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Tospovirus

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Glossary

Ambisense genome Viral RNA genome with open reading frames in both the viral- and viral complementary (vc) sense on the same genome segment.
Envelope Membrane-like structure that packages genome segments.
IGR The intergenic region is the untranslated, A-U rich region found between the two open reading frames on the S and M RNA segments.
Negative sense genome Viral RNA genome that codes for proteins in the vc sense. Transcription of vc mRNA is required for translation of viral proteins.
Nucleocapsid Viral RNA encapsidated in the nucleoprotein.
Protoplast Plant cell lacking its cell wall.
RNP Ribonucleoprotein complex consisting of the viral RNA genome segment, nucleoprotein, and a small number of polymerase molecules.
Virion Quasispherical structure containing the viral genome and bounded by a membrane-like envelope.

History

Diseases now known to be caused by tomato spotted wilt virus (TSWV) were first reported in 1915 and were shown to be of viral etiology by 1930. This taxon of plant viruses was categorized as a monotypic virus group consisting of a single virus (TSWV) until the report of impatiens necrotic spot virus (INSV) in 1991. Thus, most of the characteristics which define the genus *Tospovirus* were obtained through investigation of TSWV even after the discovery of additional viruses in the genus (Table 1). Biological investigations beginning in the 1940s revealed a virus that had an unusually large host range and occurred in nature as a complex mixture of phenotypic isolates. However, it was one of the least stable viruses and most difficult plant viruses to mechanically transmit. Although the enveloped virions were observed in the 1960s, molecular characterization and elucidation of the genome organization were not completed until the early 1990s. The virus was shown to be vectored by thrips in the 1930s and later transmitted in a persistent manner. Thrips were demonstrated to be a host for replication of the virus and that replication was required for transmission in the early 1990s. Later it was recognized that limited, localized replication may occur in thrips that does not result in the thrips becoming viruliferous. Advances in gene function and cellular biology have been limited due to the absence of a robust *in vitro* plant or thrips cell culture system, and lack of an efficient reverse genetics system. However, limited progress has been made utilizing gene expression systems and classical viral genetics.

Taxonomy and Classification

Tospoviruses constitute the only genus of plant-infecting viruses in the family *Bunyaviridae*; however, these viruses share many molecular characteristics typical of other members of this virus family. They have an enveloped virion containing the viral genome which is distributed among three RNA segments that replicate in a manner consistent with that of other negative strand viruses. All three segments have highly conserved, complementary

Table 1 List of *Tospovirus* species[a, b, c]

Tospovirus species	*Abbreviation*
Groundnut bud necrosis virus (*Peanut bud necrosis virus*)	GBNV
Groundnut ringspot virus	GRSV
Groundnut yellow spot virus (*Peanut yellow spot virus*)	GYSV
Impatiens necrotic spot virus	INSV
Tomato chlorotic spot virus	TCSV
Tomato spotted wilt virus	TSWV
Watermelon silver mottle virus	WSMoV
Zucchini lethal chlorosis virus	ZLCV
Tentative *Tospovirus* species	
Capsicum chlorosis virus (*Gloxinia tospovirus*) (*Thailand tomato tospovirus*)	CACV
Chrysanthemum stem necrosis virus	CSNV
Iris yellow spot virus	IYSV
Groundnut chlorotic fan-spot virus	GCFSV
Physalis severe mottle virus	PhySMV
Watermelon bud necrosis virus	WBNV

[a]http://www.ncbi.nlm.nih.gov/ICTVdb.
[b]Whitfield AE, Ullman DE, and German TL (2005) Tospovirus–thrips interactions. *Annual Review of Phytopathology* 43: 459–489.
[c]Synonyms are indicated inside parentheses.

termini resulting in a pan-handle structure and genes with functions similar to those of viruses in other genera are located in similar locations on the genome. However, the genome organization is distinct from the other genera. The small (S) and middle (M) segments each encode two genes in opposite or ambisense polarity.

Classification of a *Tospovirus* population as a distinct species (virus) is based upon the similarity of sequence between the nucleocapsid genes of the respective viruses. This is in contrast to the system used to differentiate viruses in other genera which traditionally relied on serological neutralization of infectivity or other biological properties (hemagluttination) mediated by the glycoproteins. Tospovirus isolates with greater than 90% nucleotide similarity in the nucleocapsid gene are classified as isolates of the same species (virus). Serologically related isolates with 80–90% sequence identity are subjectively classified as strains or as distinct species depending on other criteria. Isolates with less than 80% identity are classified as distinct species.

