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Biological and genetic diversity of plasmodiophorid-transmitted viruses and their vectors

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Abstract

About 20 species of viruses belonging to five genera, *Benyvirus, Furovirus, Pecluvirus, Pomovirus* and *Bymovirus*, are known to be transmitted by plasmodiophorids. These viruses have all positive-sense single-stranded RNA genomes that consist of two to five RNA components. Three species of plasmodiophorids are recognized as vectors: *Polymyxa graminis, P. betae,* and *Spongospora subterranea*. The viruses can survive in soil within the long-lived resting spores of the vector. There are biological and genetic variations in both virus and vector species. Many of the viruses have become the causal agents of important diseases in major crops, such as rice, wheat, barley, rye, sugar beet, potato, and groundnut. Control measure is dependent on the development of the resistant cultivars. During the last half a century, several virus diseases have been rapidly spread and distributed worldwide. For the six major virus diseases, their geographical distribution, diversity, and genetic resistance are addressed.

Keywords Soil-borne viruses; *Benyvirus*; *Furovirus*; *Pecluvirus*; *Pomovirus*; *Bymovirus*; Vector transmission; Plasmodiophorids; *Polymyxa*; *Spongospora*

Introduction

There is a group of viruses to be transmitted by olpidium and plasmodiophorid vectors, which is called soil-borne fungus-transmitted viruses. The vector species are obligate parasites of plant roots and have similar development stages. They were previously considered to belong to the fungi, but plasmodiophorids are at present classified as protists rather than fungi (Braselton 2001). Species in chytrid fungus *Olpidium* transmit viruses with isometric, kinked filamentous (naked nucleocapsid) or rod-shaped particles, whereas plasmodiophorid species transmit viruses with rod-shaped or filamentous particles with rare exceptions. These viruses are highly diverse, belonging to 12 genera in at least four families (Rochon et al. 2004). Two types of virus-vector relationships have been recognized, termed *in vitro* (non-persistent) and *in vivo* (persistent) (Campbell 1996).

Plasmodiophorid-transmitted viruses are known to be about 20 species belonging to five genera *Furovirus*, *Pecluvirus*, and *Pomovirus* (family *Virgaviridae*), *Benyvirus* (family unassigned), and *Bymovirus* (family *Potyviridae*) (Adams et al. 2009). Three species *Polymyxa graminis*, *P. betae*, and *Spongospora subterranea* are recognized as virus vectors. The viruses can be present in the plasmodiophorids during all stages of the life cycle and they can persist for many years in resting spores (so-called *in vivo* transmission). Most of the viruses cause severe diseases in major crops, such as rice, wheat, barley, oat, sugar beet, potato, and peanut. Recently, some virus diseases have been more widely distributed throughout the world and become serious problems in agriculture, because the eradication of the disease is very difficult and infestations are usually permanent. Control measures such as agronomic management or chemical treatments in diseased fields are not available; therefore, genetic resistance is the most promising approach for the disease control. This review summarizes recent aspects of biological properties of the plasmodiophorid-transmitted viruses and their vectors, emphasizing more studies involving biological and genetic variability, and for six major virus diseases, their geographical distribution, variation, and genetic resistance are described.

Viruses transmitted by plasmodiophorid vectors

Virus species and their genome structure

Table 1 lists viruses transmitted by plasmodiophorid vectors. They are all positive-sense single-stranded RNA (ssRNA) viruses belonging to five genera and some unclassified viruses (Adams et al. 2009; Rochon et al. 2004). Furo-, peclu-, pomo- and benyviruses have rod-shaped particles, whereas bymoviruses have flexuous filaments. For unclassified viruses, Aubian wheat mosaic virus has rod-shaped particles (Hariri et al. 2001). Exceptions are Watercress yellow spot virus (WYSV) (Tomlinson and Hunt 1987) and Watercress chlorotic leaf spot virus (WCLSV), which have isometric particles (about 38 nm) and resemble dianthoviruses (Walsh et al. 1989).

Fig. 1a shows the genome structure of the five genera. Furo-, peclu-, and bymoviruses have a bipartite genome, whereas pomoviruses have a tripartite genome. For benyvirus,

Beet necrotic yellow vein virus (BNYVV) has four to five ssRNA components, but other benyvirus, Burdock mottle virus (BdMoV), has only two components (Rush 2003). Among four genera with rod-shaped viruses, some common elements are present (Fig. 1a). One is that viral replicase is encoded by the longest ssRNA segment. Second is that at the 5′ end of the genome segment, viral coat protein (CP) is encoded, followed by a larger polypeptide which is produced by translational readthrough (RT) of the CP stop codon (CP-RT) with the exception of that of pecluviruses, in which this region is expressed via leaky scanning as an independent protein (p39). Additional common element in four genera is that small cysteine-rich protein (CRP) at the 3′ end of the genome is encoded, with the exception of some pomoviruses. Furovirus encodes the movement protein (MP) belonging to the 30K superfamily, whereas pomo-, peclu-, and benyviruses encode the triple gene block (TGB) that comprises TGB1, TGB2, and TGB3.

Bymovirus has a bipartite RNA genome (Fig. 1a), which differed from other virus genera in the *Potyviridae* that have a monopartite ssRNA genome. RNA1 encodes eight proteins, including the P3, cytoplasmic inclusion protein (CI), genome-linked protein (VPg), serine proteinase, nuclear inclusion protein b, and CP. RNA2 encodes cysteine proteinase (P1) and putative vector-transmission factor (P2).

Virus movement

Three distinct cell-to-cell movement strategies are employed by plasmidophorid-transmitted viruses. Furovirus encodes movement protein belonging to the 30K superfamily, whereas beny-, pomo-, and pecluviruses encode the TGB family (Fig. 1a). The movement protein of bymoviruses is likely associated with CI (Wei et al. 2010).

