Transmission and Movement of Plant Viruses

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Abstract

Plant viruses are obligate parasites and their survival depend on being able to spread from one susceptible organism to another. Viruses cannot penetrate the intact plant cuticle and the cellulose cell wall. Therefore penetration is made trough wounds in the surface layers, such as in mechanical inoculation and transmission by vectors. There is specificity in the mechanism by which the plant viruses are naturally transmitted. They are important economically only if they can spread from plant to plant rapidly. They are contagious agents that differ in their transmissibility. No transmission of virus occurred when the virus titer in the inoculum was too low and there is no susceptibility between virus, vector, and host. Also the presence of some substances in the inoculum, which inhibited the infection process, hampered the transmission of viruses. Knowledge of the ways in which plant viruses spread is essential for the development of control measures.

Keywords

Transmission • Plant viruses

2.1 Introduction

Plant viruses must go through two stages during their infection cycle. First, they must replicate inside host cells, employing cellular systems; they have to move to adjacent cells (short-distance movement) and, through the vascular system, reach other tissues and organs (long-distance movement). Second, viruses must spread to new hosts; to do that, they have to cross cellular barriers to enter cells. For most plant viruses this process is assisted by vector organisms (Matthews 1991). Transmission from plant to plant is an essential process for virus survival. Plant viruses have developed several strategies to perform this task efficiently, in many cases involving the existence of specific viral gene

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products known to facilitate the transmission process (Hull 1994; Gray 1996; Van den Heuvel et al. 1999).

Plant viruses are transmitted in different ways in nature-mechanical transmission, through the soil, by grafting, by planting material, through pollen, by seed, and by animal and vegetable vectors.

2.2 Mechanical and Contact Transmission

Mechanical transmission is when the viral particles contained in the juice of the diseased plant penetrate trough fresh wounds and infect the living cells of the healthy plant. Shortly after injury the cells die and cannot be the starting points for the penetration of viruses in plants. Most often, these wounds are obtained by touch and rubbing the leaves and stems of diseased plants that grow nearby. Not all viruses, however, infect in this way, but only highly infectious as TMV and PVX. Mechanical infections in tobacco, tomatoes, and other plants whose leaves are covered with plant trichomes are frequent. Touching the trichomes they break and juice of diseased plants is mixed with the juice of the healthy. For this contributes planting, breaking off sprouts, wringing, and other operations by which only one sick plant can contaminate the hands of the workers and the tools and infect many other healthy plants.

Grafting is an old established method to propagate the plants vegetatively. This is the easy way of transmission of virus from the scion or bud to rootstock through sap. The effectiveness of inoculation of sap-transmissible viruses can be increased by dusting the leaves by fine carborundum powder prior to inoculation (Rawlins and Tompkins 1936). The reported sap-transmitted virus includes cucumber mosaic virus (CMV), tobacco mosaic virus (TMV), potato virus X (PVX), and some geminiviruses. It implies direct transfer of sap from wounded plant to healthy plant on tools, hands, clothes, or machinery. PVX and Pepino mosaic virus can easily be spread by farm implements. The ability of these viruses to be spread by sap in the field is due to their extreme stability.

2.3 Transmission by Soil, Drainage, and River Water

By its nature, transmission by soil is also a mechanical transmission in which are grown diseased plants. In this case the source of infection is the remains of diseased plants in which certain viruses such as TMV, cucumber green mottle mosaic virus (CGMMV), and PVX retain their infectivity continuously. In very rare cases, viruses released from the roots of diseased plants and adsorbed to soil particles cause infections. (Smith et al. 1969). Highly infectious viruses in tomatoes – tomato mosaic virus (ToMV) and cucumber (CGMMV) – are being widely disseminated in greenhouses where the plants are grown hydroponic. In this case the nutrient solution acts as a carrier of these viruses. Viruses are isolated from the rivers passing through major cities such as the Thames, which flow into the city's canals (Tomlinson et al. 1982, 1984).

2.4 Transmission by Grafting

The safest way to transmit viruses is through the tissues from diseased to healthy plants. In vegetatively propagated crops, this transfer plays a big role because through it people transmit those viruses that do not carry mechanically or by vectors. Typically, in order to ensure the infection is necessary to obtain the bond between the graft and the substrate. Transmission by grafting is practiced for identifying viruses that infect not mechanically or were transmitted hardly by juice. In the natural conditions infections by grafting are possible not only in the vegetative propagation of plants, but spontaneously – in coalescence of roots of growing adjacent sick and healthy plants. They are particularly important for viruses that are found primarily in the roots, as in the prune dwarf virus in peach. Transmission by grafting is typical for *Potyviruses* like plum pox virus (PPV) in plums and PVY in pepper and tomato.

2.5 Transmission by Planting Material

With few exceptions, the viruses are in varying concentrations in nearly all tissues of the diseased plants. Therefore planting material obtained from such plants as cuttings, seedlings, buds, tubers, bulbs, etc., carries viral infection. That is why this mode of transmission and spread of viruses is essential in vegetatively propagating crops such as fruits, vine, berries, hops, potatoes, bulbs, and flowers. Regular transmission of the viruses in the generation of vegetatively propagated crops leads to the so-called degeneration. Plants received from infected propagating material are source of infection for neighboring plants. Thus from generation to generation the percentage of diseased plants is increasing and the yield is decreasing. This degeneration is quickly and typically for crops with a short growing season, such as potatoes. Therefore identification of potato viruses (PVY, PLRV, PVM, and PVS) in time is in great importance to stop the spread of disease and degeneration of potato cultivar. More often for 2–3 years, diseased tubers reach 100 %, so that its further cultivation is unprofitable and inappropriate.