Geographic Distribution

TSWV, the type member of the tospoviruses is found worldwide in temperate regions in association with its thrips vector. The wide host-range of TSWV and its thrips vector is consistent with the geographic distribution. Other tospoviruses have more well-defined distribution. For example, GBNV, WBNV, and WSMoV, that are transmitted by *Thrips palmi*, a thrips species found only in the subtropics are only known to occur in Southeast Asia. Another anomaly is INSV. While INSV is reported to occur around the world, it is almost entirely limited to greenhouse-grown floral crops.

Host-Range and Virus Propagation

TSWV has one of the most diverse host-ranges of any plant-infecting virus. The virus infects over 925 plant species belonging to 70 botanical families, both monocots and dicots. In addition, TSWV infects approximately ten thrips species. Important economic plants susceptible to TSWV include tomato, potato, tobacco, peanut, pepper, lettuce, papaya, and chrysanthemum. Other tospoviruses (e.g., IYSV) have much narrower host-ranges and thus the broad host-range of TSWV is not characteristic of the genus. These viruses can be transmitted mechanically or by their thrips vector, but are not transmitted transovarially, by plant seeds or pollen. Purified RNA preparations are not infectious. There are no robust plant or insect culture systems for tospoviruses. However, plant and insect protoplasts have been successfully inoculated.

Virion Properties

Tospovirus virions are quasispherical, enveloped particles 80–120 nm in diameter (**Figure 1**). Two viral coded glycoproteins, G_N and G_C, are embedded in the viral envelope and form surface projections 5–10 nm long. Ribonucleoprotein (RNP) particles consisting of the viral RNA encapsidated in the nucleoprotein (nucleocapsid), and a small number of polymerase molecules are contained within the envelope. Nucleocapsids are pseudocircular due to noncovalent bonding of the complementary RNA termini. Intact virions as well as carefully prepared RNPs retrieved from sucrose or $CsSO_4$ gradients are infectious. There are several reports that TSWV and INSV isolates, while infectious, are defective for virion formation.

Genome Properties

Tospoviruses have a single-stranded, tripartite RNA genome with segments designated as L, M, and S in order of decreasing size (**Figure 2**). The termini of each of the RNA segments consist of an eight nucleotide sequence (5′ AGAGCAAU 3′) that is strictly conserved among all tospoviruses. The remaining untranslated region at the termini also has a high degree of complementarity. Base pairing at the termini between the inverted complementary sequences supports a pan-handle structure that most likely serves as a promoter for replication.

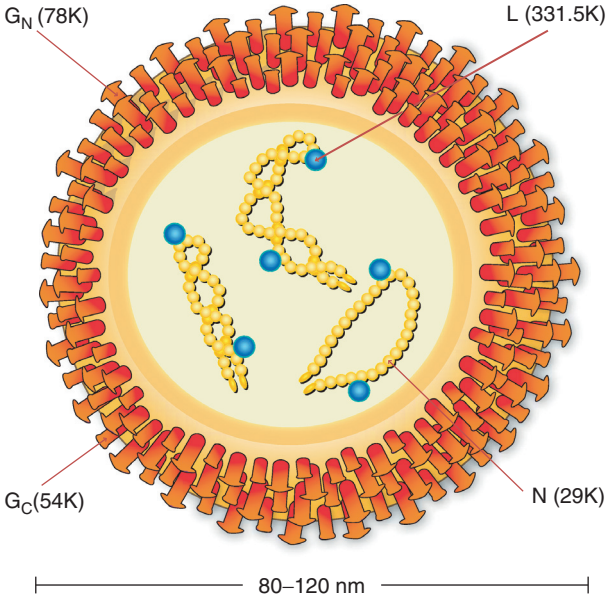

Figure 1 Tospovirus quasispherical virion particles. The S, M, and L RNA genomic segments are encapsidated by the nucleoprotein, are in association with L protein molecules, and form pan-handle structures due to the complementarity of their 5′ and 3′ ends. The glycoproteins G_N and G_C are embedded within the viral envelope.

