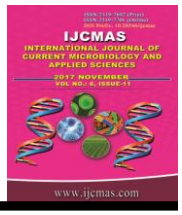
Int.J.Curr.Microbiol.App.Sci (2017) *6*(11): 358-367

International Journal of Current Microbiology and Applied Sciences
ISSN: 2319-7706 **Volume 6 Number 11 (2017) pp. 358-367**
Journal homepage: http://www.ijcmas.com



Review Article https://doi.org/10.20546/ijcmas.2017.611.040

Rhizomania - A Review

Licon Kumar Acharya[1*], Surabhi Hota[2] and Kartik Pramanik[3]

[1]Plant pathology, Centurion University of Technology and Management, India
[2]Indira Gandhi Krishi Viswavidyalaya, India
[3]Horticulture, Centurion University of Technology and Management, India
**Corresponding author*

Keywords

Rhizomania, Benyvirus, Polymyxabetae, Rz1, Tandem technology.

Article Info

Accepted:
04 September 2017
Available Online:
10 November 2017

A B S T R A C T

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 *Polymyxabetae*. 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 characteristics 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 generatedhigherprotectionlevelsthan*Rz*1.

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 rootlets along the main tap root. Though Rhizomania was first described in Italy in1959, 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 *Polymyxabetae*, 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, NH_2–N, NH_4–N and betaine concentrations with respect to healthy roots (Uchino and Kanzawa, 1995). However, BYNVV 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 4-5µ min size which moves 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 noncruci form 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 sporosores 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 stecklings (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 *Polymyxabetae* 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 inneutralor 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 (*Chenopodiumquinoa*).

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 *Polymyxabetae*/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 monogenic 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 virustiters 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). Toput 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).

References

AbeH and Tamada T. 1987. Atesttubeculturesystemformultiplication of *Polymyxabetae* and *Beetnecrotic yellow vein virus* in rootlets of sugarbeet. In: *Proceedings of the Sugar Beet Research Association, Japan.* 29: 34-38.

Acosta-Leal R, Brian BK, and Rush CM. 2010. Host effect on the genetic diversification of Beet necrotic yellow vein virus single-plant populations. Phytopathology. doi: 10.1094/PHYTO-04-10-0103.

Acosta-Leal R and Rush CM. 2007. Mutations associated with resistance-breaking isolates of *Beet necrotic yellow vein virus* and their allelic discrimination using Taqman technology. *P hytopathology*. 97: 325-330

Adams MJ, Antoniw JF, Mullins JG. 2001. Plant virus transmission by plasmodiophorid fungi is associated with distinctive transmembrane regions of virus-encoded proteins. *Archives of Virology*. 146: 1139-1153.

Adams MJ. 1990. Epidemiology of fungally transmitted viruses. *Soil Use and Management.* 6: 184-189.

Archibald JM and Keeling PJ. 2004. Actin and ubiquitin protein sequences support acercozoan/ foraminiferan ancestry for the plasmodiophorid plant pathogens. *Journal of Eukaryotic Microbiology*. 51: 113-118.

Barr DJS. 1988. Zoosporic plant parasites as fungal vectors of viruses: Taxonomy and life cycles of species involved. In: Cooper JI, Asher MJ (Eds) *Developments in Applied Biology II Viruses with Fungal Vectors*, Association of Applied Biologists, Wellesbourne, UK, pp. 123-137.

Baulcombe D. 2004. RNA silencing in plants. *Nature.* 431: 356-363.

Baulcombe, D. 2005. RNA silencing. *Trends in Biochemical Sciences.* 30: 290-293.

Biancardi E, Lewellen RT, Biaggi MD, Erichsen AW and Stevanato P. 2002. The origin of rhizomania resistance in sugarbeet. *Euphytica.* 127: 383-397

Bouzoubaa, S, Niesbach-Klosgen U, Jupin I, Guilley H, Richards K and Jonard G. 1991. Shortened forms of beet necrotic yellow vein virus RNA-3 and-4: Internal deletions and a subgenomic RNA. *Journal of General Virology.* 72: 259-266.

Braselton, JP. 1988. Karyology and systematics of plasmodiophoromycetes. In: Cooper JI, Asher MJ (Eds) *Developments in Applied Biology II Viruses with Fungal Vectors*, Association of Applied Biologists, Wellesbourne, UK, pp. 139-152.

Büttner G, Büchse A, Holtschulte B and Märlander B. 2004. Pathogenicity of different forms of *Beet necrotic yellow vein virus* (BNYVV) on sugar beet – is there evidenced for the development of pathotypes? In: Proceedings of the 67th IIRB Congress, February 2004, Brussels (B).

Büttner G, Mutzek E and Bürcky K. 1994. Possibilities of rhizomania forecasts as decision aids for the selection of suitable varieties. *GesundePflanzen.* 46: 33-39.