2.6 Transmission Through Pollen

The virus transmitted by pollen may infect the seed and the seedlings which grow from it or it can also infect the plant through the fertilized flower. The pollen transmission is known to occur mainly in fruit trees like sour cherry. The ILAR (PPV, prune dwarf virus, prunus necrotic spot virus) viruses are known to be transmitted through pollen.

2.7 Transmission Through Seeds

Viruses that are transmitted trough seeds have some common properties. Most of them are mechanically transmitted easily; in infected plants mainly produce symptoms of mosaic and necrosis due to changes in the parenchymatous tissue. Aphids carry viruses transmissible through seeds but these are mainly nonpersistent viruses. Most cases of transmission of viruses trough seeds was by nematodes. Especially easy they carry the seed of annual weeds (Lister and Murant 1967).

Many important virus diseases are known to be transmitted by seeds. Bean common mosaic virus (BCMV) and CMV were among the first reported to be transmitted through seeds (Reddick and Stewart 1919). Pea seed-borne mosaic virus has been dispersed throughout the world in infected seeds. Seed-borne virus transmission involves virus-host interaction, a floral-infection stage, and the influence of the environment. Infection of an embryo with a virus is the most important factor of plant virus transmission through seed. TMV is a very stable virus that remains infectious on the surface of the seed coat. During germination or planting, seedlings get infected with TMV as a result of mechanical infection (Taylor et al. 1961; Broadbent 1965). Southern bean mosaic virus is found in the seed coat. The transmission frequency is, however, very low, and the virus is inactivated during the process of seed transmission (Crowley 1959; McDonald and Hamilton; 1972; Uyemoto and Grogan 1977). Melon necrotic spot virus is also seed transmitted, but no infection occurs when seeds containing the virus are sown in soil without the fungal vector *Olpidium bornovanus* (Hibi and Furuki 1985).

In general, plants infected after or shortly before the onset of flowering escape virus transmission. Seed transmission depends upon the ability of the virus to infect micro- and mega gametophyte tissues that give rise to infected pollen and ovaries. Ovule-based virus transmission is quite common, and few seed-transmissible viruses infect their progeny through pollen (Carroll and Mayhew 1976a, b; Carroll 1981; Hunter and Bowyer 1997). In ovule-based transmission, the virus infects floral parts early in their development. In pollen transmission, on the other hand, the virus is able to infect the floral meristems and pollen mother cells at an early stage, before the appearance of the callose layer (Hunter and Bowyer 1997). The virus-host interaction

plays a significant role in determining the frequency of seed transmission. Different isolates of the same virus show differences in frequency in the same or different cultivars of the same host (Timian 1974; Wang et al. 1993; Johansen et al. 1996, details in later part). Age of plant and environmental factors such as temperature also affect transmission rate (Hanada and Harrison 1977; Xu et al. 1991; Wang and Maule 1997).

2.8 Transmission by Vectors

2.8.1 Virus Transmission by Insects

In nature, most of the viruses are transmitted by vectors. These are organisms able to carry-over the virus from one plant to another over a short or long distance. The majority of plant virus vectors belong to the Arthropoda, in the classes Arachnida and Insecta (Harris 1981). Bennett first reported transmission of virus by insect (Bennett 1940).

Insects transfer viruses in persistent and nonpersistent manner (Watson and Roberts 1939). Persistently transmitted viruses are acquired from a diseased plant and the vector cannot transfer it to healthy plant immediately. First, the virus has to circulate within the midgut of the insect and later reach to the salivary system. The period between the acquisition of virus by vector and transmission to healthy plant is called latent period. Nonpersistent viruses are acquired by the vector and transmitted in a few seconds. The potato virus Y (PVY) is transmitted in nonpersistent manner, while potato leaf roll virus (PLRV) is persistent in its vector, *Myzus persicae*.

Aphids are the most important group of vectors because of their abundance and feeding behavior (Harris 1991). Leafhoppers and plant hoppers also are important vectors of many viruses, and they have a similar feeding mechanism (Nault and Ammar 1989). Treehoppers, thrips, whiteflies, mealybugs, mites, beetles, and other insects are also vectors of different viruses (Matthews 1991). From all known plant viruses, around 70 % are insect transmitted, and more than 50 % of those are transmitted by homopteran vectors (Francki et al. 1991). In some cases, the virus is able to replicate in vector cells. Specificity and selectivity of the transmission process influence the epidemic spread of diseases caused by plant viruses (Ferris and Berger 1993). Therefore, it is of great importance to study the transmission process with the ultimate practical purpose of designing effective strategies of controlling the spread of many economically important diseases.

2.8.1.1 Classification of Transmission Modes

Relationships of plant viruses and their insect vectors can be differentiated according to the duration of retention inside the vector. Acquisition of the virus from the vector spans from initiation of probing in the plant until the vector becomes able to transmit the virus. Period of latency is the time required after acquisition before the virus can be readily inoculated while the retention is the period for which the vector remains virulent.

Noncirculative and circulative transmission can be differentiated based on the sites of retention and the routes of movement through the vector (Matthews 1991). Noncirculative viruses are associated temporally with the surfaces of the digestive tract of the vector. These viruses have no latency period, and they are lost after molting. Noncirculative viruses can be either nonpersistent or semipersistent. Nonpersistent viruses are acquired in brief periods like seconds, and they can be inoculated immediately after acquisition, and retention is limited to short periods. Transmission is considered semipersistent when its efficiency increases directly with duration of acquisition and inoculation periods.