For furo-, beny-, pomo-, and pecluviruses, the CP is not required for cell-to-cell movement of virus, but it is essential for long-distance movement, except for pomoviruses (*Potato mop-top virus,* PMTV, and *Beet soil-borne virus*) which can move systemically in plant in the absence of CP (Torrance et al. 2011). It is generally thought that TGB1 interacts with viral RNA and possibly other viral or plant factors to form a

ribonucleoprotein (RNP) complex, whereas TGB2 and TGB3 facilitate the RNP transport to the periphery of the cell and delivery to plasmodesmata. It is shown that PMTV RNA is transported either as RNP complex containing TGB1 or encapsidated in virions containing TGB1 and that nucleolar passage of TGB1 may be important for long-distance movement of both RNP complex and virions (Torrance et al. 2011). For furovirus *Chinese wheat mosaic virus* (CWMV), two putative transmembrane domains of the 37K movement protein are important for movement function and intracellular transport of the 37K protein (Andika et al. 2013).

In the case of BNYVV, RNA3 is responsible for the long-distance movement in *Beta* species (Lauber et al. 1998; Tamada et al. 1989) but not in *Nicotiana benthamiana* (Rahim et al. 2007). The RNA3 core sequence, termed 'coremin', is important for the long-distance movement (Ratti et al. 2009). For bymovirus *Barley yellow mosaic virus* (BaYMV), RNA2-encoded P1 protein is essential for systemic infection, whereas P2 protein facilitates virus systemic movement (You and Shirako 2010).

Vector transmission

The CP-RT portions of beny-, furo-, and pomoviruses, the p39 protein of pecluvirus, and the P2 protein of bymovirus (Fig. 1a) are believed to play an important role in the transmission process. The C-terminal portion of the RT, p39, or P2 proteins frequently undergoes deletions and this is correlated with loss of vector transmission. There is very little direct sequence similarity among these proteins but some structural similarity that might be involved in transmission (Dessens and Meyer 1996). Adams et al. (2001) showed that RT domains and P2 proteins but not p39 protein contain two transmembrane domains (T1 and T2). The region between T1 and T2 is predicted to be on the inside of the membrane and therefore the virus would initially be on the outside, suggesting that these conserved transmembrane regions are involved in attachment to the zoosporangial plasmodesmata and may assist virus particles to move between the cytoplasms of the plant host and the vector. The CP-RT proteins of BNYVV and PMTV are shown to be present at one extremity of the virus particles. A KTER motif in the BNYVV RT domain is required for vector transmission (Tamada et al. 1996), In the case of BNYVV and *Beet soil-borne mosaic virus* (BSBMV), the RNA4-encoded

protein is involved in efficient vector transmission (D'Alonzo et al. 2012; Rahim et al. 2007).

RNA silencing suppression

The CRPs of furoviruses (*Soil-borne wheat mosaic virus,* SBWMV, and CWMV), pecluvirus (*Peanut clump virus*, PCV), and benyviruses (BNYVV, BSBMV, and BdMoV) (Fig. 1a) have been shown to function as RNA silencing suppressors (Andika et al. 2012; Chiba et al. 2013; Dunoyer et al. 2002; Sun et al. 2013; Te et al. 2005). However, pomovirus PMTV p8 CRP does not have the suppression activity, but is identified as a pathogenicity factor (Lukhovitskaya et al. 2005). Thus far, CPRs were known to be involved in viral genome accumulation, efficient virus movement, or symptom modulation. Indeed, the C-terminal region of CWMV CPR is involved in symptom severity, whereas the N-terminal and central regions are important for silencing suppression (Sun et al. 2013). BNYVV p14 CRP accumulates in the nucleolus and the cytoplasm, and the suppression activity correlates with long-distance movement (Chiba et al. 2013). Meanwhile, the silencing suppression activity of the CRPs generally is lower in shoots, but the suppression of BNYVV CPR is more efficient in roots (Andika et al. 2012). In addition, it is known that the roots have less of RNA silencing activity (Andika et al. 2005).

Disease induction and resistance

BNYVV, BaYMV, and *Barley mild mosaic virus* (BaMMV, *Bymovirus*) have been well-studied on disease induction and resistance. For BNYVV, RNA3-encoded p25 protein determines rhizomania symptoms in sugar beet roots (Tamada et al. 1999). The p25 protein also functions as an avirulence factor, and this interaction is controlled by single amino acid changes in p25 protein (Acosta-Leal et al. 2008; Chiba et al. 2008). Mutational pathways to the emergence of new resistance-breaking p25 variant viruses have been suggested (Chiba et al. 2011). The p25 protein that has a nucleo-cytoplasmic shuttling activity (Vetter et al. 2004) is shown to target the sugar beet 26S proteasome involved in the induction of resistance response via interaction with an F-box protein

(Thiel et al. 2012). RNA5-encoded p26 is associated with symptom severity (Bornemann and Varrelmann 2013; Chiba et a. 2011).

For BaYMV and BaMMV, the virus-encoded VPg (Fig. 1a) is involved in the breaking of host resistance. The recessive resistance genes *rym4* and *rym5* encode barley eukaryotic translation initiation factor 4E (Hv-eIF4E). An interaction between VPg and eIF4E is shown to be implicated in the breaking of *rym4*- and *rym5*-mediated resistance (Kanyuka et al. 2005; Kühne et al. 2003; Nishigawa et al. 2008; Stein et al. 2005; You and Shirako 2013), similar to the interaction between viruses in the genus *Potyvirus* and their hosts (Ruffel et al. 2002).

Plasmodiophorid vectors

Taxonomic characteristic

P. graminis, P. betae, and *S. subterranea* are recognized as vectors of plant viruses. They have been classified in the order Plasmodiophorales and family *Plasmodiophoraceae,* and currently 41 species belonging to 12 genera are recognized (Neuhauser et al. 2010). They were previously considered to belong to the fungi, but are now included in the kingdom Protista (Braselton 2001). Three out of 12 genera, *Polymyxa, Spongospora,* and *Plasmodiophora,* are of significant agronomic importance. For example, *Plasmodiophora brassicae* causes the important clubroot disease of brassicaceous plants, and *S. subterranea* is the agent of powdery scab of potato and is also a virus vector. Phylogenetic analysis of ribosomal DNA (rDNA) suggests that *Polymyxa* are very closely related to *Ligniera* and *Sorosphaera,* while *Spongospora* and *Plasmodiophora* are more distantly related (Bulman et al. 2001; Ward and Adams 1998).

