Review Article

Rhizomania - A Review

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ABSTRACT

The productivity of sugar beet is strongly limited by several biotic stresses, among them rhizomania is one of the important factor causing yield loss of 20–50% or more. Beet necrotic yellow vein virus is the etiological agent of the destructive disease. The BNYVV belongs to the Benyvirus genus and is transmitted by the soil-borne fungus Polymyxa betae. Virus can survive within thick-walled resting spores (cystosori) of P. betae for more than two decades in soil. Root proliferation is the well-known characteristic of the viral infection that leads to yield and sugar losses. No authorized chemical treatment for rhizomania exists. Crop rotation does not appreciably reduce disease risk because of the long-term survival of cystosori. The only effective way to control it is by using a seed variety with resistant genetics. Genetic resistance to BNYVV was initially identified by the Holly Sugar Company in 1983. The dominant gene was coined as the holly gene or Rz1 but it confers partial resistance. Extensive use of sugar beet cultivar showing partial resistance although allows containment of sugar yield, on the other hand it permits the viruliferous vector to be amplified and therefore emergence of resistance breaking isolates. To counter this risk, in the United States SESVander Have has developed Rhizomania resistant varieties based on the “Tandem Technology®”. The hybrid possesses resistance that combines the ‘Holly’ gene with another source of resistance from Beta maritima of which SESVander Have is the sole holder. In addition artificially generated resistance represents an alternative to the natural resistance and generated higher protection level than Rz1.

Keywords
Rhizomania, Benyvirus, Polymyxa betae, Rz1, Tandem technology.

Introduction

Rhizomania is considered as one of the most serious diseases of sugar beet worldwide. This disease primarily attacks the root of the plant from the soil covering stage in June onwards. The term ‘Rhizomania’ is Greek for ‘root madness’ and was chosen by Canova in 1966 due to one of the most characteristic symptoms of the disease: a proliferation of lateral rootletls along the main tap root. Though Rhizomania was first described in Italy in 1959, but the etiological agent was not known until 1973, when two Japanese plant pathologists Tamada and Baba showed that rhizomania is caused by a phytovirus named Beet necrotic yellow vein virus, BNYVV (Tamada and Baba, 1973).

The BNYVV belongs to the Benyvirus genus and is transmitted by the soil-borne fungus Polymyxa betae, a member of the Plasmodiophoromycetes (Koenig and Lennefors, 2000). The fungus is an
intracellular obligate parasite restricted to the roots of Chenopodiaceae forming spores (cystosori), which are the resistant stage and preserve the virus in the soil for many years (Richards and Tamada, 1992).

The BNYVV genome organization consists of several genomic single-stranded plus-sense RNAs. RNAs 1 and 2 encode “house-keeping” genes allowing replication and cellular translocation whereas RNAs 3, 4 and 5 are necessary for vector-mediated infection and disease development in sugar beet roots (Richards and Tamada, 1992).

This disease primarily attacks the root of the plant leading to abnormal proliferation of secondary rootlets around the tap root, necrotic rings in the root section, and chlorotic leaves.

From a production point of view, the disease reduces root yield by 45–50% or more and sugar content by 60–79% (Casarini Camangi, 1987). Rhizomania has caused major reductions in root yield and quality wherever it occurred. Fortunately, strong genetic tolerance to BNYVV, conferred by the Rz1 gene, was identified (Biancardi et al., 2002).

Geographical distribution

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).

Sixty years after the discovery of the virus in Italy, Rhizomania is widespread in many Europeans countries and is also present in other sugar beet growing areas including United States, CIS countries, China and Japan (McGrann et al., 2009).

Symptoms

The Rhizomania syndrome refers to root madness (Rhizo: root; Mania: madness). Infected sugar beets display more or less a dwarfism that reduces the tap root size, which harbors necrosis. Infection shapes a wine-glass-like taproot and induces rootlet proliferations that become necrotic, abundant and fragile. These root symptoms reduced water uptake that provoke leaf fading. Sometimes, when the infection becomes systemic, vein yellowing, necrosis and foliar local lesions appear. 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. Hayasaka et al., (1988) suggested that chlorosis might be due to a deficiency in mineral nutrients caused by inhibition of nutrient absorption by roots severely damaged by virus infection. Diseased roots present higher contents of reducing sugars, K and Na, and lower content of total N, NH₂–N, NH₄–N and betaine concentrations with respect to healthy roots (Uchino and Kanzawa, 1995). However, BNYVV can also cause latent infections with no visible symptoms. This is especially the case under cool spring conditions (Lindsten, 1986).

