IDHAYA COLLEGE FOR WOMEN-KUMBAKONAM

DEPARTMENT OF MICROBIOLOGY

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HISTORY AND NOMENCLATURE OF PLANT VIRUS

INTRODUCTION

The discovery of plant viruses causing disease is often accredited to A. Mayer (1886) working in the Netherlands demonstrated that the sap of mosaic obtained from tobacco leaves developed mosaic symptom when injected in healthy plants. However the infection of the sap was destroyed when it was boiled. He thought that the causal agent was the bacteria. However, after larger inoculation with a large number of bacteria, he failed to develop a mosaic symptom.

HISTORY

In 1898, Martinus Beijerinck, who was a Professor of Microbiology at the Technical University the Netherlands, put forth his concepts that viruses were small and determined that the "mosaic disease" remained infectious when passed through a Chamberland filter-candle .This was in contrast to bacteria microorganisms, which were retained by the filter. Beijerinck referred to the infectious filtrate as a "contagium vivum fluidum", thus the coinage of the modern term "virus".

After the initial discovery of the 'viral concept' there was need to classify any other known viral diseases based on the mode of transmission even though microscopic observation proved fruitless. In 1939 Holmes published a classification list of 129 plant viruses. This was expanded and in 1999 there were 977 officially recognized, and some provisional, plant virus species.

The purification (crystallization) of TMV was first performed by Wendell Stanley, who published his findings in 1935, although he did not determine that the

RNA was the infectious material. However, he received the Nobel Prize in Chemistry in 1946. In the 1950s a discovery by two labs simultaneously proved that the purified RNA of the TMV was infectious which reinforced the argument. The RNA carries genetic information to code for the production of new infectious particles.

More recently virus research has been focused on understanding the genetics and molecular biology of plant virus genomes, with a particular interest in determining how the virus can replicate, move and infect plants. Understanding the virus genetics and protein functions has been used to explore the potential for commercial use by biotechnology companies. In particular, viral-derived sequences have been used to provide an understanding of novel forms of resistance. The recent boom in technology allowing humans to manipulate plant viruses may provide new strategies for production of value-added proteins in plants.

NOMENCLATURE AND CLASSIFICATION OF PLANT VIRUSES

- Traditionally, viruses are named after the most conspicuous symptom they produce on the first host. A virus causing mosaic on tobacco is called Tobacco mosaic virus, whereas the disease itself is called as tobacco mosaic.
- There have been frequent changes in the nomenclature and classification of viruses and generic names have been adopted.
- A genus is usually considered as a population of virus species that shares common characteristics and are different from other population of species.
- Currently 70 genera of plant viruses have been recognized.
- The genera are named either after the type species (Caulimovirus after Cauliflower mosaic virus) or are given a descriptive name often from a Greek or Italian word for a major feature of a genus, e.g., Closterovirus from

the Greek word ‘kloster’ meaning ‘a spindle or thread’ – descriptive of virus particle shape; Geminivirus from the Latin word ‘geminous’ meaning twins to describe the particles.

- Secondly, genera are grouped together into family on common characteristics.
- There are 14 families recognized for plant viruses, such as Reoviridae and Rhabdoviridae, which are common with animal viruses. However, 22 genera have not yet been assigned any family and are called ‘floating genera’.
- The family is either named after type member genus (e.g., Caulimoviridae named after Genus Caulimovirus) or given a descript to be named associated with genus for a major feature of family, e.g., Geminiviridae descriptive of virus particles.
- Only three orders have been accepted so far by International Committee for Taxonomy of Viruses (ICTV). The mononegavirales contains, among others the Rhabdoviridae in which there are two plant virus-families

USE OF VIRUS NAMES

The ICTV sets rules which are regularly revised on virus nomenclature and the orthography of taxonomic names.

- The last word of the species is ‘virus’; and suffix word for a genus is ‘virus’, for a subfamily is ‘virinae’, for a family is ‘viridae’, for an order is ‘virales’.
- In formal taxonomic usage, the virus order, family, genus and species names are printed in italics or underlined with first letter being capitalized.
- Other words in species names are not capitalized unless they are proper nouns or parts of proper noun.

- Also in formal usage, names of taxons should proceed the name being used e.g. Family Caulimoviridae, the Genus Closterovirus, the species Potato virus Y.
- However, in less formal instances which are widely used, the taxonomic unit is omitted.
- The plant viruses are classified on the basis of structure, physico-chemical properties, serological relationships, activities in the host plants and transmission.