Life cycle

The life cycle of plasmodiophorids has two phases: the sporangial phase, producing secondary zoospores, and the sporogenic phase, producing primary zoospores via the formation of resting spores (Fig. 1b) (Braselton 1995). The penetration process of a

zoospore is as follows (Fig. 1c): encystment of the zoospore at the cell wall surface; development in the encysted zoospore of a tubular structure (Rohr) that contains a dense dagger-like body (Stachel); production of an adhesive outgrowth (adhesorium) from the encysted zoospore; and injection of the Stachel and zoospore contents through the adhesorium, host cell wall, and plasma membrane into the cytoplasm. During the sporangial phase, the nucleus undergoes non-cruciform mitotic-divisions and the plasmodium is cleaved, resulting in aggregates of zoosporangia. The zoosporangia develop exist tubes and secondary zoospores are released. During the sporogenic phase, the nucleus undergoes cruciform mitotic divisions and the sporogenic plasmodium is cleaved, resulting in aggregates of unicellular resting spores (sporosori or cystosori). Individual resting spores release a primary zoospore that initiates another round of infection (Fig. 1b). In *Polymyxa* species, secondary zoospores develop either sporangial or sporogenic phases (Fig. 1b). Factors that determine which stages to develop are unknown, and the two phases are present redundantly in root epidermal tissues.

Virus acquisition and transmission

It is considered that the virus acquisition and transmission are taking place either when zoospores penetrate the host cells and transfer the contents into the cytoplasm (Fig. 1c) or at the young plasmodium stage when a thin membrane boundary is between the plasmodiophorid and host cytoplasms (Fig.1b). The precise mechanism of the process is unknown. Generally, acquired viruses are thought to be carried inside the resting spores and zoospores, but attempts to detect virus particles in the resting spores have been unsuccessful. However, Driskel et al. (2004) observed that for furovirus SBWMV, viral RNA and MP but not CP were detected in resting spores, suggesting that *P. graminis* might not transmit SBWMV particles to host cells, but an RNP complex consisting of MP and RNA. In the case of bymovirus *Wheat spindle streak mosaic virus* (WSSMV), however, CP was detected in resting spores. Meanwhile, BNYVV proteins involved in virus replication and movement, although CP and CP-RT were less detectable, were detected within zoosporangia and resting spores of *P. betae*, which suggests that viral translation may occur within the vector (Lubicz et al. 2007).

Variation

P. graminis and *P. betae* are morphologically indistinguishable, and the two species are originally separated by host range (Barr 1979; Barr and Asher 1992; Braselton 2001). *P. graminis* primary multiplies in grass and cereal species in the *Gramineae,* whereas *P. betae* is a parasite of species in the *Chenopodiaceae* and some related plants. Two *Polymyxa* species can be clearly distinguished by the rDNA analysis (Ward and Adams 1998).

There is a remarkable variation in the host specificity of *P. graminis* isolates from various origins (Table 2). Differences in susceptibility and multiplication rate on infected plants are observed between various isolates from distinct plants adapted to specific climate regions, from a similar plant but originating from distinct areas, and from distinct plants in the same country (Adams and Swaby 1988; Legrève et al. 1998). For examples, *P. graminis* isolates from temperate regions can grow well at 17–20 °C, whereas *P. graminis* isolates from tropical climate show more aggressiveness and have a higher temperature optimum of 27–30 °C. On the basis of the ecological characteristics and rDNA analysis, five special forms are proposed as shown in Table 2 (Legrève et al. 2002).

The host range of *P. betae* is restricted to species of *Chenopodiaceae* and a few related species. However, populations of *P. betae* from a single field soil are heterogenous, with individual isolates showing significant variability in host specificity. For examples, isolates from certain plant species may not infect plants from other families, or even other plants within the same family (Abe and Ui 1986; Barr 1979; Barr and Asher 1992). Based on such host range differences, three formae speciales are proposed; *P. betae* f. sp. *betae*, *P. betae* f. sp. *amaranthi* and *P. betae* f. sp. *portulacae* (Table 2). *P. betae* f. sp. *betae* is only a vector of BNYVV, but other forms are not (Abe and Tamada 1986).

S. subterranea occurs as two distinct pathogenic strains. One strain (*S. subterranea* f. sp. *subterranea*) causes the powdery scab disease of potatoes and is a vector of PMTV. The other strain (*S. subterranea* f. sp. *nasturtii*) causes watercress crook root disease and transmits WCLSV (Tomlinson and Hunt 1987) and WYSV (Walsh et al. 1989). Two strains are morphologically indistinguishable, but the potato strain does not infect

watercress and conversely, the watercress strain does not infect potato and tomato (Tomlinson 1958). Two forms (I and II) of *S. subterranea* f. sp. *subterranea* are proposed based on the rDNA analysis (Qu and Christ 2004). These are geographically different, but their biological differences are not known.

Geographical distribution, variation, and genetic resistance

Barley yellow mosaic disease caused by BaYMV and BaMMV

A yellow mosaic disease of barley was first reported in 1940 in Japan (Ikata and Kawai 1940; Inouye and Saito 1975) and subsequently became one of the major diseases of Japanese two-rowed malting barley. There is a considerable variation among barley varieties in symptoms and resistance, and numerous stocks were found to be resistant or immune to BaYMV (Takahashi 1983). In China, BaYMV was first found in the 1950s and caused serious losses in the mid-1970s. As in Japan, barley sources with high levels of resistance have been employed in breeding programs (Chen et al. 1992). BaYMV was reported in South Korea (Lee et al. 2006).

