Q (BVQ), which are often found in mixed infection. However, these two viruses belong to different taxonomic families and differ significantly at the serological and genetic levels. In particular, members of

the genus Pomovirus of the family Virgaviridae differ from viruses of the genus Benyvirus in having only three genomic RNA molecules (Koenig *et al.*, 1996, 1997a).

HOSTS

All cultivated forms of *Beta vulgaris* are susceptible (sugarbeet, fodder beets, beetroots, mangolds, spinach beets) and also spinach (*Spinacia oleracea*).

In Turkey, BNYVV was detected by ELISA on the following weeds: *Heliotropium europaeum*, *Solanum nigrum*, *Plantago major*, *Cichorium intybus*, *Polygonum aviculare*, *Datura stramonium* (Yanar *et al.*, 2006).

The following plant species have also been reported as hosts of BNYVV: *Gomphrena globosa* (Al Musa & Mink, 1981), *Chenopodium album*, *Chenopodium capitatum* (Abe & Tamada, 1986), *Chenopodium polyspermum* (Hugo *et al.*, 1996), *Amaranthus retroflexus*, *Xanthium strumarium*, *Chenopodium album*, *Chenopodium vulvaria*, *Cirsium arvense*, *Chamomilla recutita*, *Sonchus asper*, *Sonchus arvensis*, *Polygonum aviculare*, *Polygonum persicaria*, *Portulaca oleracea*, *Veronica hederifolia*, *Datura stramonium*, *Solanum nigrum*, *Tribulus terrestris* (Kutluk Yilmaz *et al.*, 2000). The role of most of these plants in the biology of BNYVV remains unknown.

Experimentally, by sap inoculation, the virus can be transmitted to most plant species of the Chenopodiaceae family and to several plant species of the families Aizoaceae, Amaranthaceae, Caryophyllaceae and Solanaceae (Tamada & Baba, 1973; Horvath, 1994; Hugo *et al.*, 1996).

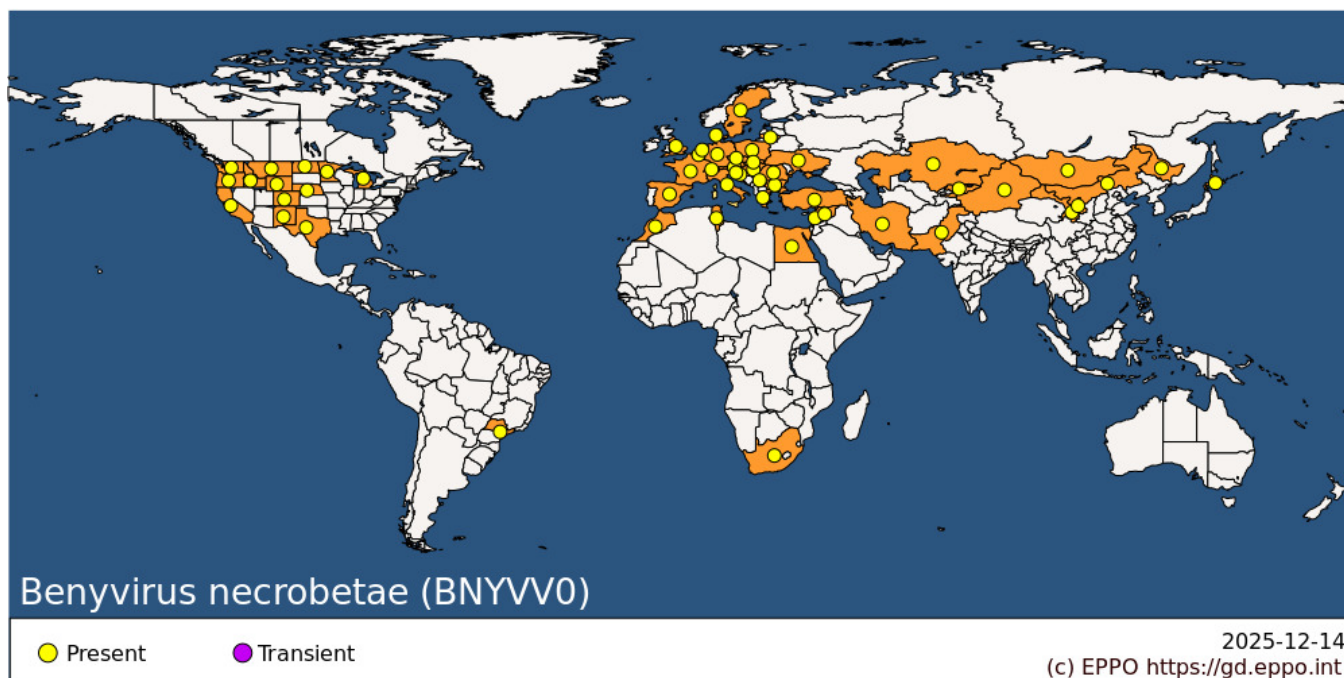
Host list: *Amaranthus retroflexus*, *Beta vulgaris*, *Blitum capitatum*, *Chenopodium album*, *Chenopodium vulvaria*, *Cichorium intybus*, *Cirsium arvense*, *Datura stramonium*, *Gomphrena globosa*, *Heliotropium europaeum*, *Lipandra polysperma*, *Matricaria chamomilla*, *Persicaria maculosa*, *Plantago major*, *Polygonum aviculare*, *Portulaca oleracea*, *Solanum nigrum*, *Sonchus arvensis*, *Sonchus asper*, *Spinacia oleracea*, *Tribulus terrestris*, *Veronica hederifolia*, *Xanthium strumarium*

GEOGRAPHICAL DISTRIBUTION

In the EPPO region, rhizomania damage was first observed in Italy during the 1950s, in the Po Plain and the Adige Valley (Canova, 1959). From 1971 to 1982 it was observed in an increasing number of Central and Southern European countries: Austria, France, Germany, Greece, Yugoslavia (Koch, 1982). It has also been found in Eastern Europe: Bulgaria, the former Czechoslovakia, Hungary, Poland, Romania. In 1983, it was discovered further north: Belgium, northern France, the Netherlands, Switzerland (Richard-Molard, 1985). In 1987 (Hill, 1989), a single focus was discovered in Eastern England (Henry *et al.*, 1986); several more foci have been found in the same area of the United Kingdom since. The virus is absent from Ireland, and also from the Nordic countries except Sweden, where BNYVV has been reported (Lindsten, 1989).

