Plant Virus Transmission by Insects

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Most plant viruses depend on vectors for their survival and spread. Most vectors are piercing-sucking insects that transmit plant viruses in either the circulative virus (CV) or noncirculative virus (NCV). NCV are carried on the lining cuticle of vectors stylets. CVs cross the vectors' gut, move internally to the salivary glands (SG), cross the SG membranes to be ejected upon feeding.

Transmissibility of NCVs depends on motifs of coat protein and for Potyviruses and Caulimoviruses also on helper proteins (encoded by the virus). NCV proteins were found to associate with vectors' cuticle proteins. Transmissibility of CVs depends on proteins comprising the virus capsid (the coat protein and the read-through protein) and on symbionin (produced by vectors' symbionts). Passage of CV through the gut has been also associated with vectors' proteins.

To suppress plant virus epidemics, several control measures that interfere with vector landing or feeding are proposed.

Advanced article

Article Contents

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Introduction

Insect vectors of plant viruses are found in 7 of the 32 orders of the class Insecta. They transmit plant viruses by four major transmission modes and a number of viral and insect proteins have been found to control virus–vector association. Interference with vector landing and/or with the transmission process and/or with virus replication and movement is used to control plant virus epidemics.

Importance of Insect Vectors

Most plant viruses depend on vectors for their survival for two principal reasons:

1. An impermeable cuticle coats the plant epidermis, preventing entry of virus particles (animal viruses enter readily through natural openings). Most vectors are insects (noninsect vectors include mites, nematodes and fungi). Several plant viruses may spread by contact or by vegetative reproduction. Many insects such as hemipterans are well adapted to their role as vectors by their capacity to pierce the epidermis and delicately deposit the virus in the cytoplasm without risking the integrity of the plant cell. **See also**: Invertebrates and Fungi in Plant Virus Diseases; Plant Cuticle
2. Plants are rooted and lack independent mobility. Therefore, many viruses depend on insects for transport among hosts (unlike animals that, by their own mobility, transport the virus to new niches).

Insect-borne plant viruses may cause severe or even crippling losses to many annual and perennial crops. On occasion, insects are responsible for transition from a nonspreading form to the epidemic form of diseases. Outbreaks of disease caused by insect vectors are demonstrated in two examples: In perennials, the almost total destruction of the citrus industry in the 1930s in Argentina and Brazil is attributed to the aphid *Toxoptera citricidus*. This aphid was recently found in Portugal and Spain threatening the Mediterranean citriculture. In annuals, outbreaks of *Tomato spotted wilt virus* (TSWV) in the last decades is attributed to the spread of the thrips *Frankliniella occidentalis*. **See also**: Epidemiology of Plant Virus Diseases; Virus Diseases of Cereals; Virus Diseases of Tropical Crops

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Taxonomy

Insect vectors of plant viruses are found in 7 out of the 32 orders of the class Insecta. The majority of vectors are found in the two orders of insects with pierce-sucking mouthparts: Thysanoptera (6) and Hemiptera (300). Fewer vector species are found in 5 orders of chewing insects (number in parenthesis): Orthoptera (10), Dermaptera (1), Coleoptera (30), Lepidoptera (4) and Diptera (2). Apparently, the feeding organs of the Hemiptera are the principal reason for their successful role as vectors. Description of the biology and flight behaviour of insect vectors is beyond the scope of this article. **See also**: Insecta (Insects)

Mechanisms of Transmission

Progress in the molecular biology of viruses and their vectors has assisted greatly in identifying motifs in the viral genome and in viral and vector proteins, thus adding to the understanding of the process of virus transmission by insects.