In Europe, BaYMV was first found in Western Germany in 1978 and later in many other countries (Kühne 2009). It is noteworthy that BaYMV rapidly became widespread over large areas, particularly in Germany and the UK. In addition to BaYMV, a second virus named BaMMV, was reported to induce the yellow mosaic disease in winter barley in Europe (Huth and Adams 1990). BaMMV was also detected in Japan (Nomura et al. 1996).

In Japan, eight strains in five pathological groups (I to V) of BaYMV have been identified based on the response of differential cultivars, and named pathotypes I-1, I-2, I-3, II-1, II-2, III, IV, and V (Table 3) (Kashiwazaki et al. 1989; Okada et al. 2004; Sotome et al. 2010). In Europe, BaYMV strains are distinguished based of the infectivity of *rym4* and *rym5* cultivars, and the resistance-breaking virus strain, named BaYMV-2, is becoming increasingly important (Kühne 2009). Several strains probably occur in China but these are less well defined (Chen et al. 1996). The phylogenetic analysis (Nishigawa et al. 2008) showed that Japanese strain I is most closely related to

the Chinese isolate, and these two strains form one cluster with European isolates, whereas Japanese strains II, III, and IV, and the Korean isolate form another cluster. Two BaMMV strains have been identified based on the infectivity of *rym5* cultivars in Japan and in Europe (Table 3) (Hariri et al. 2003; Nomura et al. 1996).

So far, 15 recessive *rym* genes and three dominant *Rym* genes have been identified in the germplasms of *Hordeum vulgare* or *H. bulbosum* genotypes (Kai et al. 2012; Kühne 2009; Werner et al. 2003). These resistance genes are localized on chromosomes 1H (*rym7*), 2H (*Rym16*^{Hb}), 3H (*rym4, 5, 6, 10,* and *Rym17*), 4H (*rym1, 8, 9, 11, 12*, *13,* and *18*), 5H (*rym3*), 6H (*rym15* and *Rym14*^{Hb}), and 7H (*rym2*). The *rym1–rym6* genes have been used as sources for BaYMV-resistant cultivars (Kühne 2009; Ordon et al. 2005).

Wheat yellow mosaic disease caused by *Wheat yellow mosaic virus* (WYMV) and WSSMV

A yellow mosaic disease of wheat was first reported in Japan in the 1920s (Sawada 1927) and the agent was identified as WYMV (Inouye 1969). Since then, the disease has been widely distributed in western Japan. However, the disease was sporadically found in northern Japan (Tohoku area) in the end 1980s (Ohto 2005) and in Hokkaido in the 1990s (Kusume et al. 1997). The similar disease was found in China, where WYMV has been widely spread (Chen 1993; Chen et al. 1999; Han et al. 2000). Another wheat-infecting bymovirus is WSSMV that was first reported in Canada (Slykhuis 1960) and about a decade later in the US, where it rapidly became widespread across the country. In Europe, WSSMV has been recorded in France, Italy, Germany, and Belgium (Kühne 2009). It was also observed in Africa (Zambia) and India.

Ohto (2006) showed that Japanese WYMV isolates are grouped into three strains (called types) based on the infectivity to different wheat cultivars (Table 3). The distribution of these type strains was geographically different. In China, two geographically different strains were reported (Chen et al. 2000).

A number of wheat varieties with the higher level of resistance to both WSSMV and WYMV have been developed and available. Inheritance of resistance to the viruses is

complex and influenced by many factors, and resistance in wheat seems to be controlled by one to three genes. Resistance genes for WYMV and WSSMV have been mapped to wheat chromosome 2D in the US cultivar 'Geneva' (Khan et al. 2000) and in the Chinese cultivar 'Yangfu 9311' (Liu et al. 2005b), respectively. The European cultivar 'Ibis' was found to have excellent WYMV resistance in Japan, and this resistance gene was also mapped in chromosome 2D (Nishio et al. 2010).

Wheat mosaic disease caused by SBWMV, SBCMV, and CWMV

A mosaic disease of winter wheat was first found in Illinois and Indiana in 1919, and the casual agent was named SBWMV (McKinney 1923). Similar disease of wheat was observed in Japan in the 1920s (Ikata and Kawai 1940; Sawada 1927). It has been found in China since the early 1970s (Chen 1993). In Europe, SBWMV-like disease was first recorded in Italy in the 1960s and later in France, the UK, Germany, Turkey, and Belgium (Kühne 2009). The disease was reported from Brazil, Africa (Zambia), South Korea, and New Zealand (Kühne 2009).

Thus, wheat-infecting furoviruses from the different geographic regions induce the same or similar type of symptoms in wheat plants as original SBWMV from the US. However, sequence analyses of these virus genomes showed clear sequence differences (>70%) among isolates occurring in the US, Asia, and Europe (Diao et al. 1999; Shirako et al. 2000). Based on the guidelines of proposal from classification, therefore, the viruses from China and Europe were classified as separate species and designated CWMV and SBCMV, respectively (Torrance and Koenig 2005). On these bases, Japanese isolate SBWMV-JT also may represent an additional species (as listed in Table 1). Thus, there are at least four species in wheat-infecting furoviruses. The cases of such separated species may lead to more complexity and confusion from a practical point, when new virus species introduced in area where one virus species is present or when mixed infection occurred between the species in field. In fact, SBWMV was recently found in Germany (Kühne 2009) and in Hokkaido (Shirako et al. 2012), and an isolate (SBWMV-Mar) from France is very closely related to Japanese SBWMV-JT (Hariri and Meyer 2007) (Table 1). Furthermore, the fact that recombinant viruses can be formed between some of these viruses (Miyanishi et al. 2002) suggests that they

could be regarded as strains of SBWMV. Thus, the classification of species of wheat-infecting furoviruses remains as a matter of debate (Hao et al. 2012; Kühne 2009).