In the Russian Federation, BNYVV was detected by ELISA in several farms in Belgorod, Voronezh and Lipetsk regions (Ryazantsev *et al.*, 2012). However, the presence of this virus was not detected by subsequent official monitoring conducted by NPPO of Russia in 2011-2013 and by surveys conducted by All-Russian Plant Quarantine Center in 2014-2022 in the main beet-growing regions of the Russian Federation. According to the official data of the National report, BNYVV is absent in the territory of the Russian Federation in 2021 (National report on the quarantine phytosanitary status of the territory of the Russian Federation in 2021, 2022).

The disease is now considered to occur in most sugarbeet-growing countries in the EPPO region.



EPPO Region: Austria, Belgium, Bulgaria, Croatia, Czechia, Denmark, France (mainland), Germany, Greece (mainland), Hungary, Italy (mainland), Kazakhstan, Kyrgyzstan, Lithuania, Morocco, Netherlands, Poland, Romania, Serbia, Slovakia, Slovenia, Spain (mainland), Sweden, Switzerland, Tunisia, Türkiye, Ukraine, United Kingdom (England)

Africa: Egypt, Morocco, South Africa, Tunisia

Asia: China (Gansu, Heilongjiang, Neimenggu, Ningxia, Xinjiang), Iran, Islamic Republic of, Japan (Hokkaido), Kazakhstan, Kyrgyzstan, Lebanon, Mongolia, Pakistan, Syrian Arab Republic

North America: United States of America (California, Colorado, Idaho, Michigan, Minnesota, Montana, Nebraska, New Mexico, North Dakota, Oregon, Texas, Washington, Wyoming)

South America: Brazil (Sao Paulo)

BIOLOGY

The virus has rod-shaped particles, with a helical symmetry; their diameter is about 20 nm and their length is 390, 265, 105, 90 and 80 nm, these corresponding to five RNAs (RNA-1 to RNA-5), respectively (Putz *et al.*, 1988; Tamada *et al.*, 1989). The genome of the virus consists of single-stranded, positive-sense linear RNA molecules. Four RNA molecules are known to be part of all isolates: RNA-1 (6.7 kb), RNA-2 (4.7 kb), RNA-3 (1.8 kb), and RNA-4 (1.5 kb) (Bouzoubaa *et al.*, 1987). The genome of some isolates also contains RNA-5 (1.45 kb) which is part of the 80 nm virions. Isolates of the virus containing RNA-5 have been found in Japan, China, France, and Kazakhstan (Koenig *et al.*, 1997b; Miyanishi *et al.*, 1999; Koenig & Lennefors, 2000). In Japan, about half of the studied virus isolates contain RNA-5. BNYVV isolates containing RNA-5 have been found to be more virulent than isolates lacking RNA-5 (Tamada *et al.*, 1996).

RNA-1 contains a single open reading frame (ORF) potentially encoding a polypeptide with a molecular weight of 237 kDa that contains information required for viral genome replication. This ORF includes three replication-associated domains: methyltransferase, NTP-linked helicase, and polymerase (Hehn *et al.*, 1997).

RNA-2 contains six ORFs. One of them encodes a coat protein with a molecular weight of 21 kDa (Ziegler *et al.*, 1985). The RNA-2 site near the N-terminal site is responsible for the assembly of viral particles (Schmitt *et al.*, 1992).

The coat protein gene of BNYVV consists of 567 nucleotides. The sequence identity similarity for the nucleotides in the coat protein gene of different BNYVV isolates varies from 95.2 to 100% (Lennefors *et al.*, 2005).

The central region of RNA-2 contains a cluster of three genes (known as the triple gene block - TGB) encoding

proteins with molecular weights of 42, 13 and 15 kDa. Protein synthesis for the protein with a molecular weight of 42 kDa occurs directly on subgenomic RNA-2 suba, and protein synthesis for those with molecular weights of 13 and 15 kDa occurs on a dicistronic subgenomic matrix RNA - RNA-2 subb (Gilmer *et al.*, 1992). TGB proteins have amino acid sequences similar to those of potex-, carla-, horde-, pomo-, and pecluviruses, and are responsible for the intercellular movement of the virus.

The open reading frame (ORF) adjacent to the 3'-end of RNA-2 encodes a cysteine-rich protein with a molecular weight of 14 kDa, which is expressed on another subgenomic RNA (Gilmer *et al.*, 1992).

RNA-3 encodes a protein with a molecular weight of 25 kDa (P25) that is soluble in vivo (Niesbach-Klosgen *et al.*, 1990) and present in both the cytoplasm and nuclei of infected leaf cells. This protein stimulates virus multiplication in the roots and the systemic movement of the virus, and is also responsible for rhizomania symptoms (Koenig *et al.*, 1991; Lauber *et al.*, 1998; Tamada *et al.*, 1999; Vetter *et al.*, 2004). The P25 protein also acts as an avirulence factor in the leaves of some resistant sugarbeet lines, and this interaction is controlled by a single amino acid replacement (Chiba *et al.*, 2008). There is also a short ORF on RNA-3 with the N (necrotic) gene, which overlaps the C-terminus of the 25 kDa ORF. This gene induces the development of local necroses in infected plants (Jupin *et al.*, 1992). RNA-3 was also found to be responsible for systemic (vascular) movement of virus in plants (Tamada *et al.*, 1989).

RNA-4 encodes a protein with a molecular weight of 31 kDa (P31), which is soluble in vivo (Niesbach-Klosgen *et al.*, 1990) and is important for virus transfer by the fungus vector (Tamada & Abe, 1989), and is also interrelated with root-specific silencing of genes (Rahim *et al.*, 2007).

RNA-5 contains a single ORF encoding a protein with a molecular weight of 26 kDa that is correlated with disease symptom intensity in beet roots (Tamada *et al.*, 1999). A synergistic effect of RNA-5 on the protein P25 encoded by RNA-3 was found (Tamada *et al.*, 1996). The presence of RNA-5 is not required for virus survival (Tamada *et al.*, 1989).

Most virus isolates can be subdivided into two groups (pathotypes) according to differences in the nucleotide sequence in the envelope protein gene: A and B (Kruse *et al.*, 1994; Koenig *et al.*, 1995; Saito *et al.*, 1996; Miyanishi *et al.*, 1999; Schirmer *et al.*, 2005).

Group A isolates have been found in most European countries, the USA, Iran, China and Japan, i.e. practically in all sugarbeet growing regions (Kruse *et al.*, 1994; Ratti *et al.*, 2005; Sohi & Maleki, 2004; Schirmer *et al.*, 2005). Group B isolates are predominantly found in Northern European countries: Sweden (Lennfors *et al.*, 2000), Germany, North-West France (Kruse *et al.*, 1994; Koenig *et al.*, 2008), Belgium, the United Kingdom, Lithuania, the Netherlands (Ratti *et al.*, 2005) but has also been identified in Japan (Miyanishi *et al.*, 1999) and China (Li *et al.*, 2008).