Major transmission modes: persistent versus nonpersistent; circulative versus noncirculative

Plant viruses demonstrate a high level of specificity for the group of insects that may transmit them (a virus that is transmitted by one type of vector will not be transmitted by another). This article deals with plant viruses that do not propagate in their vector. The list of insect-borne virus groups and their vectors is summarized in **Table 1**. **See also**: Plant Virus Identification; Viruses and Plant Disease

Modes of transmission

In the 1930s, Watson and Roberts proposed modes of virus transmission by insects. The basis for their assigning viruses to these modes was the duration of virus retention in the vector. Originally, they proposed two modes: nonpersistent for short retention or for 'less than the time the virus survives in leaf extracts'; and persistent for extended retention, often for life. However, several viruses showed an intermediate retention in their vector. This led Sylvester to designate the term semipersistent viruses. In time, a different terminology was proposed for modes of transmission, based on the site at which the virus is carried in the insect. Thus, nonpersistent viruses were termed stylet-borne, whereas persistent viruses were termed circulative. In time, additional parameters were attached to each of the modes of transmission (**Table 2**). Nonpersistent viruses are acquired and inoculated during brief probing times, do not require a latent period in the vector, and are transmitted by many aphid species. Semipersistent viruses need longer periods (hours) for acquisition and transmission than do nonpersistent viruses. They have a narrower range of vector species. However, they too need no latent period and are lost when the vector moults. In persistent viruses, the longer the acquisition and inoculation times the higher is the rate of transmission. They mostly have a narrow

Table 1 Groups of viruses and insect species that serve as vectors

Virus groups	Mode	Persistence	Presence in vector	Insects involved
Alfamovirus	N	Few hours	External	Aphids
Badnavirus	S	Days	External	Mealybugs and leafhoppers
Begovirus	C	Weeks	Internal	Whiteflies
Carlavirus	N	Few hours	External	Aphids
Caulimovirus	N	Many hours	External	Aphids
Closterovirus	S	Many hours	External	Aphids or mealybugs
Comovirus	S	Days	Internal	Beetles
Cucumovirus	S	Few hours	External	Aphids
Curtovirus	C	Weeks	Internal	Leafhoppers
Enamovirus	C	Weeks	Internal	Aphids
Fabavirus	N	Few hours	External	Aphids
Luteovirus	C	Weeks	Internal	Aphids
Poleroviruses	C	Weeks	Internal	Aphids
Machlomovirus	SP	Many days	External	Leafhoppers
Mastrevirus	C	Weeks	Internal	Leafhoppers
Potyvirus	N	Few hours	External	Aphids
Sequivrus	SP	Few hours	External	Aphids
Sobemovirus	SP	Days	Not determined	Beetles
Tymovirus	SP	Days	Not determined	Beetles
Waikavirus	SP	Few days	External	Leafhoppers

C, circulative; N, nonpersistent; SP, semipersistent. Internal = virus cross gut and salivary gland barriers. External = virus does not cross the gut barrier; it remains attached to the foregut epithelium.

Table 2 Principal characteristics of the modes of virus transmission by insects

	External (noncirculative)		Internal-circulative[a]
Feature	Nonpersistent	Semipersistent	Persistent
Duration of retention	Brief (few hours)	Intermediate (few days)	Long (days to months)
Duration of acquisition and transmission	Brief (seconds)	Intermediate (hours)	Long (hours to days)
Latent period	Not required	Not required	Required
Tissue where virus is acquired and inoculated	Epidermis and parenchyma	Epidermis, parenchyma and phloem	Mostly parenchyma and phloem
Pre-acquisition fasting	Increase transmission	No effects	No effect
Passage through moult	Negative	Negative	Positive
Insect species specificity	Low	Intermediate	High
Sequential inoculation	Poor	Intermediate	Good

[a]Internal-circulative = virus cross gut and salivary gland barriers.

range of vectors, pass through moult and need a latent period.

Many thorough biological, microscopical, immunological, molecular techniques and electronic devices have subsequently been used to elucidate the mechanisms of transmission. Two principal modes of transmission emerged: (1) circulative or internal, where the virus crosses body barriers and enters the circulatory system of the insect and accumulates inside the salivary glands and (2) noncirculative or external, where the virus remains attached to the cuticle (cuticle-borne) of the insect and does not cross body barriers.