In the US, Japan, Europe, Brazil, and China, resistant cultivars that are adapted to respective countries have been developed and cultivated. There are many studies on the inheritance of virus resistance in wheat, but the results are not consistent. Two major resistances genes *Sbm1* and *Sbm2* for SBCMV are mapped on chromosomes 5DL and 2BS, respectively (Bass et al. 2006; Maccaferri et al. 2011). The *Sbm1* gene for SBWMV was also shown to be located to the same region on 5DL (Hao et al. 2012).

Resistance to furoviruses in wheat is thought to be due to a block on viral translocation from roots to shoots. Interestingly, it is suggested that the mechanism of resistance to SBCMV involves the efficient disassembly of virus particles and either an inhibition of further synthesis of viral CP or its proteolytic degradation (Lyons et al. 2009).

Sugar beet rhizomania caused by BNYVV

Rhizomania of sugar beet was first recorded in northern Italy in the early 1950s (Canova 1959) and the causal agent BNYVV was identified in Japan in 1973 (Tamada and Baba 1973). During a few decades, BNYVV has been spread throughout Europe and Middle East (Asher 1993; McGrann et al. 2009). It was first found in Japan in 1965, in China in 1978, and in the US in 1983 (Rush et al. 2006; Tamada 1999). In almost all areas, BNYVV has spread rapidly and widely.

BNYVV isolates were classified into two types, A and B (Kruse et al. 1994). A further group, P-type, that additionally contained an RNA5 molecule was isolated from France, but was closely related to the A-type (Miyanishi et al. 1999). A-type virus is distributed more worldwide, while the B-type virus is found in limited areas of Europe. Chiba et al. (2011) analyzed five genes from 73 isolates collected throughout the world and showed that worldwide BNYVV isolates consist of eight strains (Table 3) that derived from at least four original lineages (A-I, A-II, A-III, and B types). These strains are clearly different geographically. For examples, Italy strain isolates (from A-III type source)

were first found in Italy in the early 1950s, and have spread in Europe, to the Middle East and to the US during three decades. Similarly, Germany strain isolates (from B type source) have found in limited areas of Germany and France in the early 1970s, and have then spread in Europe. These two strains are free from RNA5. Other six strains, Japan-D, Japan-O, China-B, France-P (=P-type), China-H, and China-X, which are originally isolated from China, Japan, and France have been found in limited areas with a few exceptions. Most isolates of the six strains contain RNA5.

Chiba et al. (2011) also suggests that these four original lineages and their progeny strains probably existed in native hosts in East Asia long before sugar beet was cultivated. For the last half a century, BNYVV sources with diverse origins have introduced infection to cultivated sugar beet plants in different areas during different periods and have spread extensively.

Since rhizomania resistance tests had begun in northern Italy in the mid-1960s (Biancardi et al. 2002), the first resistant cultivar 'Rizor' was introduced in 1985 (Asher 1993). Subsequently, the 'Holly' resistance gene (*Rz1*) with higher levels of resistance, has been incorporated in many current cultivars. Cultivars possessing other resistance genes, such as *Rz2, Rz3, Rz4,* and *Rz5,* have been produced (Grimmer et al. 2007; McGrann et al. 2009). These resistant genes have been mapped at two distinct loci on chromosome III, in which the first locus was represented by alleles *Rz1*, *Rz4,* and *Rz5* and the second by alleles *Rz2* and *Rz3*.

Severe symptoms have been found in *Rz1*-resistant cultivars in some areas of the US (Liu et al. 2005a; Rush et al. 2006). Indeed, resistance-breaking variant viruses in the Italy A-III type strain are shown to be generated by amino acid changes at positions 67 and 68 in the p25 protein (Acosta-Leal et al. 2008; Chiba et al. 2008, 2011). Newly generated p25 variant viruses are more advantageous in resistant sugar beet cultivars than previous virus isolates, but these variant viruses probably coexist as quasispecies in the field.

Potato tuber spraing disease caused by PMTV

A spraing disease of potato caused by PMTV was first reported in Northern Ireland, England, and Scotland (Calvert and Harrison 1966). In Scandinavia, PMTV was detected in Norway and Finland in the 1960s and the mid-1970s, respectively (Latvala-Kilby et al. 2009). PMTV occurs also in South America, Canada, and recently the US (Xu et al. 2004). The likely origin of PMTV is considered to be the Andean region of South America. In Japan, PMTV was first recorded in Hiroshima in 1980 (Imoto et al. 1986). Since then, there were no record of PMTV in Japan, but in 2005, the spraing symptom was found on potato cultivar 'Sayaka' in Hokkaido (Maoka et al. 2006). Maoka et al. (2011) suggests that PMTV is widely present throughout Japan, but soil infestations with PMTV are not always associated with spraing symptoms.

The genetic variability of worldwide PMTV isolates is so far only limited, but two types based on different sequences of RNA2 (CP and RT) and RNA3 (p8 CRP) are found (Table 3), each showing only little genetic variability (Latvala-Kilby et al. 2009). However, two distinguishable variants in RNA2 and RNA3 occur in different combinations and mixed infections in fields in Finland (Latvala-Kilby et al. 2009). The biological differences of these variants are not known.

In the Nordic countries, potato cultivars with higher level of resistance to PMTV are not available, but significant differences in cultivars are observed in development of spraing symptoms (Sandgren 1995; Sandgren et al. 2002). PMTV is rarely found to spread from underground parts to the foliage of plants in these areas. Likewise, variations of the symptom degrees are observed among Japanese potato varieties (Nakayama et al. 2010). No foliage symptoms are observed in Japan.

Peanut clump disease caused by PCV and *Indian peanut clump virus* (IPCV)

A clump disease of peanut is caused by PCV in West Africa (Thouvenel et al. 1976) and by IPCV in Pakistan and the Indian sub-continent (Reddy et al. 1983). These viruses have been known since the 1920s. Five and three serotypes in PCV and IPCV, respectively, were reported (Table 3) (Manohar et al. 1995; Nolt et al. 1988), suggesting that there is a large variability among isolates of either PCV or IPCV. Phylogenetic

analyses showed clusters usually grouped according to their geographical origin (Dieryck et al. 2009; Naidu et al. 2003).