The L RNA is 8.9 kbp and codes for the L or RdRp protein in the viral complementary (vc) sense (Figure 2). The M and S RNAs are in ambisense orientation. The M RNA is 4.8 kbp and codes in the viral sense for the nonstructural protein NSm and for the G_N/G_C precursor glycoprotein in the vc sense. The S RNA is 2.9 kbp and codes in the viral sense for the nonstructural protein NSs and the nucleocapsid protein in the vc sense (Figure 2).

TSWV M and S RNA IGRs have variable lengths, are A–U rich, and are the most hypervariable regions of the genome. The 5′ and 3′ ends of the IGRs are conserved, separated by variable sequences, deletions, and insertions. In addition, highly conserved sequences are embedded within the S RNA IGR. A 33 nucleotide (nt) duplication occurring in the S RNA IGR of some isolates has been correlated with loss of competitiveness in mixed infections of isolates with and without the duplication. A 31nt conserved sequence, with significantly higher GC-content compared to the remaining S RNA IGR, has also been found in some TSWV isolates. The IGRs of the M and S segments have high inclination for base pairing thought to be involved in initiation and termination of transcription. There is speculation that the termination of transcription is dictated by a conserved nucleotide sequence (CAACUUUGG) in the center of the S and M RNA IGR or that it is due to a secondary structure highly stabilized in the 31nt region referred to above.

Full length molecules of the M and S RNAs are found in infected tissue and purified virions in both the viral and vc sense (approximate ratio of 10:1), consistent with ambisense segments from other viruses. Defective interfering RNAs (DIs) associated with attenuated symptom expression and increased replication rate are also frequently observed. DIs from the L ORF in TSWV infected tissue are the result of and associated with attenuated infectivity. Deletions, frameshift, and nonsense mutations in the G_N/G_C ORF have been shown to interfere with thrips transmissibility and virion assembly. Recently, frameshift and nonsense mutations with unknown effect have also been identified in the N ORF. The formation of DIs is favored by repeated mechanical passage in certain plant hosts, high inoculum concentration, and low temperatures. Available evidence supports the hypothesis that secondary structure rather than sequence is the primary determinant of the site of deletion. There is also a high frequency of DIs that maintain the original reading frame resulting in translation of truncated proteins whose existence was confirmed in nucleocapsid preparations.

Protein Properties

The 331.5 kDa L protein encoded by the L RNA has been identified as the putative RdRp, through sequence homology with other members of the *Bunyaviridae* and identification of sequence motifs characteristic of polymerases. RdRp activity has been associated with detergent-disrupted TSWV virion preparations. The 33.6 kDa NSm protein encoded by the M RNA has been shown to induce tubule structures in plant protoplasts and insect (*Spodoptera* and *Trichoplusia*) cells. Induction of tubules in plants, ability to change the size exclusion limit of plasmodesmata, an early expression profile and complementation of cell-to-cell and systemic movement in a movement-defective tobacco mosaic virus vector is evidence that NSm is the TSWV movement protein and that it supports long-distance movement of viral RNAs. In thrips, NSm does not aggregate into tubules, indicating that this protein might not have any function in the vector's life cycle. It is also known that NSm specifically interacts with the N protein, the At-4/1 intra- and intercellular trafficking plant protein and binds single-stranded RNA in a sequence-nonspecific manner. An NSm homolog is absent in the animal infecting *Bunyaviridae*. The 127.4 kDa G_N/G_C precursor glycoprotein also coded by the M RNA contains a signal sequence that allows its translation on the endoplasmic reticulum. Proteolytic cleavage of the polyprotein does not require other viral proteins. The M_rs of G_N and G_C is 78 kDa and 54 kDa respectively. Evidence for the involvement of the glycoproteins in thrips transmission is provided by: (1) their interaction with proteins of the thrips vector, (2) their association with the insect midgut during acquisition, (3) the loss of thrips transmissibility of envelope-deficient mutants, (4) the presence of a