Mechanism of Nonpersistent Transmission

Virus particles, but not their naked nucleic acids, are the pathogenic units that are transmitted by insects to initiate infection. However, viral nucleic acids (either deoxyribonucleic acid, DNA or ribonucleic acid, RNA) are sufficient to cause infection when introduced to plant cells by artificial means (rubbing, bombardment, agro-infection, etc.). This suggests that protein molecules encapsidating the nucleic acid are needed to interact with specific sites present in the vector. Investigation of the role of the coat protein (CP) in virus transmissibility was possible due to the occurrence of virus strains that differ in their specificity for vector species; and the occurrence of strains that have lost transmissibility after continuous mechanical inoculation (see details in the following sections). Recently, the precise location and chemical nature of the first noncircultive virus receptor within the vector mouthparts has been identified. Also, the specific probing behaviour activities of insect vectors linked to the transmission of plant viruses have also been elucidated with the help of electronic devices. **See also**: Viral Capsids and Envelopes: Structure and Function; Virus Structure

Role of the capsid protein in the transmission of nonpersistent viruses (Perry, 2001; Raccah *et al.*, 2001; Pirone and Perry, 2002)

Cucumoviruses

For *Cucumber mosaic virus* (CMV), Gera and coworkers (cited in Perry, 2001) provided evidence that the genome of a poorly transmissible strain became transmissible when encapsidated *in vitro* with the CP of highly transmissible strain. A follow-up of these studies was achieved by Perry *et al*. (1998) (cited in Perry, 2001 who designed chimaeric RNA 3 cDNA (complimentary DNA) constructs to introduce mutations in the CP. As a result of these studies, three amino acid mutations in the CP were found to affect transmission of CMV by *Aphis gossypii*. In a recent study, these authors discovered that the transmissibility of CMV by *Myzus persicae* requires two mutations in the CP (in position 25 and 214) in addition to the mutations in position 129, 162 and 168 that were reported in their former study (cited in Perry, 2001). **See also**: *Bromoviridae* and Allies

Potyviruses

To identify the determinants of a potyvirus transmission by aphids, the amino acid sequences of the CP of aphid transmissible (AT) and nonaphid-transmissible (NAT) virus strains were compared. The comparison revealed a conserved amino acid triplet, Asp-Ala-Gly (DAG) within the highly nonconserved and exposed *N*-terminal end of the CP. The NAT strains were found to have a mutated triplet. A mutation from Gly to Glu (DAG to DAE) was introduced in the CP of an AT strain of *Tobacco vein mottling virus* (TVMV), rendering it nontransmissible. The role of the DAG motif of the CP in aphid transmission was then confirmed also for a NAT strain of *Zucchini yellow mosaic virus* (ZYMV) by changing Thr to Ala (DTG to DAG), this time restoring transmissibility. Effects on transmission of TVMV were noted not only for the DAG triplet but also for amino acids in the immediate vicinity of it (Atreya *et al*.,

1990; Gal-on *et al*., 1992; Lopez-Moya *et al*., 1999 all cited in Raccah *et al*., 2001). **See also**: *Potyviridae*

Electron-microscopic studies provided evidence that the DAG motif in potyviruses is involved in retaining the virus in the aphid's mouthparts. The mechanism is apparently via an interaction of the DAG with the helper component (HC), as recently shown by the protein-blotting overlay technique (Blanc *et al*., 1997; Peng *et al*., 1998, cited in Raccah *et al*., 2001).

Potexviruses

Potato aucuba mosaic virus (PAMV) is not transmissible by aphids, but its transmission is possible when it is assisted by potyviruses. The DAG motif of the CP sequence of PAMV is not present in *Potato virus X* (PVX), but transfer of the DAG motif from PAMV to PVX, resulted in its becoming aphid-transmissible (Baulcombe *et al*., 1993 cited in Raccah *et al*., 2001). **See also**: Potexviruses and Carlaviruses – Short Filamentous Viruses

Virus-encoded proteins that affect noncirculative virus transmission by insects (Blanc *et al*., 2001; Raccah *et al*., 2001)

The vectors of potyviruses and caulimoviruses cannot transmit purified virus particles unless these are presented in mixture with a nonstructural virus-encoded protein.