BNYVV isolates containing RNA-5 have a limited distribution in France (Schirmer *et al.*, 2005), Germany (Koenig *et al.*, 2008) and the United Kingdom (Ward *et al.*, 2007), but are common in Asia (Koenig & Lennfors 2000; Li *et al.*, 2008; Miyanishi *et al.*, 1999). Most BNYVV isolates from Japan, China, and France containing RNA-5 belonged to group A (Miyanishi *et al.*, 1999).

Among isolates from France containing RNA-5, a new group of genetic variants called group P was identified (Koenig *et al.*, 1995, 1997b). Subsequently, isolates of this group were identified in Kazakhstan (Koenig & Lennfors, 2000).

The presence of nucleotides G194 and A448 and amino acid residues R17 and I102, respectively, in the envelope protein gene is characteristic of group P isolates (Miyanishi *et al.*, 1999; Koenig *et al.*, 2000). Group P isolates are very similar to group A isolates in genome features (Miyanishi *et al.*, 1999).

Isolates not containing RNA-5 but characteristic of group P by the nucleotide sequences in the P25 gene have been identified in the USA (Liu & Lewellen, 2007) and Iran (Mehrvar *et al.*, 2009).

Differences were found between these three groups of isolates in pathogenicity and in reproduction rate in plants of different beet cultivars (Heijbroek *et al.*, 1999). In particular, RNA-5-containing isolates were reported to be more pathogenic to sugarbeet plants than other BNYVV isolates (Tamada *et al.*, 1996; Miyanishi *et al.*, 1999). In addition, group P isolates show higher virus titre than isolates belonging to groups A and B, particularly when infecting

resistant cultivars (Tamada *et al.*, 1996; Heijbroek *et al.*, 1999).

Phylogenetic analysis of CP, P25, P26 and P31 genes for 75 BNYVV isolates of different geographical origin was performed in Japan. Phylogenetic analysis of individual genes and combined sequences revealed 8 clusters of isolates: i) Italian isolates; ii) isolates from Germany; iii) isolates from Japan-O; iv) isolates from China-B; v) isolates from Japan-D; vi) isolates from France-P; vii) isolates from China-H; viii) isolates from China-X (Chiba *et al.*, 2011).

The highest genome divergence was found for BNYVV isolates from Japan and China. Therefore, it has been suggested that the original BNYVV was most likely distributed in native hosts in East or Central Asia rather than in the Middle East or Europe. The original BNYVV isolate lines probably existed on native host plants in East Asia long before sugarbeet cultivation began. BNYVV has spread to sugarbeet from native host plants and soils only recently, most likely only in the last half century (Chiba *et al.*, 2011).

The vector of BNYVV is the plasmodiophorid species *Polymyxa betae* (Plasmodiophoraceae). Earlier this organism was attributed to fungi, but then was allocated to the Plasmodiophorales (or Plasmodiophorida) order (Braselton, 2001).

Polymyxa betae is an intracellular parasite restricted to the roots of Chenopodiaceae. It is present in most soils where beet has been grown (in all parts of Europe). Viral particles of BNYVV have been observed in the zoospores of *P. betae*. The spores (cystosori), which are the resistant stage, preserve the virus in the soil for many years. Dried infected roots and dry soil retained infectivity for more than 15 years. A similar duration of persistence of infectivity is also observed under field conditions (Tamada, 1999).

The main factors for infection and development of *P. betae*, its developmental cycle, and its subsequent reactivation are high soil temperature and humidity. Heavy rains in the spring period (April-May) after sowing at temperatures above 15°C cause earlier infestation. High temperatures (20-27°C) shorten the development cycle of *P. betae* and accelerate the spread of rhizomania in the fields (Asher, 1993). In contrast, it was found that the infection of beet roots by *P. betae* and the transmission of the virus is partly inhibited by low temperatures (Goffart & Maraite, 1992). Rhizomania develops more actively in alkaline or neutral soils (Abe, 1987; Asher, 1988). Soil pH within 6.0-8.0 has been found to be optimal for the development of *P. betae* (Ui, 1973; Abe, 1974).

P. betae was previously thought to have no direct pathogenic effect on the growth of sugarbeet plants. However, in Turkey the infection of soil with a non-viruliferous population of *P. betae* caused more than 3-fold weight loss in young beet plants regardless of their resistance to BNYVV (Kultuk Yilmaz, 2010). Evidence of pathogenicity of *P. betae* for sugarbeet plants was also obtained in the USA (Gerik & Duffus, 1988) and the United Kingdom (Brunt *et al.*, 1991).

DETECTION AND IDENTIFICATION

Symptoms

Beets grown in heavily infested fields show quite characteristic symptoms on developed roots: uncoordinated proliferation of partially necrosed small roots (known as 'salt and pepper beard') which gives its name to the disease (rhizomania - root madness). The root is often constricted (funnel-shaped) and cutting the root shows browning of the vascular ring, or even of the whole tip of the root.

In less heavily infested fields, symptoms may be less extensive and may affect only one lateral root, without constriction, and possibly without the 'beard'. Some of these symptoms can be due to other causes (nematodes, poor soil structure, etc.). The presence of tumour-like deformations, especially on the rootlets, is characteristic.

The most useful leaf symptom is visible at the end of the growing season, after rainfall; leaves become very pale-green, translucent and upright, and are distributed in patches throughout the field. The leaf yellowing followed by necrosis along the veins, seen in Japan and giving the virus its name (Tamada, 1975), is highly characteristic but infrequent.

However, BYNVV can also cause latent infections with no visible symptoms. This is especially the case under cool spring conditions (Lindsten, 1986).

Usually, the disease is present as patches in the field. At the beginning of summer, slowing down of growth can be observed after 2-3 months of crop growth; early wilting is also observed during dry periods.

Morphology

The virus is rod-shaped, with a helical symmetry; its diameter is about 20 nm; most isolates have a quadripartite genome, displayed as particles, the lengths of which are 390, 265, 100 and 85 nm, these correspond to 4 RNAs strands with 7100, 4800, 1800 and 1500 nucleotides, respectively (Bouzoubaa *et al.*, 1987). The genome of some isolates also contains 1.45 kb RNA-5, which is part of the 80 nm long virions (Tamada *et al.*, 1996).