Circulative viruses need translocation inside the vector to be transmitted. Most of these viruses are found in vascular tissues of plants, and some cannot be inoculated mechanically. They need a latent period after acquisition. Circulative transmission can be classified into non-propagative and propagative. Non-propagative transmission occurs when the virus does not replicate in the vector, although it needs to cross barriers in the digestive tract of the vector to reach the hemolymph and, from there, the salivary glands to be inoculated during subsequent feeding. In propagative transmission, the virus is

able to replicate inside cells of the vector during its circulation; thus, the virus is a parasite of both plants and insects. In some cases, the virus can even be passed on transovarially to the vector progeny.

2.8.1.2 Nonpersistent and Semipersistent Transmission

Most of plant viruses are circulative (nonpersistent and semipersistent). In most cases, the number of virions needed for transmission may be too low (Walker and Pirone 1972), and extremely sensitive and specific methods of detection are needed to identify the presence of virus within the vector (Plumb 1989). Although retention time is generally considered short, its duration may depend on specific conditions, and, in practice, nonpersistent viruses have been shown to be retained for sufficient time to travel rather long distances in their vectors (Zeyen and Berger 1990). As is typical of piercing-sucking insects, aphids make brief insertions of their stylets to probe the adequacy of the plant as a food source, sucking sap and injecting saliva in the process. As a result, acquisition and inoculation of nonpersistent viruses occur during these probes (Lopez-Abella et al. 1988).

The acquisition of noncirculative viruses is related to intracellular ingestion by the vector, and the inoculation of the virus occurs during salivation (Martin et al. 1997). The transmissibility of viruses belonging to the genus *Cucumovirus*, on the other hand, depends on characteristics of only the capsid protein (CP) of the virions. For *Potyvirus* and *Caulimovirus*, vector transmission depends on characteristics of both the CP and the helper component (Pirone and Blanc 1996; Pirone 1977).

2.8.1.3 Circulative Non-propagative Transmission

Circulative non-propagative plant viruses are transmitted across vector membranes, and they have to survive inside the vector during circulation until they are inoculated in the host plant. The digestive system of insects can be divided into foregut, midgut, and hindgut. Entry of circulative plant viruses into the hemolymph may occur during their passage along the digestive tract through the midgut or hindgut. Once in the hemolymph, the virus moves to the salivary glands and passes into the saliva to be excreted later through the salivary duct (Gray 1996). For chewing insects such as beetles, the actual route of circulation could be different, with the viruses being transported across salivary gut membranes to the hemolymph. However, this process might not be totally essential, and the virus might be directly inoculated from the regurgitant (Wang et al. 1992).

2.8.1.4 Circulative Propagative Transmission

Some virus genera consist of plant viruses with complex infection cycles. They can replicate in the cells of their insect vectors, being parasites of both plants and animals. Propagative relationships include a long-term association with the vector that may have adverse effects on the insect host, for instance, in longevity and fecundity. In some cases, propagation includes transovarial transmission of the plant virus to the vector progeny. Propagative viruses encode genes that are differentially expressed in their infection cycle (Falk et al. 1987). Propagative plant viruses belong to families including viruses that also infect animal hosts (*Bunyaviridae*, *Reoviridae*, and *Rhabdoviridae*) and to the genera *Marafivirus* and *Tenuivirus*.

2.8.2 Virus Transmission by Nematodes

Many viruses are transmitted by soilborne nematodes. The three genera of nematode – *Xiphinema*, *Longidorus*, and *Trichodorus* – of the order Dorylaimida are known to transmit plant viruses. Nematode's vectors feed on cells of root tips with their stylet, acquiring viruses. The virus is retained within the gut or esophagus and transmitted to plants during feeding of nematodes. There are 38 *Nepoviruses* and 3 *Tobraviruses* already have been reported to be transmitted by soilborne nematodes (Williamson and Gleason 2003).

The stylets of Longidorids consist of an odontostyle, surrounded by a stylet guide sheath, for penetration of root tip cells as deep as the vascular cylinder, and a stylet extension, the odontophore, with nerve tissues and protractor muscles. The odontophore passes into the esophagus and the esophageal bulb, containing large gland cells that secrete saliva (Brown et al. 1995). During the feeding process, the stylet is inserted and after salivation the cytoplasm of penetrated cells is ingested. Trichodorids usually feed on epidermal cells by pressing their lips against the cell wall that is torn by the stylet so that the cell contents can be sucked in. Subsequently, the food passes through the pharynx and esophagus into the gut (Brown et al. 1995).

The natural distribution of *Longidorus* and *Trichodorus* spp. depends mainly on climate. Most *Xiphinema* spp. are found in the tropics and the Mediterranean. In contrast, the number of *Trichodorus* and *Paratrichodorus* spp. tends to decrease from north to south (Dijkstra and De Jager 1998). Soil type is another important factor that plays a role in the distribution of some longidorids and trichodorids. The vertical distribution of *Longidorus* and *Trichodorus* spp. shows great variation. *Longidorus* spp. prefer surface- rooted hosts; hence, most of them live in the upper soil layers, about 20 cm deep (Taylor 1967). In contrast, *Xiphinema* spp. are present in large numbers around deep-rooted host plants at depths varying from 20 cm to a couple of meters, depending on the type of soil (Taylor 1972). Usually, nematodes move to deeper layers in the soil during dry or very cold periods.