Potyviruses

The helper phenomenon was first reported by Kassanis and Govier (cited in Raccah *et al*., 2001) in experiments in which the NAT in PAMV became transmissible from plants co-infected with the C strain of *Potato virus Y* (PVY^{C}). Later, Govier and Kassanis demonstrated that potyvirus transmission requires a factor present in the supernatant fraction (termed helper component, HC) in addition to the virus particles. They showed that transmission occurs only if the virus is acquired in mixture or after the acquisition of the HC. This led to the 'bridge' hypothesis, where the HC binds to aphid mouthparts on one side and to virions on the other. This binding ensures virus retention until release into the next host. Sequencing of the potyviral genome and identification of its translation products assisted in characterizing it as a nonstructural protein encoded by the HC-Pro region of the potyvirus genome. The helper function in transmission was assigned to the *N*-terminal and central regions of the HC-Pro. Most HCs have a predicted molecular mass in the range 50–60 kDa. The proposed biologically active form is a dimer with molecular mass of 110–150 kDa. Domains that are involved in vector transmission were traced by comparing strains with active and inactive HCs. Direct proof was obtained for TVMV, where loss of HC activity was associated with a mutation from Lys to Glu (K to E) in the highly conserved Lys-Ile-Thr-Cys (KITC) motif of TVMV HC-Pro. This mutation was also found in mutants of PVY and ZYMV HCs. The KITC motif of the HC is not involved in binding to virions transmission-defective ZYMV-Ct with E instead of K in the KLSC motif was bound efficiently to virions in overlay blotting experiments (Peng *et al*., 1998 cited in Raccah *et al*., 2001). **See also**: Virus Diseases of Potatoes

A domain in the central region of the HC-Pro gene, Pro-Thr-Lys (PTK) was found to be associated with HC assistance in transmission of ZYMV. A mutation from Pro to Ala in the PTK motif resulted in loss of helper activity. The PTK motif was found to affect the HC binding to virions in overlay blotting experiments (Peng *et al*., 1998 cited in Raccah *et al*., 2001). A proposed model summarizing the interaction between the virions, the HC and the aphid stylets is depicted in **Figure 1**. **See also**: Mutagenesis: Site-specific

The role of the HC in retaining the virus in the stylet was shown by comparing aphids fed on mixtures of transmissible TEV or TVMV virions and functional PVY HC or TVMV HC (motif KITC) with those fed on nonfunctional HC (motif EITC) (Wang *et al*., 1996 cited in Raccah *et al*., 2001).

Caulimoviruses

Caulimoviruses have also adopted a helper-dependent transmission strategy, but in a rather more complex manner than potyviruses. CaMV (cauliflower mosaic viris) requires two viral-encoded nonstructural proteins, P2 and P3. A P2-P3-virion complex is formed, with P2 binding to the aphid whereas P3 binding to the virions (Drucker *et al*., 2002). Interestingly, aphids may first acquire P2 from infected mesophyll cells, and P3-virion complexes can subsequently be taken up from other mesophyll or phloem cells. Furthermore, the HC motif directly involved in specific vector recognition was identified at position 6 of the *N*-terminus of P2. A single mutation of one aa that may

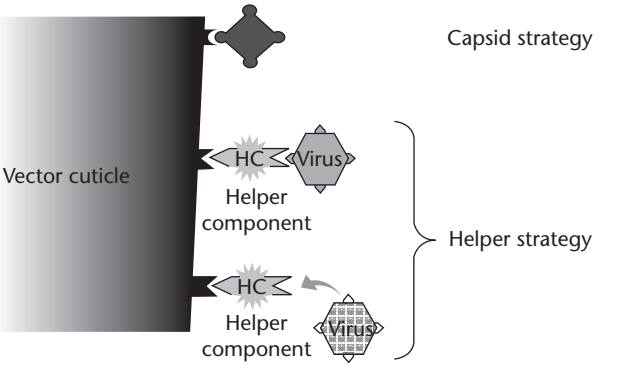

Figure 1 Model describing the different strategies for virus–vector interaction in noncirculative transmission. These strategies enable retention of virus particles on the maxillary stylets at the surface of the cuticular lining. In the capsid strategy, a motif of the coat protein directly binds to the vector's receptor. In the helper strategy, virus–vector binding is mediated by the helper component (HC), which creates a 'molecular bridge' between the two. HC can be acquired alone and thereby allow HC-transcomplementation as symbolized by the arrow and a different shading for the virus particle subsequently acquired. Reproduced by permission from Froissart, R, Michalakis, Y and Blanc, S (2002) Helper component transcomplementation in the vector transmission of plant viruses. *Phytopathology* **92**: 576–579.

appear spontaneously changes the spectrum of vectors transmitting CaMV (Moreno *et al*., 2005). **See also**: *Caulimoviridae* (Plant Pararetroviruses)

Is helper protein used in other virus–vector systems?