Detection and inspection methods

In beet, the most efficient and easy detection method is an ELISA test, done on raw juice extracted from lateral roots or from the tip of the taproot (Putz, 1985). The sensitivity threshold is 2-6 ng of virus per g of tissue. Results obtained in this way are more reliable than those obtained by inoculation of indicator plants (*Chenopodium quinoa*). Quick test methods are now available (Schaufele *et al.*, 1995). It is necessary to take into account that BNYVV antiserum from different manufacturers may differ in its analytical sensitivity, analytical specificity and background values.

Molecular methods, with various modifications, are also widely used for the detection and identification of BNYVV, such as a one-step RT-PCR with primers BNYVV 016 (F)/ (BNYVV 017 (R) (Henry *et al.*, 1995; Morris *et al.*, 2001), real-time PCR with primers BNYVV-CP 26F/ BNYVV-CP 96R and probe BNYVV-CP 56T (Harju *et al.*, 2005), multiplex RT-PCR for simultaneous detection of BNYVV, Beet soil borne virus, Beet virus Q and their vector *P. betae* in the same test sample (Meunier *et al.*, 2003). Real-time RT-PCR with primers BNYVV-R5 96F/BNYVV-R5 203R and BNYVV-R5 123T probe (Harju *et al.*, 2005) was recommended as an additional test for detection of BNYVV isolates containing RNA-5. A number of other specific primers have also been developed by various authors to detect and identify BNYVV and to study the genetic features of the virus isolates.

The EPPO diagnostic protocol PM 7/30 (3) provides recommendations on how to detect and identify BNYVV (EPPO, 2022).

For soil or for adherent soil, a biological test is required. Beet plants are grown in suspect soil, and an ELISA test is performed on their roots. For very small soil samples, miniaturized tests have been devised (Merz & Hani, 1985). Bait plant tests to estimate levels of BNYVV in soil using pre-grown sugarbeet seedlings (Goffart *et al.*, 1989) as well as to calculate potential yield losses. However, these tests are not reliable enough to detect very low levels of BNYVV in soil and are, therefore, unsuitable for establishing whether fields are free from the virus (Büttner & Bürcky, 1990).

PATHWAYS FOR MOVEMENT

BNYVV is thought to spread mainly through the movement of soil containing dormant spores of viruliferous *P. betae* populations (e.g. global or local human-mediated movement of soil with various plant material, natural spread of vector spores by wind or water, or accidental spread of vector spores by various animals and migratory birds) (Asher, 1993; Tamada, 1999).

Spores of *P. betae* harbouring the virus can easily spread with irrigation and flood water, plant residues, soil lumps on farm equipment and vehicles, as well as on the roots of beets, potatoes, roots of vegetable root crops grown in infested plots (Richard-Molard, 1985; Heijbroek, 1988).

Manure may also be important in the disease spread since *P. betae* spores do not lose infectivity after passage through the gastrointestinal tract of animals.

Waste products from sugar production from sugarbeet, including pulp and water used to wash root crops, can also contribute to the spread of the disease (Heijbroek, 1988).

BNYVV is not thought to be transmitted by seeds and pollen. However, spread of this virus is possible with soil lumps and soil dust that may contaminate seeds during harvest and contain vector cystosori (Tamada, 2002). In Ukraine, it was found that the weight of soil particles per 1 kg of sugarbeet seeds imported from Western Europe is on average 5-6 g, and the number of sugarbeet seedlings infected with *P. betae* from this volume of soil can reach 1.6% (Nurmukhamedov & Vasilieva, 2006).

PEST SIGNIFICANCE

Economic impact

Rhizomania causes severe damage wherever it is present; losses can amount to 50-70% of root weight and two to more than four percentage points of sugar content (Merdinoglu *et al.* 1993; Prillwitz & Schlösser, 1993; Asher, 1999; Tamada *et al.*, 1999; McGrann *et al.*, 2009). Since BNYVV survives in the soil for many years without any decrease in intensity, its presence makes it necessary to avoid growing sugarbeet in heavily infested soils.

Control

Chemical control methods against the vector are either too expensive or ineffective. The search for tolerant or resistant cultivars has been actively carried out since 1978. In 1983, the first resistance gene (Rz1) to BNYVV was identified (Lewellen *et al.*, 1987). It is a dominant gene that does not prevent infection of beet plants with BNYVV and *P. betae*, but significantly suppresses virus multiplication and prevents development of rhizomania symptoms (Lewellen *et al.*, 1987; Scholten *et al.*, 1996). In plants with the Rz1 gene, BNYVV reproduction is inhibited in the lateral roots (Scholten *et al.*, 1996) and the movement of the virus into uninfected roots is disturbed (Heijbroek *et al.*, 1999). Due to the qualitative nature of the resistance induced, introgression of the Rz1 gene has been widely used in backcrossing programs to develop most modern commercial sugarbeet varieties (Asher, 1993; Lewellen *et al.*, 1987; Biancardi *et al.*, 2002; Rush, 2003). However, severe rhizomania symptoms were subsequently found in varieties containing the Rz1 gene in some areas of the USA and Europe, indicating the emergence of BNYVV strains that can overcome varietal resistance (resistance breaking or RB strains) (Liu *et al.* 2005; Liu & Lewellen, 2007). It has been suggested that the use of the Rz1 gene as the sole source of resistance for many years has caused strong selection pressure on the viral population, leading to the development of virus variants that can break this resistance (Liu *et al.*, 2005; Chiba *et al.*, 2011; Bornemann *et al.*, 2014; Kultuk Yilmaz *et al.*, 2018). This stimulated the search for additional sources of resistance to rhizomania and contributed to the discovery of resistance genes Rz2, Rz3, Rz4 and Rz5 in different forms of *Beta vulgaris* subsp. *maritima* (Scholten *et al.*, 1996; Amiri *et al.*, 2003; Gidner *et al.*, 2005; Grimmer *et al.*, 2007, 2008). In the long term, BNYVV control will depend on the resistance gene Rz2, which has also been introduced into sugarbeet cultivars (Scholten *et al.*, 1999).

It is recommended to avoid cultivation of sugarbeet in areas with high levels of rhizomania and to follow optimal crop rotations.

Phytosanitary risk

Within the EPPO region, sugarbeet is grown extensively and represents a major cash crop for agricultural producers. Considerable areas are still free from the virus, especially in Northern Europe. However, biological and epidemiological studies seem to indicate that the climatic zone where the organism can induce considerable yield losses is defined by the temperature requirements of the pathogen. Due to climate change, the area where the pathogen can cause severe damage may increase significantly.