2.8.3 Virus Transmission by Fungal Vectors

The fungi are obligate endoparasites of plants that form zoospores. They belong to the *Chytridiomycota* (*Olpidium* spp.) or the Plasmodiophoromycota (*Polymyxa* spp. and *Spongospora* spp.). Two species of *Olpidium (O. bornovanus* and *O. brassicae)*, two species of *Polymyxa (P. betae* and *P. graminis)*, and one species of *Spongospora (S. subterranea)* are natural vectors of viruses (Campbell and Sim 1994; Campbell 1996). The life cycles of the two categories of fungal vectors have much in common (Adams 1991; Campbell 1996). Thick-walled resting spores are formed inside roots or young tubers of the host plant. With the plasmodiophorids the resting spores are formed in clusters, whereas the chytrids have single resting spores. When the infected roots or tubers decay in the soil, the spores are released. Depending on the conditions in the soil, resting spores germinate and release motile primary zoospores that move to roots. The zoospores attach to the root hairs or epidermal cells, often in the zone of elongation (Campbell and Fry 1966; Temmink 1971). In this process, the flagella are withdrawn and a cyst wall is secreted. Upon encystment of the zoospore, the axonema with its axonemal sheath is withdrawn inside the zoospore body (Temmink and Campbell 1969a, b; Temmink 1971).

The two types of fungal vectors use different mechanisms for penetration of the host cell. With *Olpidium* spp., belonging to the chytrids, the protoplast of the cyst enters the host through a minute pore dissolved in the wall of the host cell. With the plasmodiophorid fungi, *Polymyxa* spp. and *Spongospora* spp., the wall of the host cell is penetrated by a stylet. As soon as the cyst has settled down on root hairs or epidermal cells of the roots it forms a tube, the end of it being pointed at the surface of the host. The tube contains the stachel. Infection proceeds rapidly by evagination of the tube, resulting in a firm attachment to the host with an adhesorium and, subsequently, in puncturing the host wall with the stachel. The stachel is released into the host cell, where after the protoplast of the cyst follows. With both types of vectors, the protoplast of the fungus moves into the cytoplasm of the host, where the young thallus evolves into a multinucleate primary plasmodium that is enveloped in a thin thallus membrane. The thallus develops into zoosporangia from which the secondary zoospores are released into soil water. With *Olpidium* spp. the zoospores escape from the sporangia through a distinct exit tube penetrating the outer wall of the host cell. In the later part of the cycle, the thallus, now enveloped in a thicker membrane, develops into resting

spores or resting sporangia that may remain viable in root debris for a long time. The fungal vectors exhibit considerable host specificity.

According to the current classification of viruses (Pringle 1999), fungus-borne viruses are found in the genera *Tombusvirus*, *Carmovirus*, *Necrovirus*, and *Dianthovirus* of the family *Tombusviridae;* *Furovirus*, *Pomovirus*, *Pecluvirus*, and *Benyvirus*; and the genus *Bymovirus* of the family *Potyviridae* (Mayo 1995).

2.9 Movement of Plant Viruses

Plant virus movement is divided into two phases: (1) cell to cell, or short distance, and (2) long distance. Cell to cell movement is when an invading virus is transported from initially infected epidermal cells through the mesophyll and phloem parenchyma in the susceptible host (Carrington et al. 1996). In the absence of such cell-to-cell movement, the infection is confined to the initially infected cell and said to be subliminal (Cheo 1970; Schmitz and Rao 1996). The majority of plant viruses encode a nonstructural protein, referred to as a movement protein (MP) for promoting viral movement between cells. In some viral systems, in addition to MP, the structural or coat protein (CP) is also required to mediate this process. Thus, the overall movement process can either be coat protein independent or coat protein dependent.

2.9.1 Coat Protein-Independent Movement

In those viral systems which do not require the CP for cell-to-cell movement, the MP alone is sufficient. The best-understood example is TMV. The first two genes from its genome encode replicase proteins and the fourth encodes the structural CP (Dawson and Lehto 1990). The third gene specifies the production of a 30 kDa protein that is not required for replication or encapsidation. A TMV mutant with deletions in this gene replicates and encapsidates in protoplasts but does not move systemically in plants (Meshi et al. 1987). This suggests that the 30 kDa protein is involved in viral spread. The Lsl mutant strain of TMV does not infect tobacco at 32 °C, whereas the parental L strain remains infectious (Nishiguchi et al. 1978). Lsl infect tobacco in the presence of L at 32 °C. This implies that L can complement the movement function of Lsl (Taliansky et al. 1982b). The virus moves from cell to cell via plasmodesmata, which are, however, too small to allow free passage of virions or viral genomes (the gateway capacity or size exclusion limit (SEL) is not sufficient). To test this, fluorescent molecules of different sizes were injected into mesophyll cells of transgenic and nontransgenic plants. Molecules no larger than 0.7 kDa moved from cell to cell in nontransgenic plants, whereas 9.4 kDa molecules moved from cell to cell in the transgenic plants that accumulate the TMV-MP (Wolf et al. 1989). Although the plasmodesmata could accommodate the passage of these large molecules, which were predicted to have diameters between 2.4 and 3.1 nm, the modified plasmodesmatal SEL was still not large enough for the passage of virions or free-folded viral RNA. The modified plasmodesmata could allow the passage of viral RNA as a single-strand complex. Since TMV mutants unable to encapsidate can move from cell to cell (Saito et al. 1990), the virus must be able to move from cell to cell either as a naked RNA or as a virus-specific ribonucleoprotein complex (Dorokhov et al. 1983).

2.9.2 Tubule-Guided Mechanism

Cells infected with cowpea mosaic virus (CPMV) have distinct tubules that penetrate the plasmodesmata (Van Lent et al. 1990). When penetrated by tubules, plasmodesmata lose their characteristic desmotubules. Since the tubules penetrate the plasma membranes of protoplasts, the tubules are not modified desmotubules (Van Lent et al. 1991). Such tubular structures are involved in cell-to-cell movement of CPMV (Kasteel et al. 1996). Two overlapping genes that produce peptides 58 kDa/48 kDa in size are needed along with the viral CP gene to establish a successful

CPMV infection (Wellink and Van Kammen 1989). The 58 kDa/48 kDa proteins are not necessary for replication, but they do localize to the tubular structures (Van Lent et al. 1990). The 48 kDa protein is involved in tubule formation (Kasteel 1999). *Nepovirus* infection also induces the formation of movement-associated tubules. An antibody raised against the 45 kDa protein of tomato ringspot virus, analogous to the CPMV 48 kDa protein, recognizes the tubules (Wieczorek and Sanfacon 1993). Spherical objects appear to move through the tubules induced by both *Nepo-* and *Comoviruses* (Deom et al. 1992).