Indirect evidence suggests that helper is involved in several other systems. The semipersistently transmitted parsnip yellow fleck virus is not transmissible by aphids unless acquired with then thriscus yellows virus. A dense material with virus-like particles was seen in aphids' mouthparts after acquisition of the virus. *Rice tungro spherical virus* (RTSV) is transmissible by several *Nephottetix* leafhopper species. RTSV assists the transmission of a second virus, the *Rice tungro bacilliform virus*. *Maize chlorotic dwarf virus* is semipersistently transmitted by leafhoppers and is considered to have helper components. Lack of vector transmissibility of purified virions led to the speculation that a helper is needed also for transmission of carlaviruses and closteroviruses (Raccah *et al*., 1990). **See also**: *Closteroviridae*; Invertebrates and Fungi in Plant Virus Diseases

Analysis of Virus Transmission by Electrical Penetration Graphs (Fereres and Collar, 2001)

Electronic devices can distinguish between the intercellular and intracellular environments, which makes it possible to know when the plant cell membranes are punctured by the insect stylets. When a cell membrane is punctured a very distinctive electrical penetration graph (EPG) signal is recorded in the form of a potential drop (pd). Other distinct waveforms and activities which are relevant to virus transmission by aphids and whiteflies are phloem salivation and phloem ingestion. Acquisition of stylet-borne viruses occurs after very brief (<1 min) probes and only when cell membranes are punctured by the stylets as shown by electron microscopy and EPG studies. Detailed analysis of direct current-EPG signals during intracellular stylet punctures allow to differentiate three specific and distinct subphases: II-1, II-2 and II-3. Acquisition of stylet-borne viruses is associated to subphase II-3. Acquisition during the first intracellular puncture is not only restricted to typical nonpersistent viruses such as CMV or PVY but it also occurs for semipersistent viruses such as CaMV. The main difference is that CaMV is preferentially acquired after committed phloem ingestion whereas typical nonpersistent viruses are only acquired during brief superficial intracellular punctures. Work conducted by Fereres and coworkers showed that subphase II-1 within the first intracellular puncture was associated to the inoculation of both PVY and CMV. Based on this finding and the fact that both salivary and alimentary canals fuse together in a common duct at the very tip of the maxillary stylets, the ingestion-salivation hypothesis was proposed. The results obtained also suggested that watery salivation was the mechanism involved in flushing out virus particles from the common duct during cell penetration. Later work using *Pea enation mosaic virus* (Genus *Enamovirus*, PEMV) as a marker for intracellular salivation confirmed this hypothesis.

Mode of transmission of viruses by beetles (Gergerich, 2001)

Beetle vectors of plant viruses are known in four families (Chrysomellidae, Coccinellidae, Curculionidae and Meloidae). Beetle-borne viruses have a unique mode of transmission. The viruses are transmitted in the beetle's regurgitant and there is no latent period in the vector. The original assumption was that regurgitant components selectively inactivate particles of beetle nontransmissible viruses. However, mixing preparations of a variety of viruses with beetle regurgitant had insignificant effect on most viruses (beetle-borne or not). Some beetle-borne viruses are circulative, as they were found to move into the insect haemolymph immediately after ingestion. Beetles can also be rendered viruliferous by injecting virus into the haemolymph. However, Wang and coworkers found that beetles may transmit viruses even if they are not carried in the haemolymph. The retention of inoculativity of beetles differs for different beetle vectors; thus, *Epilachna varivestis* retains *Cow pea severe mosaic virus* (CPSMV) for 1 day, while *Cerotoma trifurcata* transmitted the same virus for several days. The virus does not propagate in the beetle as the virus titre declines with time. Gergerich and coworkers demonstrated the unique role of the regurgitant in the infection process. Viruses not transmissible by beetles were mechanically infectious to wounded hosts, but when regurgitant was added to the inoculum mixture only beetle-borne viruses remained infectious. The inability of virus particles to infect hosts was not due to inactivation since, when purified away from the regurgitant virus particles regained infectivity. This finding suggests that an inhibitor in the regurgitant affected the host itself or the interaction between virus and host and that viruses transmissible by beetles differ from other viruses in the fast translocation to nonwounded cells through the xylem and in the manner in which they initiate primary infection. **See also**: Plant Responses to Wounding; Xylem Structure and Function