The pathogen- Beet necrotic yellow vein virus (BNYVV)

BNYVV possesses a multipartite linear positive-sense single stranded RNA genome that consists of four to five RNAs possessing 5 cap and polyadenylated 3 ends (Tamada1999). BNYVV belongs to the super group of alpha virus-like and is the type species of the Benyvirus genus. (Lee et al., 2001). The virus is rod-shaped, with a helical symmetry. Its diameter is about 20 nm with lengths proportional to the sizes of the encapsidated RNAs, i.e., 390, 265, 105, 89 and 80nm (Putz, 1977; Tamada et al., 1989).
RNA-1 and -2 are necessary and sufficient for the infection following leaf mechanical inoculations where small components are dispensable and, if they are present, can undergo deletion or disappear (Bouzoubaa et al., 1991). In natural infection, however, these small components are required. Indeed, RNA-3 allows the viral amplification in sugar beet roots and its expression influences symptoms (Tamada et al., 1989; Jupin et al., 1992), whereas RNA-4 is involved in viral transmission (Tamada and Abe, 1989). Moreover, RNA-4-encoded p31 is described as a root specific silencing suppressor (Rahim et al., 2007). Therefore, BNYVV is a unique virus as it behaves as a bipartite virus when rub inoculated or as a tetra or pentapartite virus in natural infection.

Three strains of the virus (A, B and P types) have been identified according to their structure of RNAs (Tamada, 2002). Type A is the most common and is present in most European countries as well as in North America, Japan and China. Type B is also common in France, Germany and Great Britain.

The P type is generally believed to be more aggressive and contains the additional RNA 5 and has been identified mainly near the Pithiviers area of France and Kazakhstan (Koenig and Lennefors, 2000). Type P is currently much talked about as it appears to carry the largest concentrations of the virus in the vector (Büttner et al., 2004).

**Disease cycle**

The soil borne fungus, *Polymyxabetae*, serves as a vector of BNYVV by carrying the virus to healthy roots. The association of BNYVV with the fungus is an unusual biological relationship that results in rhizomania development when a susceptible host is present and conditions are favorable for infection. *P. betae* is ubiquitous. Its distribution covers all beet-growing areas (Rush, 2003). *P. betae* belongs to the *Plasmodiophoridae*, a monophyletic group including ten genera. For a long time ranked among the lower fungi, it is now classified as a protist (Archibald and Keeling, 2004).

The biological cycle of the Rhizomania depends on the lifecycle of the vector. This vector great capacity for survival explains, firstly, the recurrence of viral diseases in contaminated fields and, secondly, their dissemination either through the soil adhering to agricultural machinery and produce or via drainage and irrigation water. The fungus forms two types of spores during its life cycle, resting spores and motile zoospores. Clusters of tiny, thick-walled resting spores, also called cystosori, enable the fungus to survive in soil for 15 years or longer in the absence of a suitable host (Adams, 1990; Maraite, 1991). The virus also can persist in these resting spores for at least 15 years. When soil conditions become favourable for infection, germination of the resting spore is triggered by the presence of a host-plant root. As resting spores germinate, motile zoospores are released that actively swim to the root surface where new infections occur. Resting spore germination releases biflagellated zoospores 4-5μm size which move through the free water in the soil before encysting on a root hair or epidermal cell of a host plant root. In order to penetrate the cell, a dagger-like structure called “Stachel (sting)” is formed in the encysted zoospore (Keskin and Fuchs, 1969) and a sticky tube develops between the cyst and the plant cell wall. The Stachel, which is injected into the host cell through the tube, pierces the cell wall and the plasma membrane and allows the zoospore content to be transferred into the cytoplasm of the host cell (Barr, 1988). Infection of roots by zoospores results in the formation of a fungus body, or plasmodium, inside the root.
In the sporangial phase this plasmodium develops into a multi-lobed zoosporangium enclosed by a thin wall within which the secondary zoospores are formed. The secondary zoospores are released outside the root, or sometimes into the deeper root cells, by small plasmodial cells, which dissolve a hole in the cell wall (Barr 1988). In the sporogenic phase noncruciform nuclear divisions are observed, with the formation of synaptonemal complexes characteristic of meiosis (Braselton, 1988). The plasmodium divides into mononucleate cells by forming membrane layers within the cytoplasm. A four to five layer wall is then deposited between the cells, with adjacent spores remaining connected by bonds between the two outer most layers (Chen et al., 1998). The zoospores formed remain in the root debris and are released into the soil by root decomposition. Within this life cycle the moments of cell fusion and karyogamy have not yet been pinpointed. Observation of double size quadriflagellate zoospores (Ledingham, 1939) suggests fusion of two zoospores, but the moment of nuclear fusion is not known. Both spore types thus formed become viruliferous or carriers of BNYVV.