PHYTOSANITARY MEASURES

Measures aim to prevent spread into new countries and to limit spread within countries where the virus is present.

Areas in which beet seed and beet stecklings are produced should be kept under regular phytosanitary observation. Any imported seed or stecklings should come from a field (or preferably area) where BNYVV does not occur. Beet seed from infested areas should be kept free from impurities (soil) and should contain no more than 0.5% inert matter (other than pelleting material) in the case of certified seed and 1% in the case of basic seed.

Countries where BNYVV does not occur would be well advised to recommend importers of root vegetables from countries where the virus is present to take special precautions concerning the disposal of waste vegetable matter, soil waste and liquid waste (CABI/EPPO, 1997).

REFERENCES

Abe H (1987) Studies of the ecology and control of *Polymyxa betae* Keskin as a fungal vector of the causal virus (Beet necrotic yellow vein virus) of rhizomania disease of sugar beet. *Report of Hokkaido Prefecture Agricultural Experiment Station* **60**, 81-85.

Abe H & Tamada T (1986) Association of Beet necrotic yellow vein virus with isolates of *Polymyxa betae* Keskin. *Annals of the Phytopathological Society of Japan* **52**, 235-247.

Abe N (1974) Factors affecting the rhizomania of sugar beet. *Bulletin of the Hokkaido Prefecture Agricultural Experiment Station* **30**, 95-102.

Al Musa AM & Mink GI (1981) Beet necrotic yellow vein virus in North America. *Phytopathology* **71**, 773-776.

Amiri R, Moghaddam M, Mesbah M, Yaghoub Sadeghian S, Ghannadha MR & Izadpanah K (2003) The inheritance of resistance to Beet necrotic yellow vein virus (BNYVV) in *B. vulgaris* subsp. *maritima*, accession WB42: Statistical comparisons with Holly-1-4. *Euphytica* **132**, 363-373.

Asher MJC (1988) Approaches to the control of fungal vectors of viruses with special reference to rhizomania. *Conference* **2**, 615-627.

Asher MJC (1993) Rhizomania. In: Cooke DA & Scott RK (eds) *The sugar beet crop, Science into practice*. Chapman & Hall, London, 311-346.

Asher MJC (1999) Sugar-beet rhizomania: the spread of a soilborne disease. *Microbiology Today* **26**, 120-122.

Biancardi E, Lewellen RT, De Biaggi M, Erichsen AW & Stevanato P (2002) The origin of rhizomania resistant in sugar beet. *Euphytica* **127**, 383-397.

Bornemann K, Hanse B, Varrelmann M & Stevens M (2014) Occurrence of resistance-breaking strains of Beet necrotic yellow vein virus in sugar beet in northwestern Europe and identification of a new variant of the viral pathogenicity factor P25. *Plant Pathology* **64**, 25-34.

Bouzoubaa S, Quillet L, Guilley H, Jonard G & Richards K (1987) Nucleotide sequence of beet necrotic yellow vein virus RNA-1. *Journal of General Virology* **68**, 615-626.

Braselton JP (2001) Plasmodiophoromycota. In: McLaughlin, D.J., McLaughlin, E.G., Lemke, P.A. (eds) *Systematics and Evolution. The Mycota*, vol 7A. Springer, Berlin, Heidelberg. https://doi.org/10.1007/978-3-662-10376-0_4

Brunt SJ, Adams MJC & Gilligan CA (1991) Infection of sugar beet by *Polymyxa betae* in relation to soil temperature. *Plant Pathology* **40**, 257-267.

Büttner G & Bürcky K (1990) Experiments and considerations on the detection of BNYVV in soil by means of bait plants. *Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz* **97**, 56-64.

CABI/EPPO (1997) Beet necrotic yellow vein virus. In Smith IM, McNamara DG, Scott PR, Holderness M (eds) *Quarantine Pests for Europe*. CABI, Wallingford (GB).

- Canova A (1959) [On the pathology of sugar beet]. *Informatore Fitopatologico* **9**, 390-396 (in Italian).
- Chiba S, Kondo H, Miyanishi M, Andika IB, Han C & Tamada T (2011) The evolutionary history of Beet necrotic yellow vein virus deduced from genetic variation, geographical origin and spread, and the breaking of host resistance. *MPMI* **24**, P.207-218.
- Chiba S, Miyanishi M, Andika IB, Kondo H & Tamada T (2008) Identification of amino acids of the Beet necrotic yellow vein virus p25 protein required for induction of the resistance response in leaves of *Beta vulgaris* plants. *Journal of General Virology* **89**, 1314-1323.
- EPPO (2022) Beet necrotic yellow vein virus. Diagnostic protocol PM 7/30 (3). *EPPO Bulletin* **52**, 87-97.
- Gerik J & Duffus J (1988) Differences in vectoring ability and aggressiveness of isolates of *Polymyxa betae*. *Phytopathology* **78**, 1340-1343.
- Gidner S, Lennefors BL, Nilsson NO, Bensefelt J, Johansson E, Gyllenspetz U & Kraft T (2005) QTL mapping of BNYPV resistance from the WB41 source in sugar beet. *Genome* **48**, 279-285.
- Gilmer D, Bouzoubaa S, Hehn A, Guilley H, Richards K & Jonard G (1992) Efficient cell-to-cell movement of Beet necrotic yellow vein virus requires 3' proximal genes located on RNA2. *Virology* **189**, 40-47.
- Gilmer D & Ratti C (2017) ICTV Virus Taxonomy Profile: Benyviridae. *Journal of General Virology* **98**, 1571-1572.
- Goffart JP & Maraite H (1992) Influence of temperature on *Polymyxa betae* and beet necrotic yellow vein virus (BNYPV). *Mededelingen van de Faculteit Landbouwwetenschappen, Rijksuniversiteit Gent* **57**.
- Goffart JP, Horta V & Maraite H (1989) Inoculum potential and host range of *Polymyxa betae* and beet necrotic yellow vein furovirus. *EPPO Bulletin* **19**, 517-525.
- Grimmer MK, Kraft T, Francis SA & Asher MJC (2008) QTL mapping of BNYPV resistance from the WB258 source in sugar beet. *Plant Breeding* **127**, 650-652.
- Grimmer MK, Trybush S, Hanley S, Francis SA, Karp A & Asher MJC (2007) An anchored linkage map for sugar beet based on AFLP, SNP and RAPD markers and QTL mapping of a new source of resistance to Beet necrotic yellow vein virus. *Theoretical and Applied Genetics* **114**, 1151-1160.
- Harju VA, Skelton A, Clover GRG, Ratti C, Boonham N, Henry CM & Mumford RA (2005) The use of real-time RT-PCR (TaqMan®) and post-ELISA virus release for the detection of Beet necrotic yellow vein virus types containing RNA 5 and its comparison with conventional RT-PCR. *Journal of Virological Methods* **123**, 73-80.
- Hehn A, Fritsch C, Richards KE, Guilley H & Jonard G (1997) Evidence for in vitro and in vivo autocatalytic processing of the primary translation product of Beet necrotic yellow vein virus RNA1 by a papain-like proteinase. *Archives of Virology* **142**, 1051-1058.
- Heidel GB, Rush CM, Kendall TL, Lommel SA & French RC (1997) Characteristics of Beet soil-borne mosaic virus, a furovirus infecting sugar beet. *Plant Disease* **81**, 1070-1076.
- Heijbroek W (1988) Dissemination of rhizomania by soil, beet seeds and stable manure. *Netherlands Journal of Plant Pathology* **94**, 9-15.
- Heijbroek W, Musters PMS & Schoone AHL (1999) Variation in pathogenicity and multiplication of Beet necrotic yellow vein virus (BNYPV) in relation to the resistance of sugar beet cultivars. *European Journal of Plant Pathology* **105**, 397-405.
- Henry CM, Barker I, Morris J & Hugo SA (1995) Detection of Beet necrotic yellow vein virus using reverse transcription and polymerase chain reaction. *Journal of Virological Methods* **54**, 15-28.