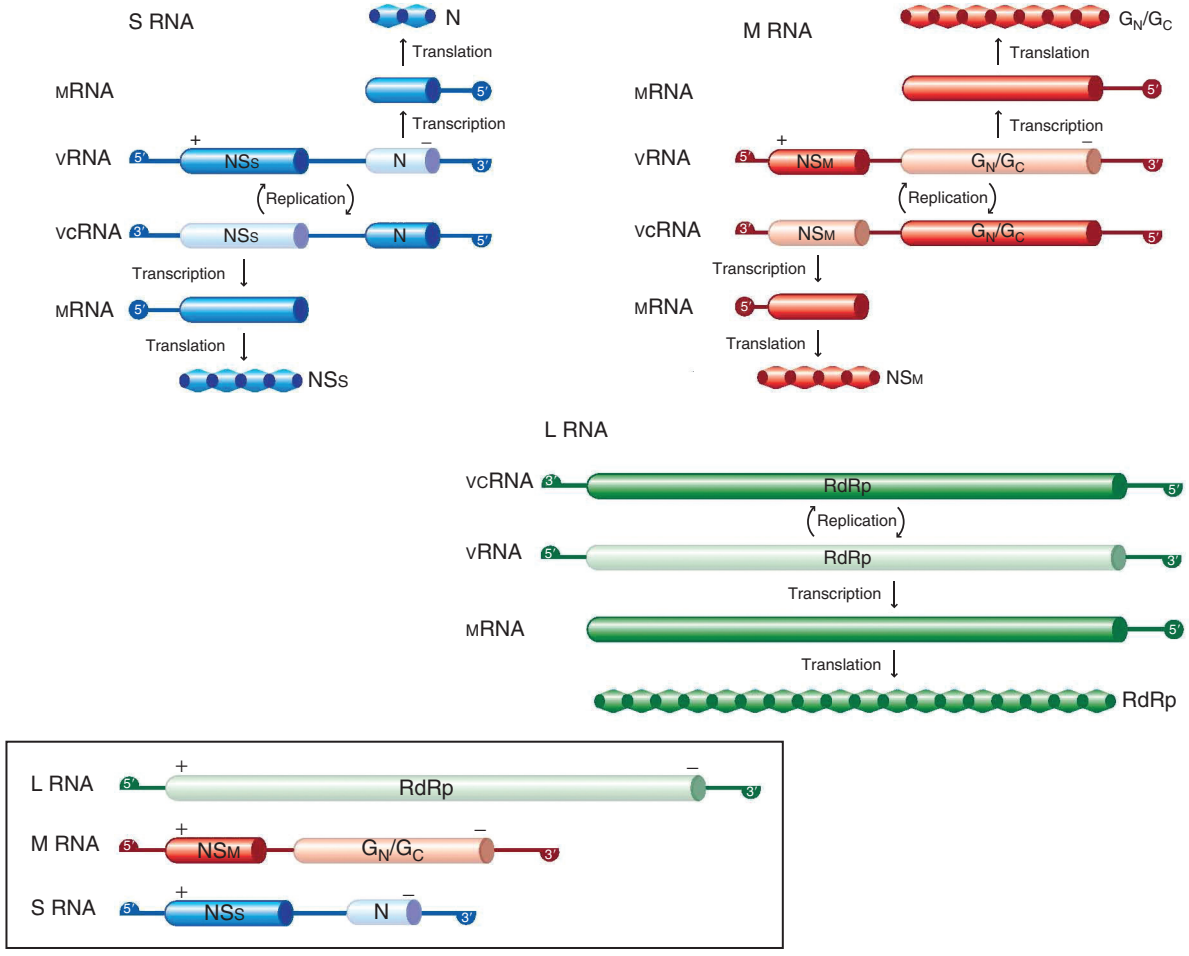

Figure 2 Tospovirus ambisense genome organization (inset in figure) and expression strategy. Positive (+) and negative (–) sense ORFs are dark and light shaded tubes respectively. Proteins from the S and M segments are translated from subgenomic mRNAs which are capped with 10–20 nt of non-viral orgain at the 5′ end.

glycoprotein sequence motif that is characteristic for cellular attachment domains and (5) the observation that only reassortants with the M RNA of a thrips-transmissible isolate rescue thrips transmissibility. Specifically, G_N is involved in virus binding and/or entry in thrips midgut cells, whereas G_C is a possible fusion protein playing a significant role in pH-dependent virus entry. It is also believed that these proteins are implicated in virion assembly. The NSs protein encoded on the S RNA is 52.4 kDa and accumulates to high levels as loose aggregates or paracrystalline arrays of filaments. NSs has RNA silencing suppressor activity, affects symptom expression in TSWV-infected plants, and is not present in the mature virus particle. The N protein, also encoded by the S RNA, ranges in size from 29 to 3 kDa depending on the virus. This protein encapsidates the viral RNA segments, is highly abundant, and is the predominant protein detected in serological assays. A 'head-to-tail' interaction of the nucleoprotein N terminus (aa 1–39) with the C terminus (aa 233–248) results in multimerization.