PCV and IPCV are transmitted by seeds. This is quite unusual for plasmodiophorid-transmitted viruses. Natural hosts that were first reported are groundnut, but PCV and IPCV can infect a number of cereal crops and graminaceous weeds. PCV and IPCV are transmitted by two formae speciales, *P. graminis* f. sp. *tropicalis* and *P. graminis* f. sp. *subtropicalis* (Table 2) (Dieryck et al. 2011). These vectors produce heavy infestation in the roots of pearl millets, sorghum, and sugarcane (Legrève et al. 2000), which indicates that these graminaceous plants are well adapted to the vectors. In contrast, groundnut that is a poor host for the vectors does not support such an increase. Thus, PCV and IPCV seem not to be viruses of groundnut, so that they could be regarded as cereal viruses that infect groundnut opportunistically (Dieryck et al. 2009).

Concluding comments

Plasmodiophorid-transmitted viruses are genetically very diverse. However, these viruses have common elements that are the CP-RT proteins (furo-, pomo-, and benyviruses) and the analogues (p39 of pecluviruses and P2 of bymoviruses) (Fig. 1a). For BNYVV and PMTV, the CP-RT protein that is expressed as a minor protein is shown to be present at one extremity of the virus particles. Such proteins are strongly implicated in vector transmission. Interestingly, these proteins except for p39 contain structurally two complementary transmembrane domains (Adams et al. 2001), suggesting that these conserved transmembrane regions may be involved in attachment to the zoosporangial plasmodesmata between the cytoplasm of the plant host and the vector.

Local and systemic movement strategies of plasmodiophorid-transmitted viruses are greatly diverse. Generally, the CP is not required for cell-to-cell movement of the virus, but it is essential for long-distance movement except for pomoviruses (Torrance et al. 2011). It is suggested that viruses move in the form of an RNP complex, in which the TGB1 protein plays an important role in both viral cell-to-cell and systemic movement.

Interesting, in the case of SBWMV, the RNP complex consisting of MP and RNA but not virions was detected in resting spores of *P. graminis* (Driskel et al. 2004). Furthermore, for BNYVV, viral proteins involved in virus replication and movement were detected within zoosporangia and resting spores of *P. betae* (Lubicz et al. 2007). These results suggest that viral translation and movement may occur within the vector. Further studies will need to examine whether the plasmodiophorid is actually a host for viruses or simply a vector for virus transmission.

Plant virus vectors are only three species out of 42 species in the order *Plasmodiophorids,* some species of which are known as parasites of aquatic angiosperms or brown algae. These organisms are known to be infected with marine viruses with ssRNA genomes, in which the virus is transmitted via water on lysis of the host cell (Lang et al. 2009). Neuhauser et al. (2011) have discussed the possibility of plasmodiophorids as vectors to enter the host organism through thick cell walls. There are no direct relationships between such marine viruses and plasmodiophorid-transmitted plant viruses. However, the finding of *Chara australis* virus (CAV) isolated from a fresh-water green alga is quite interesting, because CAV has rod-shaped particles similar to tobamoviruses and its polymerase is most closely related to that of benyviruses (Gibbs et al. 2011).

There are intriguing and important relationships between *Polymyxa*-transmitted viruses and their host plants (e.g., wheat and three furoviruses or two bymoviruses, barley and two bymoviruses, and cereals and two pecluviruses). Each of these viruses in the genus induces similar symptoms in each host plant, but there are sequence differences (>70%) and geographical differences. Considering the wide diversity in genomes among species, it can be speculated that they have evolved independently in different countries. Thus, populations of these virus species existed already on indigenous host plants, such as perennial grass species, in which they have coevolved and to which they are well-adapted. However, when susceptible plant species or varieties are introduced or when virus-carrying plasmodiophorids were introduced to maiden land, they may cause severe damage to susceptible plant species. Good examples are pecluviruses and BNYVV. Groundnut that is a native of South America was introduced to Africa and Asia, where original pecluviruses had already existed in native grass plants. Also,

BNYVV-carrying *P. betae* that may have root in East Asia, although the native hosts are unknown, was introduced to Europe, where sugar beet had been grown. Thus, it is considered that groundnut and sugar beet became "new-encounter" hosts for each virus.

During the last half a century, it is clear that most of plasmodiophorid-transmitted viruses have spread more widely, and some viruses have migrated worldwide (e. g., BNYVV, BaYMV, and PMTV). Along with introduction of viruses to new areas and successive cultivation of resistant cultivars, it will be easy to emerge new resistance-breaking variant viruses by amino acid changes in pathogenicity-related viral genomes. Furthermore, accumulation of inoculum sources containing virus-carrying plasmodiophorids will increase the possibility to emerge the new viruses or variant viruses by recombination or reaassortants of viral genomes. Indeed, mixed infections of viruses in cereal crops are usually observed between different genera or between different species or strains (Kanyuka et al. 2003; Kühne 2009).

Furthermore, wheat, barley, and sugar beet are grown as agriculture crops in extremely divers climatic regions in the world. These crops are often grown in rotation in the same fields (Rush 2003). Isolates of *P. graminis* from various geographical loci around the world have shown considerable diversity in specific ecological and biological characteristics. *P. betae* has not shown the same degree of diversity, but there are some pathotypes (Abe and Ui 1986). This situation will provide an increasing opportunity to evaluate variability within populations of *P. betae* and *P. graminis* (Rush 2003; Smith et al. 2012). Ecological and biological studies on the plasmodiophorid vector are required further.