Mechanism of Nonpropagative, Circulative Transmission (Brault *et al*., 2001; Gray and Gildow, 2003)

Circulative (internal) viruses are carried in the interior of the vector body. Some of the circulative viruses propagate in the insect and are therefore termed circulative-propagative. A list of circulative viruses is given in **Table 1**. The luteoviruses and PEMV are the best-studied

circulative viruses (Gray and Gildow, 2003). **See also**: Luteoviruses

Transmission cycle

The transmission cycle of a circulative virus includes five stages: (a) Ingestion from the infected host plant to bucal cavity and intenstines of the vector, (b) Passage of the virus through the vector's gut, (c) Retention in the haemocoel or other internal tissues, (d) Passage of the virus to the salivary glands and then (e) via the salivary duct in the maxillary stylets to internal tissue (mostly phloem) of the host plant (**Figure 2**). Ingestion: The aphid stylets, while piercing and sucking are inserted intercellularly to reach the phloem. When sap is ingested, virus ingestion takes place. Then, the virus passes from the gut lumen to the haemocoel. A specific and active transport of a circulative virus through the gut occurs when being recognized by the epithelial cells. Virus particles are retained in the haemolymph for several weeks. Survival in the haemolymph may depend on the presence of symbionin (see later). In the Luteoviridae, virus particles that are carried in the haemolymph, need to cross the basal lamina of the accessory salivary gland (ASG) to be ejected by the salivary secretions to the plant tissues. The basal lamina of ASG consists of collagen that may serve as a selective filter, allowing differential binding and passage virus particles. On the way to exterior, virus particles must be transported across a third preferential barrier, the plasmalemma of the ASG, by receptor-mediated endocytosis. It is likely that the virus movement across these barriers is involved with different viral proteins or protein domains. **See also**: Clathrin-coated Vesicles and Receptor-mediated Endocytosis; Digestive System of Invertebrates; Phloem Structure and Function; Virus and Host Plant Interactions

Role of viral capsid protein for insect transmission of circulative viruses

Protein subunits are important for the specificity of transmission of circulative viruses. Rochow (1969) (Brault *et al.*, 2001), showed that strains of *Barley yellow dwarf virus* (BYDV) that are transmitted by one aphid species become transmitted by another aphid species if co-infected with another strain of BYDV. This phenomenon was attributed by Rochow to heteroencapsidation, where the nontransmissible RNA is encapsidated with some protein subunits of the transmissible strain.

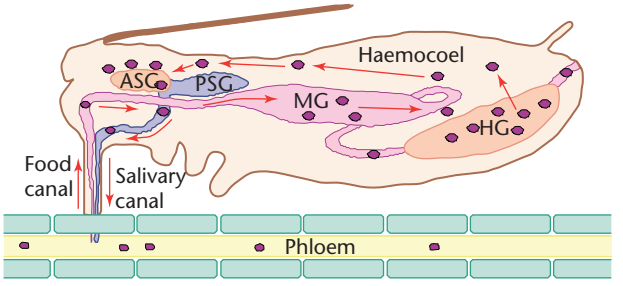

Figure 2 Schematic diagram of an aphid feeding and luteovirus transmission. Arrows indicate the circulative route for t PSG, principal salivary gland. From Chay CA, Gunasinge UB, Dinesh-Kumar SP, Miller WA and Gray SM (1996) Aphid transmission and systemic plant infection determinants of barley yellow dwarf luteovirus-PAV are contained in the coat protein readthrough domain and 17-KDa protein, respectively. *Virology* **219**: 57–65.