Replication

Replication of viral RNA and assembly of virions occurs in the cytoplasm of both plant and insect cells. Tospovirus replication, however, has mainly been described based on plant infection. Upon entry into the plant cell, the virus loses its membrane and releases infectious nucleocapsids into the cytoplasm. In thrips cells, infection by tospoviruses is accommodated by binding of the viral surface glycoproteins to a host cell receptor(s) (possibly a 50 kDa and/or a 94 kDa protein). This is followed by release of infectious nucleocapsids into the cytoplasm, through fusion between the viral and thrips membranes possibly initiated by low pH. Depending on the concentration of N protein, the viral RNA is either transcribed or replicated. At low N concentrations, the polymerase transcribes mRNAs that are translated into the virus proteins. Translation of proteins from the S and M ambisense RNAs occurs from subgenomic mRNAs (Figure 2). The S and M subgenomic mRNAs are capped

at the 5′ terminus with 10–20 nucleotides of nonviral origin indicating that tospoviruses utilize a cap-snatching mechanism to regulate transcription. Leader sequences of alfalfa mosaic virus have also been detected as caps of TSWV mRNA in mixed infections of the two viruses. The TSWV transcriptase has a reported preference for caps with multiple base complementarity with the viral template. Upon increase of N protein concentration, the polymerase switches its mode to replication with the viral RNA serving as the template. Replicated viral RNAs form RNPs that can presumably associate with the NSm protein for movement through plasmodesmata to adjacent plant cells through tubular structures. Alternatively, RNPs form new virions by associating with the glycoproteins and budding through the Golgi membranes. Virions are initially double-membraned, but soon coalesce and form groups of virions with a single membrane surrounded by another membrane.

Pathogenicity and Cytopathology

Tospoviruses are noted for the severity of the diseases they cause in plants. Symptoms are highly variable, depending on the virus, the virus isolate, the host plant, time of the year, and environment, and are thus of little diagnostic value. Chlorosis, necrosis, ring or line patterns, mottling, silvering, and stunting often appear on inoculated and systemically infected leaves. Systemic invasion of plants is frequently nonuniform. Stems and petioles may exhibit necrotic lesions. Observed symptoms often mimic disease and injury caused by other biotic and abiotic stresses. Infection of younger plants results in severe stunting and high mortality rates. TSWV has been shown to affect more severely *Datura*, *Nicotiana,* and *Physalis* plants under a specific temperature regime (daytime, $29 + 2\,^{\circ}C$, nighttime, $24 + 3\,^{\circ}C$). The effect of tospovirus infection on thrips has been controversial, due to the confounded effects of plant host, virus, and environment on the insect vector and the genetic variability of thrips and virus populations. TSWV infection of *F. occidentalis* provided evidence that thrips exhibit an immune response to the virus. Recent work with TSWV-infected *F. fusca* reared on infected foliage indicated a direct effect of the virus on thrips resulting in reduced fitness. The same study showed that the plant infection status and the TSWV isolate have also an effect on the insect, explaining the variable results obtained from independent studies of virus pathogenicity on the insect vector.

Tospoviruses induce characteristic cytopathic structures that are host and virus-isolate dependent. In addition to virions, inclusions of viroplasms consisting of the NSs or N protein may be abundant in the cytoplasm. NSs may aggregate in loose bundles (e.g., TSWV) or in highly ordered paracrystalline arrays (e.g., INSV). Excess N protein occurs in granular electron dense masses. NSs and nucleocapsid protein inclusions have been observed in infected plant and insect cells. NSm protein induces tubule structures in plant protoplasts and insect cells.