- Henry CM, Jones RAC & Coutts RHA (1986) Occurrence of a soil-borne virus of sugar beet in England. *Plant Pathology* **35**, 585-591.
- Hill SA (1989) Sugar beet rhizomania in England. *EPPO Bulletin* **19**, 501-508.
- Hirano S, Kondo H, Maeda T & Tamada T (1999) Burdock mottle virus has a high genome similarity to Beet necrotic yellow vein virus. In Proceedings of the 4th Symposium of the International Working Group on Plant Viruses with Fungal Vectors (Monterey, US, 1999-10-05/08), pp. 33-36.
- Horváth J (1994) Beet necrotic yellow vein furovirus new host. *Acta Phytopathologica et Entomologica Hungarica* **29**, 109-118.
- Hugo S, Henry C & Harju V (1996) The role of alternative hosts of *Polymyxa betae* in transmission of Beet necrotic yellow vein virus (BNYVV) in England. *Plant Pathology* **45**, 662-666.
- Jupin I, Guilley H, Richards KE & Jonard G (1992) Two proteins encoded by Beet necrotic yellow vein virus RNA3 influence symptom phenotype on leaves. *EMBO Journal* **11**, 479-488.
- Koch F (1982) [Rhizomania of sugar beet]. In: *Compte Rendu du 45e Congrès d'Hiver de l'Institut International de Recherches Betteravières*, pp. 211-238. Institut International de Recherches Betteravières, Brussels, Belgium.
- Koenig R, Beier C, Commandeur U, Lüth U, Kaufmann A & Lüddecke P (1996) Beet soil-borne virus RNA 3 – A further example of the heterogeneity of the gene content of furovirus genomes and of triple gene block-carrying RNAs. *Virology* **216**, 202-207.
- Koenig R, Commandeur U, Loss S, Beier C, Kaufmann A & Lesemann DE (1997a) Beet soil-borne virus RNA2: similarities and dissimilarities to the coat protein gene carrying RNAs of other furoviruses. *Journal of General Virology* **78**, 469-477.
- Koenig R, Haeberlé AM & Commandeur U (1997b) Detection and characterization of a distinct type of Beet necrotic yellow vein virus RNA5 in a sugar beet growing area in Europe. *Archives of Virology* **142**, 1499-1504.
- Koenig R, Jarausch W, Li Y, Commandeur U, Burgermeister W, Gehrke M & Lüddecke P (1991) Effect of recombinant Beet necrotic yellow vein virus with different RNA compositions on mechanically inoculated sugar-beets. *Journal of General Virology* **72**, 2243-2246.
- Koenig R, Kastirr U, Holtschulte B, Demi G & Varrelmann M (2008) Distribution of various types and P25 subtypes of Beet necrotic yellow vein virus in Germany and other European countries. *Archives of Virology* **153**, 2139-2144.
- Koenig R & Lennefors BL (2000) Molecular analyses of European A, B and P type sources of Beet necrotic yellow vein virus and detection of the rare P type in Kazakhstan. *Archives of Virology* **145**, 1561-1570.
- Koenig R, Lüddecke P & Haeberle AM (1995) Detection of Beet necrotic yellow vein virus strain, varieties and mixed infections by examining single-strand confirmation polymorphism of immunocapture RT-PCR products. *Journal of General Virology* **76**, 2051-2055.
- Koenig R, Pleij CWA & Büttner G (2000) Structure and variability of the 3' end of RNA 3 of Beet soil-borne pomovirus – a virus with uncertain pathogenic effects. *Archives of Virology* **145**, 1173-1181.
- Kruse M, Koenig R, Hoffmann A, Kaufmann A, Commandeur U, Solovyev AG, Savenkov I & Burgermeister W (1994) Restriction fragment length polymorphism analysis of reverse transcription-PCR products reveals the existence of two major strain groups of beet necrotic yellow vein virus. *Journal of General Virology* **75**, 1835-1842.
- Kutluk Yilmaz ND, Erkan N & Bicken S (2000) Weeds as hosts for rhizomania's agent. *Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz* **27**, 167-171.
- Kutluk Yilmaz ND & Sokmen MA (2010) Occurrence of soilborne sugar beet viruses transmitted by *Polymyxa betae*

northern and central Turkey. *Journal of Plant Pathology* **92**, 507–510.

Kutluk Yilmaz ND, Uzunbacak H, Arli-Sokmen M & Kaya R (2018) Distribution of resistance-breaking isolates of Beet necrotic yellow vein virus differing in virulence in sugar beet fields in Turkey. *Acta Agriculturae Scandinavica, Section B — Soil & Plant Science* **68**, 545-554.

Lauber E, Guilley H, Tamada T, Richards KE & Jonard G (1998) Vascular movement of beet necrotic yellow vein virus in *Beta macrocarpa* is probably dependent on an RNA 3 sequence domain rather than a gene product. *Journal of General Virology* **79**(2),385-393.

Lee L, Telford EB, Batten JS, Scholtkof KBG & Rush CM (2001) Complete nucleotide sequence and genome organization of Beet soilborne mosaic virus, a proposed member of the genus Benyvirus. *Archives of Virology* **146**, 2443-2453.