Virus transmission by plasmodiophorids was for many years regarded as a passive mechanism, which occurred during mixing of plant cell cytoplasms and the protozoan, prior to membrane formation (Campbell, 1996). However, recent research has revealed the special role played by some viral proteins in the process of transmission by the vector. The BNYVV capsid protein readthrough (RT) domain plays an important part in the transmission process, since deletions in the C-terminal portion of this domain are correlated to loss of virus transmission. Substituting the four KTER amino acids located in position 553 to 556 of the RT domain by the ATAR motif completely blocks transmission (Tamada et al., 1996). A comparative analysis of the viral genomes transmitted by plasmodiophorids, which do not have the same genomic organisation, has identified the presence of two complementary transmembrane domains in the RT domains of the capsid protein of Beny-, Furo- and Pomovirus and in the P2 proteins of Bymovirus (Adams et al., 2001). Deletion or substitution of the second domain also blocks transmission by the vector. The molecular model is not yet detailed, but the transmembrane helical sequences may perhaps determine a particular structure facilitating membrane invagination and virus movement through the membrane of the vector (Adams et al., 2001).

**Dispersal and growth factors**

The main means of spread is roots of infected plants, infected beet stocklings (possibly imported by breeders), and soil containing *P. betae* carrying BNYVV (which could accompany beet roots or potatoes, or possibly beet seed, or any vegetables grown on infested land etc.) (Hleibieh et al., 2007). Sugar beet waste, washing water and agricultural equipment (especially 4 Beet necrotic yellow vein furovirus harvesters) have been shown to be the main carriers at the local level. Stable manure can also play a role in the dispersal of BNYVV since *P. betae* is capable of passing undamaged through the digestive tracts of animals (Heijbroek, 1988).

The environmental factors that will contribute to the development of the disease are those that favour the proliferation of its vector in the soil. Usually three important conditions are essential for proliferation and root infection by *Polymyxa betae* such as presence of a host plant; presence of free water in the soil to promote germination and facilitate zoospore access to the roots, and a sufficiently high temperature between 10 and 30°C, ideally between 20 and 25°C according
to the origin of the strains (Legrève et al., 1998; Webb et al., 2000). The soil pH and calcium content also affect vector activity. Spore germination and root infection by zoospores are affected by acid pH conditions (Abe and Tamada, 1987). They are promoted in neutral or alkaline pH soils, especially if the calcium and magnesium levels are greater than 350 and 20mg/100g of soil respectively (Goffart and Maraite, 1991).

**Disease diagnosis**

Because symptom expression varies greatly, diagnosis of rhizomania cannot be based solely on visual inspection. Instead, an accurate diagnosis is done by a serological ELISA test, or enzyme-linked immunosorbent assay. 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). To maximize the likelihood of an accurate test, sugar beet samples collected for testing should include new fibrous root growth, occurring immediately after rainfall or irrigation, and samples should arrive at the laboratory within one day after collection. 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*).

In soil or 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 and Hani, 1985). Bait plant tests to estimate soil infestation with BNYVV using pre-grown sugar beet seedlings can be used to estimate the level of infestation (Goffart et al., 1989) as well as to calculate potential yield losses. However, these tests are not reliable enough for detecting very low levels of infestation and are, therefore, unsuitable for establishing that fields are free from the virus (Büttner and Bürcky, 1990).

**Management strategies**

Continuous planting or close rotation of sugar beet increases the risk of loss due to rhizomania. Early planting, when soil temperatures are cooler, and use of production practices that result in the rapid establishment of the plant canopy, will reduce risk of loss. Early planting should be done at slightly greater plant densities to compensate for increased seedling loss in cooler soils. Büttner et al., (1994) propose a soil test to determine the risk of rhizomania, as an aid to selection of the appropriate cultivar to be sown. Extending crop rotation is advised but will only have a limited effect on the infectious potential of the soil given *Polymyxa betae*/BNYVV’s capacity to survive for decades in the soil (Hleibieh et al., 2007).