Transmission and Epidemiology

Tospoviruses are transmitted from plant to plant by at least ten thrips species in the genera *Frankliniella*, *Scirtothrips*, and *Thrips*. Among the more common vectors are *Frankliniella occidentalis*, *F. fusca*, *F. schultzei*, *F. intonsa*, *F. bispinosa*, *Thrips palmi*, *T. setosus*, and *T. tabaci*. Thrips feed on the cytoplasm of plant cells. The contents of infected cells are ingested and the virus is transported along the lumen of the digestive tract to the midgut, the primary binding and entry site into the insect cells. The brush border of the midgut lumen is the first membrane barrier that the virus encounters. The virus replicates in the midgut and crosses the basement membrane into the visceral muscle cells. The virus subsequently enters the primary salivary glands. It has been hypothesized that the virus moves from the midgut to the salivary glands through infection of ligament-like structures, or when there is direct contact between membranes of the visceral muscles and the primary salivary glands during the larval stages of development. A less plausible hypothesis is that the virus infects the salivary glands after entry and circulation in the hemocoel. Viral inoculum is introduced into plants in the insect saliva coincident with feeding on the plant by adult thrips.

The process of successful acquisition occurs only by larvae and acquisition rates decrease as larvae develop, affecting adult vector competency. Vector competency is also determined by the thrips' feeding preference on a particular host, the uniformity of distribution of virus in plant cells, the rate of virus replication in the midgut, and the extent of virus migration from the midgut to the visceral muscle cells and the salivary glands. In some instances the virus can be acquired by adult thrips and infects midgut cells, but is unable to spread further possibly due to the formation of an age-dependent midgut barrier (e.g., basal lamina). Research has shown the existence of thrips transmitters with detectable levels of virus, nontransmitters with detectable virus, and nontransmitters with no detectable virus, supporting multiple sites for vector specificity between tospoviruses and thrips. Evidence for replication of the virus in the insect vector is based on the accumulation of NSs and the visualization of viral inclusions in midgut epithelial cells, muscle cells, and the salivary glands. Although the virus is maintained transtadially throughout the life of the insect, there is no evidence for transovarial transmission. Thus, each generation of thrips must acquire the virus during the larval stages.

The primary dispersal of tospoviruses is by adult thrips and dissemination of infected somatic tissue in vegetatively propagated crops. These viruses are thought to move long distances in thrips carried by wind currents. They may also survive in commercial agricultural systems in weeds that serve as a bridge between crops. Infected summer weeds (e.g., in NC *I. purpurea*, *I. hederacea*, *M. verticillata*, *A. palmeri*, *C. obtusifolia*, *R. scabra*, *Ambrosia artemisiifolia* L., *Polygonum pensylvanicum* L., *and Chenopodium album* L.) are the principal source for spread of TSWV to winter annual weeds, from which the virus is spread to susceptible crops in spring. Secondary spread within a crop can only occur in crops that concomittantly support virus infection and reproduction of the vector as only the larval stage can acquire the virus for transmission. Transmission through plant seed and pollen has not been conclusively demonstrated. The emergence of these viruses as serious pathogens in crops has been attributed to the increased prevalence of *F. occidentalis* as an agricultural pest on a worldwide basis.

Genetics and Evolution

The knowledge base for genetics and evolution of tospoviruses has been derived almost exclusively from TSWV. TSWV has a characteristic ability to adapt to new or resistant hosts and to lose phenotypic characters following repeated passage in experimental hosts, especially *Nicotiana benthamiana*. The virus occurs in plants as a heterogeneous mutant population with one or two predominant haplotypes and 9–21 rare haplotypes. Recent research shows that natural TSWV variants evolve in nature through recombination, random genetic drift, and mutation. Intergenomic recombination is important for the genesis and evolution of ancestral TSWV lineages. Genetic drift during thrips transmission and mutation concurrent with virus population growth, shape the genetic architecture of the most recently evolved lineages. The existence of single viral strains as mutant populations and recombination in ancestral viral lineages arm TSWV with a unique genetic reservoir for causing disease and spreading in epidemic proportions in nature. Additional research at the species level supports a distinct TSWV geographical structure and the occurrence of species-wide population expansions. TSWV is also known to use reassortment of genome segments to adapt to resistant hosts under specific laboratory conditions. The determinants of adaptation to resistance in tomato and pepper have been mapped to the M and S RNAs respectively. Little is known about the thrips–tospovirus coevolution and the genetic diversity of the thrips vector itself. The altered status of *Thrips tabaci* as a TSWV vector is one of the very few likely examples of coevolution between tospoviruses and their insect vector.