Lennefors BL, Lindsten R & Koenig R (2000) First report of A and B type Beet necrotic yellow vein virus in sugar beet in Sweden. *Journal of Plant Pathology* **106**, 199-201.

Lennefors BL, Savenkov EI, Mukasa SB & Valkonen JPT (2005) Sequence divergence of four soilborne sugarbeet-infecting viruses. *Virus Genes* **31**, 57-64.

Lewellen RT, Skoyen IO & Erichsen AW (1987) Breeding sugarbeet for resistance to rhizomania: Evaluation of host-plant reactions and selection for and inheritance of resistance. *Compte Rendu du 50e Congrès d'Hiver de l'Institut International de Recherches Betteravières (IIBR), Brussels*, 139-156.

Li M, Liu T, Wang B, Han C, Li D & Yu J (2008) Phylogenetic analysis of Beet necrotic yellow vein virus isolates from China. *Virus Genes* **36**, 429-432.

Lindsten K (1986) [Rhizomania - a complicated disease in sugar beets which can also occur in Sweden]. *Växtskyddsnotiser* **50**, 111-118.

Lindsten K (1989) Investigations concerning soil-borne viruses in sugarbeet in Sweden. *EPPO Bulletin* **19**, 531-537.

Liu HY & Lewellen RT (2007) Distribution and molecular characterization of resistance-breaking isolates of Beet necrotic yellow vein virus in the United States. *Plant Disease* **91**, 847–851.

Liu HY, Sears JL & Lewellen RT (2005) Occurrence of resistance-breaking Beet necrotic yellow vein virus of sugar beet. *Plant Disease* **89**, 464-468.

Mahmood T & Rush CM (1999) Evidence of cross-protection between Beet soilborne mosaic virus and Beet necrotic yellow vein virus in sugar beet. *Plant Disease* **83**, 521-526.

McGrann GRD, Grimmer MK, Mutasa-Gottgens ES & Stevens M (2009) Progress towards the understanding and control of sugar beet rhizomania disease. *Molecular Plant Pathology* **10**, 129-141.

Mehrvar M, Valizadeh J, Koenig R & Bragard CG (2009) Iranian Beet necrotic yellow vein virus (BNYVV): pronounced diversity of the p25 coding region in A-type BNYVV and identification of P-type BNYVV lacking a fifth RNA species. *Archives of Virology* **154**, 501-506.

Merdinoglu D, Valentin P, Geyl L, Merdinoglu- Wiedemann S, Lemaire O & Putz C (1993) Amélioration génétique de la betterave pour la résistance à la rhizomanie. *Comptes Rendus de l'Académie d'Agriculture Française* **79**, 85-98.

Merz V & Hani A (1985) [A bait plant test to determine infection potential of BNYVV and *Polymyxa betae* in soil samples]. In: *Compte Rendu du 48e Congrès d'Hiver de l'Institut International de Recherches Betteravières*, pp. 421-430. Institut International de Recherches Betteravières, Brussels, Belgium.

Meunier A, Schmit JF, Stas A, Kutluk N & Bragard C (2003) Multiplex RT-PCR for the simultaneous detection of Beet necrotic yellow vein virus, Beet soil borne virus, Beet virus Q and their vector *Polymyxa betae* Keskin on sugar beet. *Applied and Environmental Microbiology* **69**, 2356-2360.

- Miyanishi M, Kusume T & Tamada T (1999) Evidence for three groups of sequence variants of Beet necrotic yellow vein virus RNA-5. *Archives of Virology* **144**, 879-892.
- Morales F, Ward E, Castano M, Arroyave J, Lozano I & Adams M (1999) Emergence and partial characterization of Rice stripe necrosis virus and its fungal vector in South America. *European Journal of Plant Pathology* **105**, 643-650.
- Morris J, Clover GRG, Harju VA, Hugo SA & Henry CM (2001) Development of a highly sensitive nested RT-PCR method for Beet necrotic yellow vein virus detection. *Journal of Virological Methods* **95**, 163-169.
- National report on the quarantine phytosanitary status of the territory of the Russian Federation in 2021 (2022). Plant Health and Quarantine, 2, pp. 2-13 (In Russian).
- Niesbach-Klösigen U, Guilley H, Jonard G & Richards K (1990) Immunodetection in vivo of Beet necrotic yellow vein virus-encoded proteins. *Virology* **178**, 52-61.
- Nurmukhamedov AK & Vasilyeva NO (2006) Possible ways of spreading sugar beet rhizomania (brief information). Ukrainian Academy of Agrarian Sciences. Institute of Sugar beet, 212-215 (in Ukrainian).
- Prillwitz H & Schlösser E (1992) Beet soil-borne virus: occurrence, symptoms and effect on plant development. *Mededelingen van de Faculteit Landbouwwetenschappen, Rijksuniversiteit Gent* **57**.
- Putz C (1985) Identification methods for beet necrotic yellow vein virus (BNYVV) and related viruses in beets. In: *Compte Rendu du 48e Congrès d'Hiver de l'Institut International de Recherches Betteravières*, pp. 391-398. Institut International de Recherches Betteravières, Brussels, Belgium.
- Putz C, Wurtz M, Merdinoglu D, Lemaire O & Valentin P (1988) Physical and biological properties of beet necrotic yellow vein virus isolates. In: Cooper JJ, Asher MJC eds. *Viruses with Fungal Vectors. Developments in Applied Biology 2*, Association of Applied Biologists, Wellesbourne, 83-97.
- Rahim MD, Andika IB, Han C, Kondo H & Tamada T (2007) RNA4-encoded p31 of Beet necrotic yellow vein virus is involved in efficient vector transmission, symptom severity and silencing suppression in roots. *Journal of General Virology* **88**, 1611-1619.
- Ratti C, Clover GRG, Autonell CR, Harju VA & Henry CM (2005) A multiplex RT-PCR assay capable of distinguishing Beet necrotic yellow vein virus types A and B. *Journal of Virological Methods* **124**, 41-47.
- Richard-Molard M (1985) Rhizomanie - situation en 1985. In: *Compte Rendu du 48e Congrès d'Hiver de l'Institut International de Recherches Betteravières*, pp. 347-360. Institut International de Recherches Betteravières, Brussels, Belgium.
- Rush CM (2003) Ecology and epidemiology of Benyviruses and plasmodiophorid vectors. *Annual Review of Phytopathology* **41**, 567- 592.
- Rush CM, French R & Heidel GB (1994) Differentiation of two closely related furoviruses using the polymerase chain reaction. *Phytopathology* **84**, 1366-1369.
- Rush MC & Heidel GB (1995) Furovirus diseases of sugar beets in the United States. *Plant Disease* **79**, 868-875.
- Ryazantsev DYu, Zhivaeva TS, Prihodko YuN & Zavriev SK (2012) [Diagnosis of sugar beet rhizomania]. *Plant Protection and Quarantine* **8**, 29-31 (in Russian).
- Saito M, Kiguchi T, Kusume T & Tamada T (1996) Complete nucleotide sequence of the Japanese isolate S of Beet necrotic yellow vein virus RNA and comparison with European isolates. *Archives of Virology* **141**, 2163-2175.
- Schaufele WR, Buchse A, Buttner G & Munzel L (1995) [RIZO-QUICK: possibility to improve the field diagnosis for rhizomania]. *Zuckerindustrie* **120**, 294-298.