Büttner, G and Bürcky K. 1990. Experiments and considerations on the detection of BNYVV in soil by means of bait plants. *ZeitschriftfürPflanzenkrankheiten und Pflanzenschutz.* 97: 56-64.

Campbell RN. 1996. Fungal transmission of plant viruses. *Annual Review of Phytopathology*. 34:87-108.

Canova A. 1959. Appunti di patologia dellabarbabietola. Informatore Fitopatologico. 9(20): 390–396.

Casarini Camangi P. 1987. Incidenzadellarizomaniasullaproduzione quali-quantitativadellabarbabietola da zucchero. *L'Informatore Agrario*.43: 149–152.

Chen JP, Wang ZQ, Hong J, Collie rCR and Adams MJ. 1998. Ultra structural studies of resting spore development in *Polymyxa graminis*. *Mycological Research.* 102: 687-691

DeBiaggi M. 1987. Méthodes de sélection, uncas concret. In: International Institute for Beet Research (IIBR) (Ed) *Proceedings of the 50th Congress of the IIRB*, 11-12 February 1987, IIRB, Brussels. Pp. 157-163.

Filipowicz, W, Jaskiewicz L, Kolb FA and Pillai RS. 2005. Post-transcriptional gene silencing by siRNAs and miRNAs. *Current Opinion in Structural Biology*. 15. 331-341.

Gidner S, Lennefors BL, Nilsson NO, Bensefelt J, Johansson E, Gyllenspetz U and Kraft T. 2005. QTL mapping of BNYVV resistance from the WB41 source in sugarbeet. *Genome.* 48: 279-285.

Goffart, JP, Horta V and Maraite H. 1989. Inoculum potential and host range of *Polymyxabetae* and beet necrotic yellow vein furovirus. *Bulletin OEPP/EPPO Bulletin.* 19: 517-525.

Goffart JP and Maraite H. 1991. Soil and agronomic factors affecting the inoculum potential of *Polymyxabetae* keskinin Belgium. *Parasitica.* 47: 165-192.

Grimmer M, Trybush S, Hanley S, Francis S, Karp A and Asher M. 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.

Hayasaka M, H. Uchino, E. Imura, and K. Kanzawa. 1988. Content of sugar and mineral nutrients of sugarbeets, classified by type of rhizomania symptoms. Proceedings of the Japanese Society of Sugar Beet Technologists. 30: 94–99.

Heijbroek W, Musters PMS and Schoone AHL. 1999. Variation in pathenogenicity and multiplication of *Beet necrotic yellow vein virus* (BNYVV) in relation to the resistance of sugar beet cultivars. *European Journal of Plant Pathology.* 105: 397-405.

Hleibieh K, Peltier C, Klein E, Schirmer A, Schmidlin L, Covelli L, Ratti C, Legrève A, Bragard C and Gilmer D. 2007. Etiologie de la rhizomanie de la betteravesucrière. Virologie. 11 (6): 409-421.

Hleibieh K, Peltier C, Klein E, Schirmer A, Schmidlin L, Covelli L, Ratti C, Legrève A, Bragard C and Gilmer D. 2007. Etiologie de la rhizomanie de la betteravesucrière. Virologie. 11 (6): 409-421.

Jupin I, Guilley H, Richards KE and Jonard G. 1992. Two proteins encoded by beet necrotic yellow vein virus RNA3 influence symptom phenotype on leaves. *The EMBO Journal.* 11: 479-488

Keskin B and Fuchs WH. 1969. The process of infection by *Polymyxabetae*. *ArchivfürMikrobiologie*, 68, 218-226.

Koch F. 1982. Rhizomania of sugar beet]. In: *CompteRendu du 45e Congrèsd'Hiver de l'Institut International de Recherches Betteravières*, pp. 211-238. Institut International de Recherches Betteravières, Brussels, Belgium.

Koenig R and 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.

Ledingham GA. 1939. Studies of *Polymyxagraminis*, n.Gen.N.Sp., a plasmodiophoraceous root parasite of wheat. *Canadian Journal of Research.* 17, 38-51

Lee L, Telford EB, Batten JS, Scholthof KB and Rush CM. 2001. Complete nucleotide sequence and genome organization of *Beet soil borne mosaic virus*, a proposed member of the genus *Benyvirus*. *Archives of Virology.* 146: 2443-2453.

Legrève A, Delfosse P, Vanpee B, Goffin A and Maraite H. 1998. Differences in temperature requirements between *Polymyxa* sp. of Indian origin and *Polymyxa graminis* and *Polymyxa betae* from temperate areas. *European Journal of Plant Pathology.* 104: 195-205

Lennefors BL, vanRoggen PM, Yndgaard F, Savenkov EI and Valkonen JP. 2008. Efficientd sRNA-mediated transgenic resistance to *Beetnecrotic yellow vein virus* in sugar beets is not affected by other soil borne and aphid-transmitted viruses. *Transgenic Research.*17: 219-228

Lewellen RT, Skoyen IO and Erichsen AW. 1987. Breeding sugar beet for resistance to rhizomania: Evaluation of host-plant reactions and selection for and inheritance. In: International Institute for Beet Research (IIBR) (Ed) *Proceedings of the 50th Congress of the IIRB*, 11-12 February 1987, IIRB, Brussels. Pp. 139-156.