Detection and Diagnosis

Tospoviruses have certain unique biological properties that are useful for diagnosis. These viruses can be mechanically transmitted by gently rubbing inoculum on plants dusted with carborundum. *Nicotiana glutinosa* L., *Chenopodium quinoa* Wild., and garden petunia give characteristic lesions that progress as spots or concentric zones, and sometimes as lethal necrosis. Tospoviruses can also be identified by electron microscopy of leaf-dip preparations on thin sections of infected plants. Additional techniques for identification are based on the enzyme-linked immunosorbent assay (ELISA) using polyclonal and monoclonal antibodies, and detection of viral-specific nucleic acids using ribo- and cDNA-probes. The reverse transcription-polymerase chain reaction (RT-PCR) is the most powerful and commonly used technique for detecting small amounts of tospovirus RNA. Real time RT-PCR has been successfully used to detect and quantify TSWV in leaf soak and total RNA extracts from infected plants and thrips. RT-PCR with degenerate primers can detect five distinct tospovirus species. Tissue selection and sampling strategy are critical factors in TSWV diagnosis and detection regardless of the technique. Because, TSWV titer varies throughout the plant and does not spread uniformly throughout plants that are 'systemic' hosts, sampling strategies should be validated in each situation.

Prevention and Control

Tospoviruses cause significant economic losses annually, due to suppressed growth, yield, and reduced quality. These viruses can be partially managed in well-defined cropping systems such as glasshouses by obtaining uninfected plant propagules, implementing a preventative thrips control program in high risk areas, together with constant monitoring of production areas for thrips and infected plants. However, these strategies are costly and require intensive management. Control in field crops is problematic due to the array of external sources of inoculum. Vector control is generally ineffective against the introduction of virus from external sources, due to thrips' high fecundity, ability to develop insecticide resistance, and to infest many TSWV-susceptible crops. Some measure of control can be achieved using thrips-proof mesh tunnels in the field and reflective mulches. Cultural practices such as utilization of virus-tested planting stock, careful selection of planting dates, removal of cull piles and weeds, rotation with nonsusceptible crops, prevention of planting TSWV-susceptible crops adjacent to each other, reduced in-field cultivation to avoid movement of thrips from infected sources, can reduce the spread of tospoviruses. In peanuts, higher plant density, planting from early until late May and application of selected

insecticides have reduced the incidence of TSWV. In flue-cured tobacco, early-season treatment with activators of plant defenses and insecticides have also significantly reduced TSWV incidence.

Deployment of resistant cultivars has provided benefits in only three of the crops infected by TSWV. Although little is known about the benefits of host resistance against most of the tospoviruses, TSWV defeated nearly every resistance gene deployed against it in many crops. Single-gene resistance is available for TSWV in a limited number of tomato (*Sw-5*) and pepper (*tsw*) cultivars. Naturally occurring, resistance-breaking isolates of TSWV have been recovered from pepper and tomato cultivars containing their respective resistance genes. A co-dominant cleaved amplified polymorphic sequence (CAPS) marker has been developed for TSWV marker-assisted selection in pepper. 'Field' resistance has been reported for some peanut varieties. Progress has been made in understanding the genetic basis of the ability of TSWV to overcome single gene resistance by mapping determinants to specific segments of the TSWV genome and characterizing the selection process. Pathogen-derived resistance utilizing the N and NSm genes has been effective in some greenhouse and field tests; however, isolates have been obtained that overcome nucleocapsid mediated resistance. Best suppression of TSWV epidemics has been achieved with the integrated use of moderately resistant cultivars, chemical, and cultural practices. The impact of these viruses on agricultural production is in large part due to the absence of durable forms of resistance in the affected crops or other highly effective control measures.

Future Perspectives

The last decade has been characterized by exciting developments in our understanding of tospovirus molecular biology, evolution, and virus–host relationships. Further progress in understanding replication and gene function requires the development of efficient reverse genetics, plant and insect culture systems for tospoviruses. In addition, effective management of these viruses will depend on a deeper understanding of thrips' genetic diversity, virus–thrips coevolution and the changes in viral and thrips population dynamics upon exertion of specific selective forces. Such understanding can be acquired only through integrated research at the interface of virology, entomology, and ecology.

See also: Tomato Spotted Wilt Virus.

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Totiviruses

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Glossary

Mycoviruses Viruses that infect and multiply in fungi.

Pseudoknot A secondary structure in viral mRNA that slows movement of the ribosome and may cause a frameshift that allows entry to an alternative reading frame during translation.