Finally, plasmodiophorid-transmitted viruses as well as other soil-borne viruses could complete their lifecycle in the underground parts of plants. Systemic movement of virus to shoots is rare, as seen in BNYVV and PMTV. Unique properties of CP, CP-RT, MP, and CRP suppressor that are encoded by the virus are thought to be strongly implicated in inefficiency of the vascular movement (Andika et al. 2005, 2012; Torrance 2011). Further studies are required to clarify the root-specific movement mechanism, associated with vector transmission. In many cases as well as in the case of virus-infested fields that resistant cultivars are grown, the viruses are restricted in roots

without any symptom in the aerial parts, in which the damage by viruses to plants may appear be no or small. Thus, so-called ‘opportunistic infection’ possibly occurs in roots. Therefore, the diagnosis and detection of the virus or virus-carrying vector from plant roots or soils are very important; thereby, ease-to-use, purpose-adapted, sensitive molecular techniques are required to be developed and employed.

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Figures

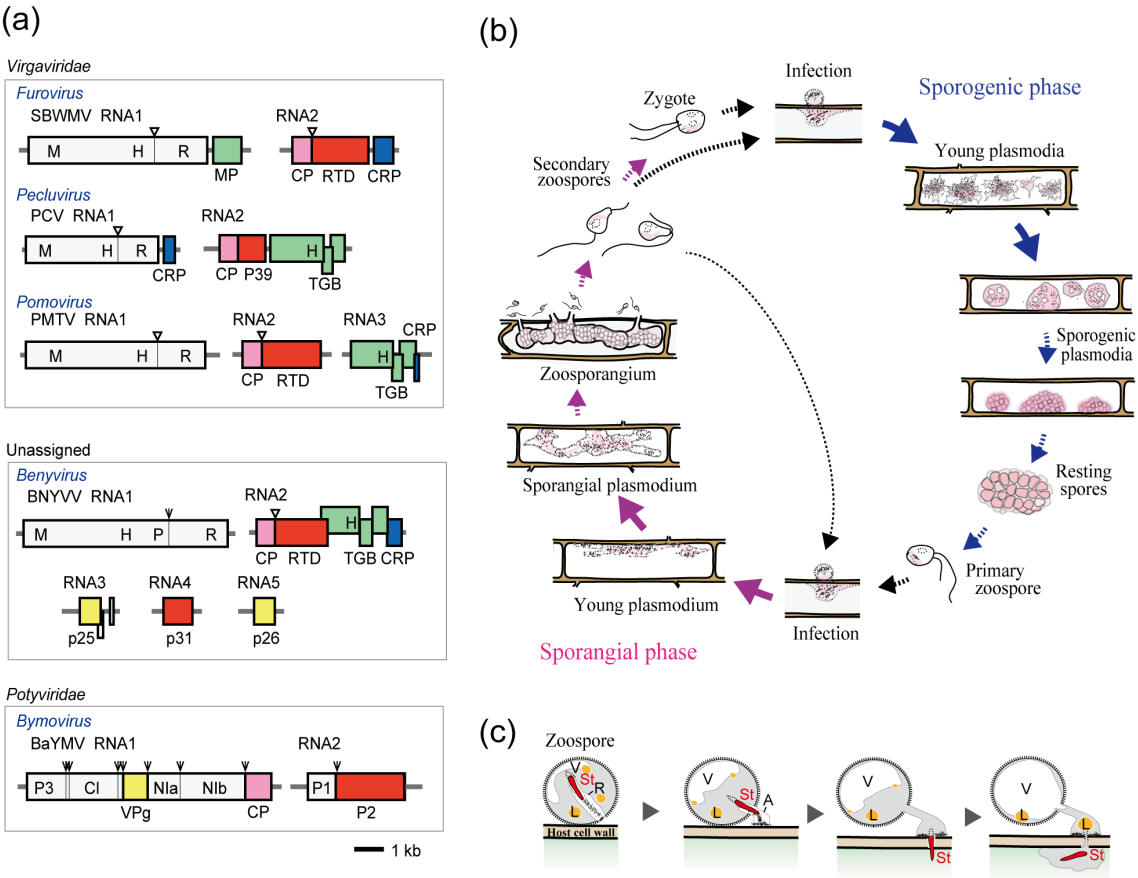

Fig.1

Fig. 1. (a) The genome structure of five genera including the plasmodiophorid-transmitted viruses. Type members are shown; SBWMV (*Soil-borne wheat mosaic virus*), PCV (*Peanut clump virus*), PMTV (*Potato mop-top virus*) BNYVV (*Beet necrotic yellow vein virus*), BaYMV (*Barley yellow mosaic virus*). Marks in domains are as follows; M=methyltransferase domain, H=helicase domain, R=RNA-dependent RNA polymerase domain, MP=movement protein, CP=coat protein, RTD=readthrough domain, CRP=cysteine-rich protein, TGB=triple gene block proteins, P=protease, CI=cytoplasmic inclusion protein, VPg=genome-linked protein, NIa=nuclear inclusion protein a-proteinase, NIb=nuclear inclusion protein b (including RNA-dependent RNA polymerase), P1=cysteine proteinase, and P2=putative vector-transmission factor. The triangles, asterisk, and arrows indicate translation readthrough sites, leaky scanning site, and protease cleavage sites, respectively. (b)

Diagram of the life cycle of *Polymyxa* species, which consists of sporangial and sporogenic phases. Solid directional lines indicate the process important for virus acquisition and transmission. (c) Diagram of zoospore encystment and penetration of root cells. Re-drawn from Kanyuka et al. (2003). St=Stachel, R=Rohr, A=adhesorium, V=vacuole, and L=lipid droplet.