2.9.3 Non-tubule-Guided Mechanism

The cell-to-cell movement of CMV is also dependent on both the MP and the CP proteins (Taliansky and Garcia-Arenal 1995; Canto et al. 1997). CP required to support CMV movement is distinct from that of BMV CP. CMV variants lacking a CP, similar to BMV failed to move from cell to cell (Canto et al. 1997). Unlike BMV, virion assembly is not a prerequisite for CMV movement, since assembly-defective CMV variants were able to induce local lesions due to efficient cell-to-cell spread (Kaplan et al. 1998; Schmitz and Rao 1998). CMV also induces tubules in transfected protoplasts. However, tubules do not contribute to viral movement, since mutant CMV RNA3 defective in tubule production is competent for cell-to-cell and systemic spread (Canto and Palukaitis 1999).

2.9.4 Movement Complementation by Heterologous Movement Proteins and Other Virus Genes

A virus normally unable to move from cell to cell in a particular plant may be able to move with the help of a second virus of heterologous origin. Despite extensive variation in morphology, host range, and genome organization, many taxonomically distinct plant viruses exhibit complementary movement functions that may be a result of MP cross-compatibility (Atabekov et al. 1990). For example, whereas TMV-L can complement the movement of TMV-Lsl under high temperatures, PVX can complement the movement of TMV in Tm-2 gene tomato plants that normally resist TMV infection (Taliansky et al. 1982a). TMV and RCNMV are functionally homologous, since the cell-to-cell spread of movement-defective variants of TMV and RCNMV can be complemented in transgenic *Nicotiana benthamiana* plants expressing heterologous MPs (Giesman-Cookmeyer et al. 1995). However, while examining the cross-compatibility between MPs of TMV and CMV, it was observed that transgenic *N. tabacum* cv. Xanthi (tobacco) plants expressing the TMV-MP gene supported cell-to-cell movement, but not the systemic movement, of a movement-defective CMV (Cooper et al. 1996). Transgenic plants accumulating CMV MP can complement the movement of a movement-defective CMV and a wild type of BMV in inoculated leaves but cannot support the movement of TMV-Lsl, RCNMV, or *potato leafroll virus* (Kaplan et al. 1995). MPs share only a few identical amino acids (Melcher 1990). Based on amino acid and structural similarities in a nontaxonomic sense, an attempt was made to group the 30 kDa MPs. Eighteen groups are identified as "30 K" superfamilies: the MPs of *Alfamo-/ILAR-*, *Badna-*, *Bromo-*, *Capillo-/Tricho-*, *Caulimo-*, *Cucumo-*, *Diantho-*, *Furo-*, *Gemini-*, *Idaeo-*, *Nepo(A)-*, *Nepo(B)-*, *Tobamo-*, *Tobra-*, *Tombus-*, and *Umbraviruses*. Five groups of possible candidates are the MPs of *Clostero-*, *Rhabdo-*, *Tenui-*, and *Waikaviruses* and the phloem proteins. These groups can be subgrouped into four different sub-superfamilies (Melcher 2000).

Virus movement is regulated by either the MP alone or the MP in combination with the CP. Other gene products, such as replicase, also appear to influence the movement process. For example, several BMV replicase mutants capable of efficient replication and packaging in protoplasts failed to systemically infect barley plants (Traynor et al. 1991). Replicase genes of BSMV

(Weiland and Edwards 1994), CMV (Gal-On et al. 1995), and TMV (Nelson et al. 1993), as well as nonstructural protein p19 of *Tomato bushy stunt virus* (Scholthof et al. 1995) and a helper component proteinase of potyviruses (Cronin et al. 1995) have demonstrated specific roles in movement.

2.9.5 Role of Host Plant in Viral Movement

Viral movement in a given host plant is regulated also by the type of host itself. An unidentified host factor is also involved in potentiating the cell-to-cell movement of progeny viruses (Deom et al. 1992). *Nicotiana benthamiana* is susceptible to many viruses. For example, BMV has a very narrow host range. However, *N. benthamiana* is susceptible to BMV infection and the virus accumulates to very high concentrations (Rao and Grantham 1995). Following viral infections such as TMV, the MP increases the plasmodesmatal SEL in the previous plant species, permitting cell-to-cell movement of progeny virus (Lucas and Gilbertson 1994). It is possible that the plasmodesmatal SEL at the bundle sheath/phloem parenchyma cell barrier is inherently higher in *N. benthamiana* than in *N. tabacum.* This can explain why *N. benthamiana* is susceptible to a heterologous MP-mediated systemic infection by CMV and also to BMV (Rao et al. 1998). Likewise, the behavior in several hosts of a hybrid virus constructed between BSMV and RCNMV suggests that host-specific factors are involved in virus transport function (Solovyev et al. 1997).

2.10 Conclusion

Plant viruses are transmitted in nature in different ways from which the most common, with most economic importance, and widespread way is transmission by insects. There are different transmission modes. Virus movement is a complex process which involves virus and host factors. All these fundamental investigations about plant virus transmission and movement are essential for epidemiology to develop controlling strategies to stop virus spread.