Viral proteins involved in transmission: The coat protein and the read-through protein (Brault *et al.*, 2001; Gray and Gildow, 2003)

PEMV and luteovirus particles are composed of two types of capsomeres. The predominant one is CP (*c.* 22–24 kDa). Another minor one, believed to be on the surface of the virion is the read-through (RT) protein (*c.* 55–58 kDa). The RT protein results from a larger protein translated via the weak stop codon of the CP. The open reading frame encodes for a 72–74 kDa protein, of which the *C*-terminal half of the resulting protein is digested yielding a 55–58 kDa proteins. This protein is also found when CP is obtained from virus preparations. Virions encapsidated with the CP alone, were not transmitted by aphids (but are found in the haemocoel following feeding). Also, these virions are infective when agro-inoculated (cited in Gray and Gildow, 2003). These findings led to the conclusion that the RT protein is needed for aphid transmission. Mutants of *Beet western yellow virus* (BWYV) without the RT protein were not detectable in the ASG and are nontransmissible by aphids. Mutants of read through domain (RTD) in various domains at the *C*-terminus did not affect aphid transmissibility. Mutation at the *N*-terminus of the RTD resulted in a protein that did not incorporate in the virus particle, but ingested particles are found in haemolymph. This suggests that the CP provide the signal for crossing the hindgut barrier while the RT is the protein that associates with the ASG. However, recent reports show that particles encapsidated with the 22 kDa CP alone, were found not only in the haemolymph but also in the ASG cells and in the salivary duct. This finding seems to be in contrast with the hypothesis that the RT is needed for crossing the ASG barrier.

Symbionin

The symbionin is produced by endosymbiotic bacteria of the genus *Buchnera* in specialized cells located in the abdomen mycetome of aphids. The RT protein was found to interact with the GroEL (a bacterial protein showing homology with symbionin). Mutational analysis of the RT protein of *Beet western yellow virus* (BWYV) attributes the virus binding capacity to a conserved region in the GroEL molecule. BWYV engineered to be encapsidated with CP alone (with no RT protein subunits) did not bind to *Buchnera* GroEL. Also, *in vivo* studies showed that BWYV virions lacking the RT protein were significantly less persistent in the haemolymph than were virions with the RT protein. This led to the hypothesis which states that the interaction between *Buchnera* GroEL and the RT protein protects the virus from rapid degradation in the

haemolymph. Comparison of the RT domain from different luteoviruses and PEMV revealed several conserved amino acid residues that may be important for the interaction with *Buchnera* GroEL. (van den Heuvel *et al*., 1997, cited in Brault *et al*., 2001). In a recent study, Hogenhout *et al*. (1998) (cited in Gray and Gildow, 2003) demonstrated by mutational analysis of the gene encoding for *MpB* GroEL that the PLRV binding site is located in the equatorial domain and not in the apical domain of the symbionin. The exact function of the symbionin is not known. It can either confer protection of virus particle when in the haemolymph and/or facilitate passage of the virus across the ASG barrier. **See also**: Chaperonins; Endosymbionts; Immunology of Invertebrates: Humoral

Geminiviruses

The role of the CP in geminivirus transmission was determined by exchanging the CP gene of two viruses differing in vector specificity. Thus, injection of the recombinant whitefly-borne *African cassava mosaic virus* (ACMV) with the *Beet curly top virus* (BCTV) CP enabled transmission by leafhoppers. However, transmission was not achieved by leafhoppers acquiring the recombinant virus by feeding. This suggests that the CP is needed to pass from the haemocoel to the salivary glands (Hull, 1994). **See also**: *Geminiviridae*

Insect Proteins Involved in Virus–Vector Interactions

Recently, the retention sites and specific proteins acting as receptors of both noncirculative and circulative viruses have been identified. A nonglycosylated protein deeply embedded in the chitin matrix of the aphid's maxillary stylets is involved in the retention of CaMV. This protein receptor present in three effective vector species but absent in a nonvector is located exclusively at the stylet tips in the bottom bed of the common duct where the food and salivary canals fuse together (Uzest *et al*., 2007). Using a proteomic approach, four cuticular proteins that were extracted, separated and identified from *Myzus persicae* were able to bind *in vitro* to active potyviral HC-Pro but not to the mutated HC-Pro of the same virues (Dombrovsky *et al*., 2007).