- Schirmer A, Link D, Cognat V, Moury B, Beuve M, Meunier A, Bragard C, Gilmer D & Lemaire O (2005) Phylogenetic analysis of isolates of Beet necrotic yellow vein virus collected worldwide. *Journal of General Virology* **86**, 2897-2911.
- Scholten OE, De Bock TSM, Klein-Lankhorst RM & Lange W (1999) Inheritance of resistance to Beet necrotic yellow vein virus in *Beta vulgaris* conferred by a second gene for resistance. *Theoretical and Applied Genetics* **99**, 740-746.
- Scholten OE, Jansen RC, Keizer LCP, DeBock TSM & Lange W (1996) Major genes for resistance to Beet necrotic yellow vein virus (BNYVV) in *Beta vulgaris*. *Euphytica* **91**, 331-339.
- Shmitt C, Balmori E, Jonard G, Richards KE & Guilley H (1992) In vitro mutagenesis of biologically active transcripts of Beet necrotic yellow vein virus RNA2: Evidence that a domain of the 75-kDa readthrough protein is important for efficient virus assembly. *Proceedings of the National Academy of Sciences of the USA* **89**, 5715-5719.
- Sohi HH & Maleki M (2004) Evidence for presence of types A and B of Beet necrotic yellow vein virus (BNYVV) in Iran. *Virus Genes* **29**, 353-358.
- Tamada T (1975) Beet necrotic yellow vein virus. *CMI/AAB Descriptions of Plant Viruses* No. 144. Association of Applied Biologists, Wellesbourne, UK.
- Tamada T (1999) Benyviruses. In: Webster R & Granoff A (eds). *Encyclopedia of virology*, 2nd ed., Vol.II., Academic Press, New York, 154-160.
- Tamada T (2002) Beet necrotic yellow vein virus. *CMI/AAB Descriptions of Plant Viruses* **391**, 5s.
- Tamada T & Abe H (1989) Evidence that Beet necrotic yellow vein virus RNA-4 is essential for efficient transmission by the fungus *Polymyxa betae*. *Journal of General Virology* **70**, 3391-3398.
- Tamada T & Baba T (1973) Beet necrotic yellow vein virus from rhizomania affected sugar beet in Japan. *Annals of Phytopathological Society of Japan* **39**, 325-331.
- Tamada T, Kusume T, Uchino H, Kiguchi T & Saito M (1996) Evidence that Beet necrotic yellow vein virus RNA 5 is involved in symptom development of sugarbeet roots. *Proceedings of the Third Symposium of the International Working Group on Plant Viruses with Fungal Vectors*, 49-52.
- Tamada T, Shirako Y, Abe H, Saito M & Kiguchi T (1989) Production and pathogenicity of isolates of Beet necrotic yellow vein virus with different numbers of RNA components. *Journal of General Virology* **70**, 399-409.
- Tamada T, Uchino H, Kusume T & Saito M (1999) RNA 3 deletion mutants of Beet necrotic yellow vein virus do not cause rhizomania disease in sugar beets. *Phytopathology* **89**, 1000-1006.
- Torrance L & Mayo MA (1997) Proposed re-classification of furoviruses. *Archives of Virology* **142**, 435-439.
- Ui T (1973) A monographic study of rhizomania of sugar beet in Japan. *Proceedings of the 13th Research Meeting of the Sugar Beet Technological Cooperative of Japan* **1**, 233-265.
- Vetter G, Hily JM, Klein E, Schmidlin L, Haas M, Merkle T & Gilmer D (2004) Nucleo-cytoplasmic shuttling of the Beet necrotic yellow vein virus RNA-3-encoded p25 protein. *Journal of General Virology* **85**, 2459-2469.
- Ward L, Koenig R, Budge G, Garrido C, McGrath C, Stubbley H & Boonham N (2007) Occurrence of two different types of RNA5-containing Beet necrotic yellow vein virus in the UK. *Archives of Virology* **152**, 59-73.
- Wisler GC, Liu HY & Duffus JE (1994) Beet necrotic yellow vein virus and its relationship to eight sugar beet furo-like viruses from the United States. *Plant Disease* **78**, 995-1001.
- Yanar Y, Dide Kultuk N & Erkan S (2006) Alternative weed hosts of Beet necrotic yellow vein virus and Beet soil-

borne virus in North-East of Turkey. *International Journal of Virology* **2**, 50-54.

Ziegler V, Richards K, Guilley H, Jonard G & Putz C (1985) Cell-free translation of Beet necrotic yellow vein virus: read through of the coat protein cistron. *Journal of General Virology* **66**, 2079-2087.

ACKNOWLEDGEMENTS

This datasheet was extensively revised in 2023 by Yuri Prikhodko from All-Russian Plant Quarantine Center. His valuable contribution is gratefully acknowledged.

How to cite this datasheet?

EPPO (2025) *Benyavirus necrobetae*. EPPO datasheets on pests recommended for regulation. Available online. <https://gd.eppo.int>

Datasheet history

This datasheet was first published in the EPPO Bulletin in 1988 and revised in the two editions of 'Quarantine Pests for Europe' in 1992 and 1997, as well as in 2023. It is now maintained in an electronic format in the EPPO Global Database. The sections on 'Identity', 'Hosts', and 'Geographical distribution' are automatically updated from the database. For other sections, the date of last revision is indicated on the right.

CABI/EPPO (1992/1997) *Quarantine Pests for Europe* (1st and 2nd edition). CABI, Wallingford (GB).

EPPO (1988) Data sheets on quarantine organisms No. 162, Beet necrotic yellow vein virus. *EPPO Bulletin* **18**(3), 527-532. <https://doi.org/10.1111/j.1365-2338.1988.tb00410.x>



Co-funded by the
European Union