References

Adams MJ (1991) Transmission of plant viruses by fungi. Ann Appl Biol 118:479–492

Atabekov J, Taliansky M, Malyshenko S, Mushegian A, Kondakova O (1990) The cell to cell movement of viruses in plants. In: Pirone TP, Shaw JG (eds) Viral genes and plant pathogenesis. Springer, New York, pp 53–55

Bennett CW (1940) The relation of viruses to plant tissues. Bot Rev 6:427–473

Broadbent L (1965) The epidemiology of tomato mosaic. XI. Seed transmission of TMV. Ann Appl Biol 56:177–205

Brown DJF, Robertson WM, Trudgill DL (1995) Transmission of viruses by plant nematodes. Annu Rev Phytopathol 33:223–249

Campbell RN (1996) Fungal transmission of plant viruses. Annu Rev Phytopathol 34:87–108

Campbell RN, Fry PR (1966) The nature of the associations between *Olpidium brassicae* and lettuce big-vein and tobacco necrosis viruses. Virology 29:222–233

Campbell RN, Sim ST (1994) Host specificity and nomenclature of *Olpidium bornovanus* (= *Olpidium radicale*) and comparisons to *Olpidium brassicae*. Can J Bot 72:1136–1143

Canto T, Palukaitis P (1999) Are tubules generated by the 3a protein for cucumber mosaic virus movement? Mol Plant-Microbe Interact 12:985–993

Canto T, Prior DAM, Hellwald KH, Oparka KJ, Palukaitis P (1997) Characterization of cucumber mosaic virus. IV. Movement protein and coat protein are both essential for cell-to-cell movement of cucumber mosaic virus. Virology 237:237–248

Carrington JC, Kasschau KD, Mahajan SK, Schaad MC (1996) Cell-to-cell and long-distance transport of viruses in plants. Plant Cell 8:1669–1681

Carroll TW (1981) Seedborne viruses virus-host interactions. In: Maramorosch K, Harris KF (eds) Plant disease and vectors: ecology and epidemiology. Academic, New York, pp 293–317

Carroll TW, Mayhew DE (1976a) Anther and pollen infection in relation to the pollen and seed transmissibility of two strains of barley stripe mosaic virus in barley. Can J Bot 54:1604–1621

Carroll TW, Mayhew DE (1976b) Occurrence of virions in developing ovules and embryo sacs in relation to the seed transmissibility of barley stripe mosaic virus. Can J Botany 54:2497–2512

Cheo PC (1970) Subliminal infection of cotton by tobacco mosaic virus. Phytopathology 60:41–46

Cooper B, Schmitz I, Rao ALN, Beachy RN, Dodds JA (1996) Cell-to-cell transport of movement-defective

cucumber mosaic and tobacco mosaic viruses in transgenic plants expressing heterologous movement protein genes. Virology 216:208–213
Cronin S, Verchot J, Haldeman-Cahill R, Schaad MC, Carrington JC (1995) Long-distance movement factor: a transport function of the potyvirus helper component proteinase. Plant Cell 7:549–559
Crowley NC (1959) Studies on the time of embryo infection by seed-transmitted viruses. Virology 8:116–123
Dawson WO, Lehto KM (1990) Regulation of tobamovirus gene expression. Adv Virus Res 38:307–342
Deom CM, Lapidot M, Beachy RN (1992) Plant virus movement proteins. Cell 69:221–224
Dijkstra J, De Jager CP (1998) Practical plant virology: protocols and exercises. Springer, Berlin/Heidelberg/New York, 459 pp
Dorokhov YL, Alexandrova NM, Miroschnichenko NA, Atabekov JG (1983) Isolation and analysis of virus-specific ribonucleoprotein of tobacco mosaic virus-infected tobacco. Virology 127:237–252
Falk BW, Tsai JH, Lommel SA (1987) Differences in levels of detection for the maize stripe virus capsid and major non-capsid proteins in plant and insect hosts. J Gen Virol 68:1801–1811
Ferris RS, Berger PH (1993) A stochastic simulation model of epidemics of arthropod vectored plant viruses. Phytopathology 83:1269–1278
Francki RIB, Fauquet CM, Knudson DL, Brown F (eds) (1991) Classification and nomenclature of viruses. Fifth report of the international committee on taxonomy of viruses. Archives of Virology (Suppl. 2), Springer, Wien/New York, 450 pp
Gal-On A, Kaplan I, Palukaitis P (1995) Differential effects of satellite RNA on the accumulation of cucumber mosaic virus RNAs and their encoded proteins in tobacco versus zucchini squash with two strains of CMV helper virus. Virology 208:58–66
Giesman-Cookmeyer D, Silver S, Vaewhongs AA, Lommel SA, Deom CM (1995) Tobamovirus and dianthovirus movement proteins are functionally homologous. Virology 13:38–45
Gray SM (1996) Plant virus proteins involved in natural vector transmission. Trends Microbiol 4:259–264
Hanada K, Harrison BD (1977) Effects of virus genotype and temperature on seed transmission of nepoviruses. Ann Appl Biol 85:79–92
Harris KF (1981) Arthropod and nematode vectors of plant viruses. Annu Rev Phytopathol 19:391–426
Harris KF (1991) Aphid transmission of plant viruses. In: Mandahar CL (ed) Plant viruses, vol 2. CRC Press, Boca Raton, pp 177–204
Hibi T and Furuki I (1985) Melon necrotic spot virus. In: AAB description of plant viruses, No. 302, AAB, Wellesbourne, Warwick, UK, 4 pp
Hull R (1994) Resistance to plant viruses: Obtaining genes by non-conventional approaches. Euphytica 75:195–205
Hunter DG, Bowyer JW (1997) Cytopathology of developing anthers and pollen mother cells from lettuce plants infected by lettuce mosaic potyvirus. J Phytopathol 1(145):521–524
Johansen IE, Dougherty WG, Keller KE, Wang D, Hampton RO (1996) Multiple viral determinants affect seed transmission of pea seedborne mosaic virus in *Pisum sativum*. J Gen Virol 77:3194–3154
Kaplan JM, Shintaku MH, Li Q, Zhang L, Marsh LE, Palukaitis P (1995) Complementation of movement defective mutants in transgenic tobacco expressing cucumber mosaic virus movement gene. Virology 209:188–199
Kaplan JM, Zhang L, Palukaitis P (1998) Characterization of cucumber mosaic virus. V. Cell-to-cell movement requires capsid protein but not virions. Virology 246:221–231
Kasteel DTG (1999) Structure, morphogenesis and function of tubular structures induced by cowpea mosaic virus. PhD thesis, Wageningen Agricultural University, Wageningen, The Netherlands, 71 pp
Kasteel DTJ, Perbal C-M, Boyer J-C, Wellink J, Goldbach RW, Maule AJ, Van Lent JWM (1996) The movement proteins of cowpea mosaic virus and cauliflower mosaic virus induce tubular structures in plant and insect cells. J Gen Virol 77:2857–2864
Lister R, Murant AF (1967) Seed transmission of nematode borne viruses. Ann Appl Biol 59:49–62
Lopez-Abella D, Bradley RHE, Harris KF (1988) Correlation between stylet paths made during superficial probing and the ability of aphids to transmit nonpersistent viruses. In: Harris KF (ed) Advances in disease vector research (Volume 5). Springer, New York, pp 251–287
Lucas WJ, Gilbertson RL (1994) Plasmodesmata in relation to viral movement within leaf tissues. Annu Rev Phytopathol 32:387–411
Martin B, Collar JL, Tjallingii WF, Fereres A (1997) Intracellular ingestion and salivation by aphids may cause the acquisition and inoculation of nonpersistently transmitted plant viruses. J Gen Virol 78:2701–2705
Matthews REF (1991) Plant virology, 3rd edn. Academic, San Diego
Mayo MA (1995) Unassigned viruses. In: Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli GP, Mayo MA, Summers MD (eds) Virus taxonomy. Springer, Wien/New York, pp 504–507
McDonald JG, Hamilton RJ (1972) Distribution of southern bean mosaic virus in the seed of *Phaseolus vulgaris*. Phytopathology 62:387–389
Melcher U (1990) Similarities between putative transport proteins of plant viruses. J Gen Virol 71:1009–1018
Melcher U (2000) The “30” superfamily of viral movement proteins. J Gen Virol 81:257–266
Meshi T, Watanabe Y, Saito T, Sugimoto A, Maeda T, Okada Y (1987) Function of the 30kd protein of tobacco mosaic virus: Involvement in cell-to-cell movement and dispensability for replication. EMBO J 6:2557–5047