A similar approach was taken to show that four proteins from *Schyzaphis graminum* are involved in the ability to bind to the circulative *Cereal yellow dwarf virus*–RPV polerovirus (Yang *et al*., 2008). These proteins from *Septoria graminum* origin seem to play a key role in the high level of vector specificity, possibly by facilitating the passage of the virus through the gut and salivary gland tissues. Similarly, two proteins isolated from head tissues of the aphid vector, *Sitobion avenae*, have been identified as potential receptors for another circulative virus (BYDV–MAV; *Luteoviridae*) (Li *et al*., 2001, cited in Yang *et al*., 2008).

These findings may lead in the future to the use of viral genes encoding for proteins that are defective in the ability to assist transmission in transgenic plants. This may prevent vector inoculation. Also, plants encoding for molecules (e.g. peptides) able to bind to cuticle protein receptors in the vector mouthparts may interfere with the process of virus retention. If successful, this form of virus prevention will complement those based on reduced multiplication and movement.

Control of Virus Diseases by Interfering with Vectors' Activity (Raviv and Antignus, 2004)

Herein, we will discuss measures aimed against vector activity. These measures are among the most successful approaches used to suppress virus epidemics. Other control measures (e.g. breeding for resistance to the pathogen, sanitation, prevention, natural and pathogen-derived resistance) will not be discussed herein and reader should consult the Further Reading list. Control measures against vectors and vector activities can be grouped into four classes: (1) killing the vectors with insecticides, (2) reducing the virus sources, (3) interference with vector landing on the crop and (4) interference with the transmission process. **See also**: Impact of Genetically Modified Organisms (GMOs); Plant Breeding and Crop Improvement

- *Use of insecticides*: Despite the wide range of insecticides, use of insecticides is not the preferred solution to prevent vector activity. Many viruses are introduced into crops by visiting insects that inoculate during their first probing activities. Vectors for nonpersistent (and partly semipersistent) viruses need relatively short inoculation times – much shorter than the time needed for insecticides to kill. In addition, insecticides can induce restlessness in insects, with the result that they make more inoculation attempts than do calm insects. Exceptions are vectors that colonize the crop and transmit circulative viruses, for which insecticide control may result in reduced spread of virus. **See also**: Integrated Pest Management
- *Reducing virus sources*: Use of virus-free seeds and/or propagative organs results in minimal primary infection. This can be complemented by removal of sources of infection in and around the crop, removal of plant remains from a former season and, if necessary, creation of a time gap between crops and/or space gap between plots. These operations will reduce the numbers of viruliferous insects that reach the crop.
- *Interference with vector landing on crops* is achieved by altering the attraction of insects to colours. Insects (e.g. aphids) are repelled from reflective surfaces: this effect led to the use of metallic reflective surfaces, straw

mulches or kaolin particle films. Landing can be prevented by the use of physical barriers. Insect-proof nets greatly reduced virus incidence and the need for insecticide applications against the Tomato yellow leaf curl geminiviruses in tomato. Camouflaging nets greatly reduce insect landing and also virus infection. This measure is now being used commercially for the protection of papaya from *Papaya ring spot virus* in Taiwan.

UV-absorbing plastics and nets

This novel and most promising approach was developed by Antignus and coworkers (as cited in Raviv and Antignus, 2004). Polyethylene sheets and nets that absorb UV were found to greatly reduce virus incidence. An impressive reduction of insect landing was recorded for whiteflies, aphids or thrips. The mode of action and benefits of using this type of materials has been extensively reviewed.

- *Interference with the transmission process*: Mineral oils are hydrophobic substances that interfere with virus acquisition and retention by aphids. Mineral oil of an appropriate viscosity and unsulfonated residues was found effective to reduce the efficiency of transmission by vectors. This measure is still popular for protection against nonpersistent viruses, particularly in nurseries. The mode of action seems to be by interference with virus binding by probing aphids. The leaf surface must be fully covered: full coverage demands frequent applications (up to twice a week) of large volume at high pressure. Combination of oil with pyrethroids (insecticides that have insect-repelling qualities) was tested successfully in Israel and in England (Raccah, 1986).

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