Table 1 Plant viruses transmitted by plasmodiophorids

Family Genus	Virus species[a]	(Acronym)	Vector	Natural host	Geographical distribution
Virgaviridae					
Furovirus	*Chinese wheat mosaic virus*	(CWMV)	*P. graminis*	Wheat	China
	Oat golden stripe virus	(OGSV)	*P. graminis*	Oat	Europe, US
	Soil-borne cereal mosaic virus	(SBCMV)	*P. graminis*	Wheat, rye, triticale	Europe
	Soil-borne wheat mosaic virus	(SBWMV)	*P. graminis*	Wheat, barley, rye, triticale	US, Germany, Brazil, Africa, Japan
	Soil-borne wheat mosaic virus-JT [b]	(SBWMV-JT)	*P. graminis*	Wheat, barley	Japan, France
	Sorghum chlorotic spot virus	(SrCSV)	*P. graminis*	Sorghum	US
Pecluvirus	*Peanut clump virus*	(PCV)	*P. graminis*	Peanut, sorghum, cereals	West Africa
	Indian peanut clump virus	(IPCV)	*P. graminis*	Peanut, sorghum, cereals	India, Pakistan
Pomovirus	*Beet soil-borne virus*	(BSBV)	*P. betae*	Sugar beet	Worldwide?
	Beet virus Q	(BVQ)	*P. betae*	Sugar beet	Europe, Worldwide?
	Broad bean necrosis virus	(BBNV)	Unknown	Broad bean	Japan
	Potato mop-top virus	(PMTV)	*S. subterranea*	Potato	Europe, North and South America, Japan
Unassigned family					
Benyvirus	*Beet necrotic yellow vein virus*	(BNYVV)	*P. betae*	Sugar beet, spinach	Worldwide
	Beet soil-borne mosaic virus	(BSBMV)	*P. betae*	Sugar beet	US
	Rice stripe necrosis virus	(RSNV)	*P. graminis*	Rice	West Africa, South and Central America
	Burdock mottle virus	(BdMoV)	Unknown	Burdock	Japan
Potyviridae					
Bymovirus	*Barley mild mosaic virus*	(BaMMV)	*P. graminis*	Barley	Europe, Japan, China, Korea
	Barley yellow mosaic virus	(BaYMV)	*P. graminis*	Barley	Europe, Japan, China, Korea
	Oat mosaic virus	(OMV)	*P. graminis*	Oats	Europe, US
	Rice necrosis mosaic virus	(RNMV)	*P. graminis*	Rice	Japan, India
	Wheat spindle streak mosaic virus	(WSSMV)	*P. graminis*	Wheat, rye, triticale	North America, Europe, Africa
	Wheat yellow mosaic virus	(WYMV)	*P. graminis*	Wheat	Japan, China
Unclassified viruses					
	Aubian wheat mosaic virus	(AWMV)	*P. graminis*	Wheat	France, UK
	Watercress yellow spot virus	(WYSV)	*S. subterranea*	Watercress	UK
	Watercress chlorotic leaf spot virus	(WCLSV)	*S. subterranea*	Watercress	UK

[a] Formally accepted virus species appear in italics, and tentative species are in the regular font.

[b] SBWMV-JT (Japan) was distinguished from SBWMV (US), because they shared 68 to 82% amino acid sequence identity (Shirako et al. 2000).

Table 2 Subgroups of *Polymyxa graminis*, *Polymyxa betae*, and *Spongospora subterranea*

Species	Forma specialis (rRNA subgroup)		Natural host	Optimum temperature	Reference
P. graminis	*temperate*	(I)	Barley, *Poa* sp.	15–20 ˚C	Legrève et al. (2002), Ward and Adams (1998)
	tepida	(II)	Barley, wheat, oat, rye	15–20 ˚C	Legrève et al. (2002), Ward and Adams (1998)
	tropicalis	(III)	Sorghum, peal, millet, maize	>23 ˚C	Legrève et al. (2002)
	subtropicalis	(IV)	Sorghum, pearl, millet	>23 ˚C	Legrève et al. (2002)
	colombiana	(V)	Rice	>23 ˚C	Legrève et al. (2002), Morales et al. (1999)
P. betae	*betae*		Sugar beet, Chenopodiaceae	20–25 ˚C	Barr (1979)
	amaranthi		*Amaranthus retroflexus*	20–25 ˚C	Barr (1979), Abe and Ui (1986)
	portulacae		*Portulaca oleracea, P. grandiflora*	20–25 ˚C	Abe and Ui (1986)
S. subterranea	*subterranea*	(I)	Potato	15–20 ˚C	Qu and Christ (2004)
	subterranea	(II)	Potato	15–20 ˚C	Qu and Christ (2004)
	nasturtii		Watercress	15–20 ˚C	Tomlinson (1958)

Table 3 Variability of plasmodiophorid-transmitted viruses causing agriculturally important diseases

Genus	Virus	Plant	No. of types, strains, or pathotypes [a]	Resistance breaking (host gene)	Viral factor	Reference
Bymovirus	BaYMV	Barley	8 (pathogenicity)	Yes (*rym4*)	VPg	Kashiwazaki et al. (1998), Sotome et al (2010), Kühne et al. (2003)
	BaMMV	Barley	2 (pathogenicity)	Yes (*rym5*)	VPg	Nomura et al. (1996), Hariri et al. (2003), Kanyuka et al. (2004)
	WYMV	Wheat	3 (pathogenicity)	Yes (unknown)	Unknown	Ohto (2006)
Benyvirus	BNYVV	Sugar beet	8 (phylogeny) [b]	Yes (*Rz1*)	P25, RNA5	Chiba et al. (2008, 2011)
Pomovirus	PMTV	Potato	2 (phylogeny)	Unknown	Unknown	Latvala-Kilby et al. (2009)
Pecluvirus	PCV	Groundnut	5 (serology, phylogeny)	Unknown	Unknown	Manohar et al. (1995), Dieryck et al. (2009)
	IPCV	Groundnut	3 (serology, phylogeny)	Unknown	Unknown	Nolt et al. (1988), Naidu et al. (2003)

[a] Numbers of variations are based on pathogenicity (infectivity to cultivars), phylogeny (partial genome sequence), or serology.
[b] Several p25 variants within the Italy strain isolates are identified (Chiba et al. 2011)