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

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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).

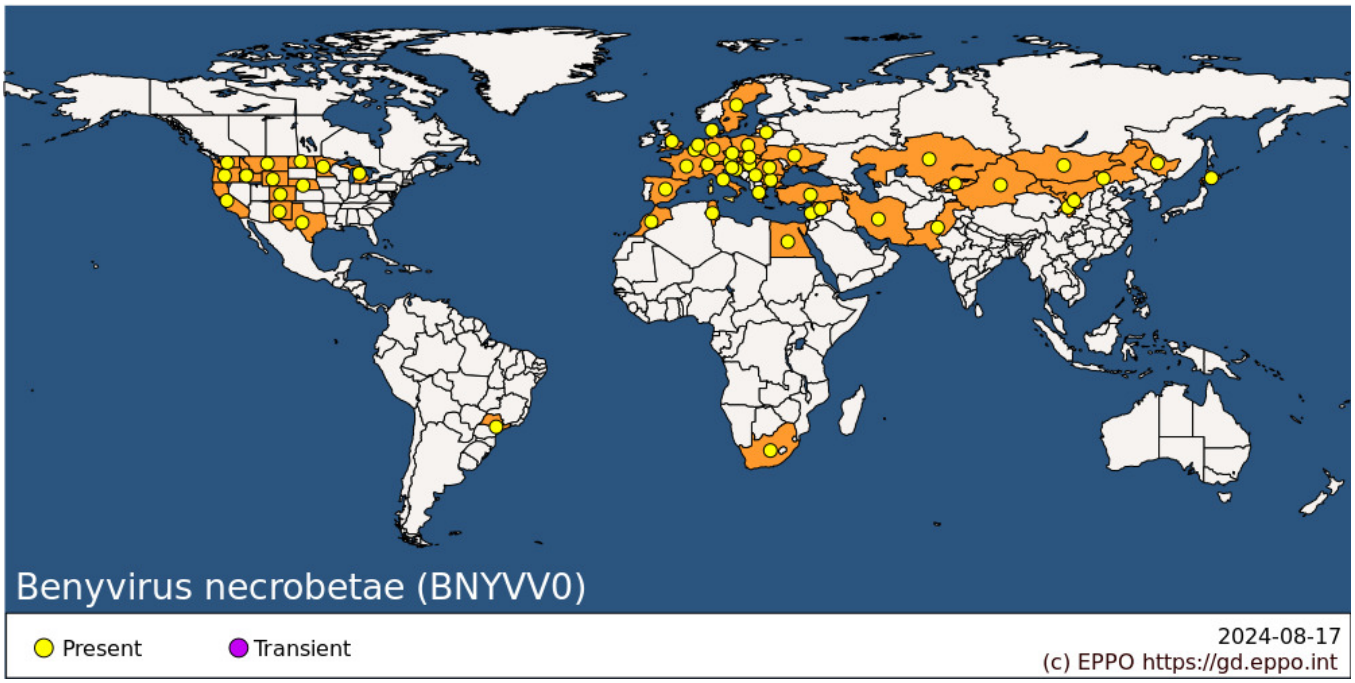
Host list: *Amaranthus retroflexus, Beta vulgaris, Blitum capitatum, Chenopodium album, Chenopodium vulvaria, Cichorium intybus, Cirsium arvense, Datura stramonium, Gomphrena globosa, Heliotropium europaeum, Lipandra polysperma, Matricaria chamomilla, Persicaria maculosa, Plantago major, Polygonum aviculare, 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, Czech Republic, 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, Japan (Hokkaido), Kazakhstan, Kyrgyzstan, Lebanon, Mongolia, Pakistan, Syria
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 (Lennefors *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 & Lennefors 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 & Lennefors, 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 Plasmodiophoridales (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)/ (BNYVVV 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).

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