Chemical control methods against the vector are either too expensive (methyl bromide soil disinfection) or ineffective. However, some soil fumigants, such as Telone II, may kill enough cystosori to reduce disease development to acceptable levels. Fumigation treatments are very expensive, and research is being done to determine their efficacy and conditions under which they should be used.

Breeding for resistance to rhizomania is the most effective strategy for controlling this disease (Scholten and Lange 2000). The development and use of new rhizomania resistant varieties is a high priority due to the increasing spread of the disease in many sugar beet cultivated areas (McGrann et al., 2009). The search for tolerant or resistant cultivars has been actively carried out since 1978 and the results obtained have been very encouraging. The first breeding project on rhizomania resistance began in 1970 in Italy.
The discovery of the first multigenic resistant source, defined “Alba type”, was originally derived from sugar beet progenitor Beta maritima belonging to Munerati’s germplasm. The sugar beet cultivar with the “Rizor” source of resistance, developed in 1985 by De Biaggi, was the first variety showing an optimum level of resistance on rhizomania infested fields (De Biaggi, 1987). Later, the source “Holly” was isolated through USDA breeding programs at Salinas (California, USA) in collaboration with Holly Sugar Company in California (Lewellen et al., 1987). These sources of resistance have good heritability and a few cycles of selection are sufficient for improving the trait. Resistances such as “Rizor” and “Holly” are classified as monogenic (Biancardi et al., 2002).

Rizor/Holly is still the most widely used source of resistance to rhizomania and, at the commercial level, the locus is commonly referred asRz1. A different mongenic resistance gene was identified at the USDA in Salinas (California, USA) in a sea beet population (WB42) originating from Denmark (Lewellen et al., 1987) and was named Rz2. Recently, Acosta-Leal et al., (2010) demonstrated that BNYVV virusteters on sugar beet plants exposed to the same original soil inoculum were higher in Rz1 sugar beets with respect to Rz2 ones. Other molecular studies performed by Gidner et al., (2005) identified another resistance gene (Rz3) on a mapping population obtained by the cross between WB41 accession derived from a B. maritima population of Denmark (Lewellen et al., 1987; Whitney 1989) and a susceptible line from the Syngenta germplasm. Grimmer et al., (2007) discovered a major QTL for rhizomania resistance in the segregating population named R36. This mapping population was derived from C50 (Lewellen and Whitney, 1993), a composite cross of B. maritima accessions with sugar beet. The QTL conferring the resistance was named Rz4. Nevertheless, further studies are needed to clarify if Rz4 is a novel resistance gene or a new allele at a

The development and use of resistant varieties to rhizomania allowed beet growers to significantly reduce the damage caused by rhizomania for more than 20 years. Extensive use of sugar beet cultivar showing partial resistance although allows containment of sugar yield, on the other hand it permits the viruliferous vector to be amplified and therefore emergence of resistance breaking isolates. Recent studies have shown an emergence of new BNYVV strains with increased virulence that could overcome Rz1 resistance (Rush et al., 2006; Acosta-Leal et al., 2010). To put it in perspective, the use of varieties carrying only a single gene for resistance against rhizomania might be inadequate for an effective control of disease.

To counter this risk, in the United States, SESVanderHave has developed Rhizomania resistant varieties based on the “Tandem Technology®”. The hybrid possesses resistance that combines the ‘Holly’ gene with another source of resistance from Beta maritima of which SESVanderHave is the sole holder (Meulemans et al., 2003). Tandem technology produces excellent results even under extreme Rhizomania pressure.

In addition artificially generated resistance represents an alternative to the natural resistance. The transgenic expressions of virus-derived sequences that form double stranded RNA (dsRNA) has been obtained and lead to BNYVV resistance (Lennefors et al., 2008). Expression of dsRNA induces RNA silencing (Baulcombe, 2004, 2005; Filipowicz et al., 2005), an innate defense mechanism against invasive nucleic acids that leads to the sequence specific degradation of RNA. High levels of resistance to rhizomania were obtained in sugar beets expressing a 0.4
kb inverted repeat construct based on a partial BNYVV replicase gene derived sequence (Lennefors et al., 2008).

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