Nault LR, Ammar ED (1989) Leafhopper and planthopper transmission of plant viruses. Annu Rev Entomol 34:503–529

Nelson RS, Li G, Hodgson RAJ, Beachy RN, Shintaku M (1993) Impeded phloemdependent accumulation of the masked strain of tobacco mosaic virus. Mol Plant-Microbe Interact 6:45–54

Nishiguchi M, Motoyoshi F, Oshima N (1978) Behaviour of a temperature sensitive strains of tobacco mosaic virus in tomato leaves and protoplasts. J Gen Virol 39:53–61

Pirone TP (1977) Accessory factors in nonpersistent virus transmission. In: Harris KF, Maramorosch K (eds) Aphids as virus vectors. Academic, London, pp 221–235

Pirone TP, Blanc S (1996) Helper-dependent vector transmission of plant viruses. Annu Rev Phytopathol 34:227–247

Plumb RT (1989) Detecting plant viruses in their vectors. In: Harris KF (ed) Advances in disease vector research, vol 6. Springer, Berlin, pp 191–209

Pringle CR (1999) Virus taxonomy—1999. The universal system of virus taxonomy, updated to include the new proposals ratified by the International Committee on Taxonomy of Viruses during 1998. Arch Virol 144:421–429

Rao ALN, Grantham GL (1995) Biological significance of the seven aminoterminal basic residues of brome mosaic virus coat protein. Virology 211:42–52

Rao ALN, Cooper B, Deom CM (1998) Defective movement of viruses in the family *Bromoviridae* is differently complemented in *Nicotiana benthamiana* expressing tobamovirus movement proteins. Virology 88:666–672

Rawlins TE, Tompkins CM (1936) Studies on the effect of carborundum as an abrasive in plant virus inoculations. Phytopathology 26:578–587

Reddick D, Stewart V (1919) Transmission of the virus of bean mosaic in seed and observations on thermal death-point of seed and virus. Phytopathology 9:445–450

Saito T, Yamanaka K, Okada Y (1990) Long-distance movement and viral assembly of tobacco mosaic virus mutants. Virology 176:329–336

Schmitz I, Rao ALN (1996) Molecular studies on bromovirus capsid protein. I. Characterization of cell-to-cell movement-defective RNA3 variants of brome mosaic virus. Virology 226:281–293

Schmitz I, Rao ALN (1998) Deletions in the conserved amino-terminal basic arm of cucumber mosaic virus coat protein disrupt virion assembly but do not abolish infectivity and cell-to-cell movement. Virology 248:323–331

Scholthof HB, Scholthof K-BG, Kikkert M, Jackson AO (1995) Tomato bushy stunt virus spread is regulated by two nested genes that function in cell-to-cell movement and host-dependent systemic invasion. Virology 213:425–438