Lindsten K. 1986. Rhizomania - a complicated disease in sugar beets which can also occur in Sweden. *Växtskyddsnotiser*. 50: 111-118.

Maraite H. 1991. Transmission of viruses by soil fungi. In: Beemster ABR, Bollen GH, Gerlagh M, Ryuissen MA, Schipers B, Tempels A (Eds) *Developments in Agricultural and Managed Forest Ecology: Biotic Interactions and Soil-Borne Diseases*, Elsevier Science Publishers, Amsterdam, NL. Pp. 67-82.

McGrann, GR, Grimmer MK, Mutasa-Go¨ttgens ES and Stevens M. 2009. Progress towards the understanding and control of sugar beet rhizomania disease. Molecular Plant Pathology. 10:129–141.

Merz V and Hani A. 1985. A bait plant test to determine infection potential of BNYVV and *Polymyxabetae*in soil samples. In: *CompteRendu du 48e Congrèsd'Hiver de l'Institut International de Recherches Betteravières*, pp. 421-430. Institut International de Recherches Betteravières, Brussels, Belgium.

Meulemans M, Janssens L and Horemans S. 2003. Interactions between major genes, and influence of the genetic background in the expression of rhizomania resistance. In: Proceedings of the 1st joint IIRB-ASSBT Congress, 26th Feb.-1st March 2003, San Antonio (USA).

Putz, C. 1985. Identification methods for beet necrotic yellow vein virus (BNYVV) and related viruses in beets. In: *CompteRendu du 48e Congrèsd'Hiver de l'Institut International de RecherchesBetteravières*, pp. 391-398. Institut International de Recherches Betteravières, Brussels, Belgium.

Putz C. 1977. Composition and structure of *Beet necrotic yellow vein virus*.

Rahim MD, Andika IB, Han C, Kondo H and 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.

Richards KE and Tamada T. 1992. Mapping functions on the multipartite genome of beet necrotic yellow vein virus. Annual

Review of Phytopathology. 30: 291–313.

Rush CM, HY Liu, RT Lewellen, and R Acosta-Leal. 2006. The continuing saga of rhizomania of sugar beets in the United States. Plant Disease. 90: 4–15.

Rush CM. 2003. Ecology and epidemiology of *Benyviruses* and plasmodiophorid vectors. *Annual Review of Phytopathology*. 41: 567-592

Scholten OE and Lange W. 2000. Breeding for resistance to rhizomania in sugarbeet: A review. *Euphytica*. 112: 219-231.

Tamada T and Baba T. 1973. Beet necrotic yellow vein virus from rhizomania-affected sugar beet in Japan. Ann Phytopathol Soc Jpn. 39:325–332.

Tamada T. 1975. Beet necrotic yellow vein virus. *CMI/AAB Descriptions of Plant Viruses* No. 144. Association of Applied Biologists, Wellesbourne, UK.

Tamada T. 2002. Beet necrotic yellow vein virus. In Descriptions of plant viruses. Available at www.dpvweb.net/dpv (cited 30 August 2010; verified 7 January 2011).

Tamada T and Abe H. 1989. Evidence that beet necrotic yellow vein virus RNA-4 is essential for transmission by the fungus *Polymyxabetae*. *Journal of General Virology*. 70: 3391-3398.

Tamada T, Schmitt C, Saito M, Guilley H, Richards K and Jonard G. 1996. High resolution analysis of the read through domain of beet necrotic yellow vein virus read through protein: AKTER motif is important for efficient transmission of the virus by *Polymyxabetae*. *Journal of General Virology*. 77: 1359-1367.

Tamada T, Uchino H, Kusume T and Saito M. 1999. RNA 3 deletion mutants of *Beet necrotic yellow vein virus* do not cause rhizomania disease in sugarbeets. *Phytopathology*. 89: 1000-1006

Uchino H and K. Kanzawa. 1995. Suppression of rhizomania by controlling soil pH at low level by ferrous sulfate in infested sugar beet field. Proceedings of the Japanese Society of Sugar Beet Technologists. 37: 114–121.

Webb CR, Gilligan CA and Asher MJC. 2000. Modelling the effect of temperature on the development of *Polymyxabetae*. *Plant Pathology*. 49: 600-607.

Whitney ED. 1989. Identification, distribution, and testing for resistance to rhizomania in *Betamaritima*. *Plant Disease*. 73: 287-290.

How to cite this article:

Licon Kumar Acharya, Surabhi Hota and Kartik Pramanik. 2017. Rhizomania - A Review. *Int.J.Curr.Microbiol.App.Sci.* 6(11): 358-367. doi: https://doi.org/10.20546/ijcmas.2017.611.040