Smith PR, Campbell RN, Fry PR (1969) Root discharge and survival of diseases. Phytopathology 59:1678–1687

Solovyev AG, Zelenina DA, Savenkov EI, Grdzelishvili VZ, Morozov S, Maiss E, Casper R, Atabekov JG (1997) Host-controlled cell-to-cell movement of a hybrid barley stripe mosaic virus expressing a dianthovirus movement protein. Intervirology 40:1–6

Taliansky ME, Garcia-Arenal F (1995) Role of cucumovirus capsid protein in long distance movement within the infected plant. J Virol 69:916–922

Taliansky ME, Atabekova TI, Kaplan IB, Morozov SY, Malyshenko SI, Atakov JG (1982a) A study of TMV TS mutant NI2519. I. Complementation experiments. Virology 76:701–708

Taliansky ME, Malyshenko SI, Pshennikova ES, Kaplan IB, Ulanova EF, Atakov JG (1982b) Plant virus-specific transport function. II. A factor controlling virus host range. Virology 122:327–331

Taylor CE (1967) The multiplication of *Longidorus elongatus* (de Man) on different host plants with reference to virus transmission. Ann Appl Biol 59:275–281

Taylor CE (1972) Transmission of viruses by nematodes. In: Kado EI, Agrawal HO (eds) Principles and techniques in plant virology. Van Nostrand Reinhold, New York, pp 226–247

Taylor RH, Grogan RG, Kimble KA (1961) Transmission of tobacco mosaic virus in tomato seed. Phytopathology 51:837–842

Temmink JHM (1971) An ultrastructural study of *Olpidium brassicae* and its transmission of tobacco necrosis virus. Mededelingen Landbouwhogeschool Wageningen 71:1–135

Temmink JHM, Campbell RN (1969a) The ultrastructure of *Olpidium brassicae.* II. Zoospores. Can J Botany 47:227–231

Temmink JHM, Campbell RN (1969b) The ultrastructure of *Olpidium brassicae.* III. Infection of host roots. Can J Botany 47:421–424

Timian RG (1974) The range of symbiosis of barley and barley stripe mosaic virus. Phytopathology 64:342–345

Tomlinson J, Faithful E, Flewett T, Beards G (1982) Isolation of infective tomato bushy stunt virus after passage through the human alimentary tract. Nature 300:637–638

Tomlinson J, Faithful E, Webb M, Frazier R, Seeley N (1984) Chenopodium necrosis: a distinctive strain of tobacco necrosis virus isolated from river water. Ann Appl Biol 102:135–147

Traynor P, Young BM, Ahlquist P (1991) Deletion analysis of brome mosaic virus 2a protein: Effects on RNA replication and systemic spread. J Virol 65:2807–2815

Uyemoto JK, Grogan RG (1977) Southern bean mosaic virus: Evidence for seed transmission in bean embryos. Phytopathology 67:1190–1196

Van den Heuvel JFJM, Franz AWE, Van der Wilk F (1999) Molecular basis of virus transmission. In:

Mandahar CL (ed) Molecular biology of plant viruses. Kluwer Academic Publishers, Boston/Dordrecht/London, pp 183–200

Van Lent J, Wellink J, Goldbach RW (1990) Evidence of the involvement of the 58K and 48K proteins in the intercellular movement of cowpea mosaic virus. J Gen Virol 71:219–223

Van Lent J, Storms MMH, Van der Meer F, Wellink J, Goldbach RW (1991) Tubular structures involved in movement of cowpea mosaic virus are also formed in infected cowpea protoplasts. J Gen Virol 72:2615–2623

Walker HL, Pirone TP (1972) Particle numbers associated with mechanical and aphid transmission of some viruses. Phytopathology 82:1283–1288

Wang D, Maule AJ (1997) Contrasting patterns in the spread of two seed-borne viruses in pea embryos. Plant J 11:1333–1340

Wang RY, Gergerich RC, Kim KS (1992) Noncirculative transmission of plant viruses by leaf feeding beetles. Phytopathology 82:946–950

Wang D, Woods RD, Cockbain AJ, Maule AJ, Biddle AJ (1993) The susceptibility of pea cultivars to pea seed-borne mosaic virus infection and virus transmission in the UK. Plant Pathol 42:42–47

Watson HA, Roberts FH (1939) A comparative study of the transmission of Hyoscyamus virus 3, potato virus Y, and cucumber virus 1 by the vectors Myzus persicae (Sulz.), M. circumflexus (Buckton) and Macrosiphum gel (Koch). Proc R Soc Lond Ser B 127:543–576

Weiland JJ, Edwards MC (1994) A single nucleotide substitution in the alpha-a gene confers oat pathogenicity to barley stripe mosaic virus strain ND18. Mol Plant-Microbe Interact 9:62–67

Wellink J, van Kammen A (1989) Cell-to-cell transport of cowpea mosaic virus requires both the 54/48K proteins and the capsid proteins. J Gen Virol 51:317–325

Wieczorek A, Sanfacon H (1993) Characterization and subcellular location of tomato ringspot nepovirus putative movement protein. Virology 194:734–743

Williamson VM, Gleason C (2003) Plant–nematode interactions. Curr Opin Plant Biol 6:327–333

Wolf S, Deom CM, Beachy RN, Lucas WJ (1989) Movement protein of tobacco mosaic virus modifies plasmodesmatal size exclusion limit. Science 246:377–379

Xu Z, Chen K, Zhang Z, Chen J (1991) Seed transmission of peanut stripe virus in peanut. Plant Dis 75:723–726

Zeyen RJ, Berger PH (1990) Is the concept of short retention times for aphid-borne nonpersistent plant viruses sound? Phytopathology 80:769–771