LATEST CLASSIFICATION

The plant viruses are classified in five major groups based on:

– Nature of the genome (RNA or DNA)
– Strandedness (single or double stranded)
– Method of replication

- Each group (not a recognized taxon) has orders, families, genera and species.
- The five groups are:
    i) Single stranded positive sense RNA [(+) RNA] viruses
    ii) Single stranded negative sense RNA [ss (-) RNA] viruses
    iii) Double stranded RNA (ds RNA) viruses
    iv) Double stranded DNA virus [ds DNA (RT)] viruses
    v) Single stranded DNA [ss DNA] viruses

I. Single stranded positive sense RNA [(+) RNA] viruses:

- Order: Nidovirales

i) Family: Bromoviridae, e.g., Bromovirus (Brome mosaic virus-BMV), Alfamovirus (Alfalfa mosaic virus-AMV), Cucumovirus (Cucumber mosaic virus-CMV) and Ilarvirus (Tobacco streak virus-TSV).
ii) Family: Closteroviridae, e.g., Closterovirus (Beet yellows virus-BYV), Ampelovirus (Grapevine leaf roll associated virus GLRaV).
iii) Family: Comoviridae, e.g., Comovirus (Cowpea mosaic virus), Fabavirus (Broad bean wilt virus), Nepovirus (Nematode transmitted polyhedral virus, like Tobacco ring spot virus).
iv) Family: Flexiviridae, e.g., Potexvirus (Potato virus X), Carlavirus (Carnation latent virus).
v) Family: Luteoviridae, e.g., Luteovirus (Barley yellow dwarf virus-BYDV) and Polerovirus (Potato leaf roll virus- PLRV)
vi) Family: Potyviridae,

This family is largest single group of plant viruses and has been studied more extensively. Members of genus Potyvirus are one of the most successful plant viral pathogens. e.g., Potyvirus (Potato virus Y-PVY), Ipomovirus (Sweet potato mild mottle virus-SPMMV) and Bymovirus (Barley yellow mosaic virus).

vii) Family: Sequiviridae, e.g., Sequivirus (Parsnip yellow fleck virus-PYFV) and Waikavirus (Rice tungro spherical virus- RTSV).
viii) Family: Tombusviridae, e.g., Tombusvirus (Tomato bushy stunt virus-TBSV), Carmovirus (Carnation mottle virus), Necrovirus (Tobacco necrosis virus-TNV)
ix) Family: Tymoviridae, e.g., Tymovirus (Turnip yellow mosaic virus- TYMV).

- Some of the very important viruses like Tobamovirus (Tobacco mosaic virus- TMV), Tobravirus (Tobacco rattle virus-TRV), Potexvirus (Potato virus X-PVX) etc. have not been assigned any family yet.
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II. Single stranded negative sense RNA [ss (-) RNA] viruses: Members of this group are only enveloped plant viruses.

Order: Mononegavirales

- Family: Rhabdoviridae, e.g., Cytorhabdovirus (Lettuce necrotic yellows virus- LNYV) and Nucleorhabdovirus (Potato yellow dwarf virus- PYDV).
- Family: Bunyaviridae, e.g., Tospovirus (Tomato spotted wilt virus-TSWV; Groundnut bud necrosis virus- GBNV)

I**II. Double stranded RNA (ds RNA) viruses :** There is no order assigned.

- Family: Rheoviridae, e.g., Fijivirus (Fiji disease virus-FDV) and Phytorheovirus (Wound tomur virus- WTV).
- Family: Partiviridae, e.g., Alphacryptovirus (White clover crypto-virus 1) and Betacryptovirus (White clover crypto-virus 2)

IV. Double stranded DNA virus [ds DNA (RT) virus]: No order has been assigned.

- Family: Caulimoviridae, e.g., Caulimovirus (Cauliflower mosaic virus- CaMV)

V. Single stranded DNA [ss DNA] virus: No order has been assigned.

- Family: Geminiviridae, e.g., Mastrevirus (Maize streak virus- MSV), Curtovirus (Beet curly top virus-BCTV), Begomovirus (Bean golden mosaic virus- BGMV), Bhendi yellow vein mosaic virus- BYMV and Cassava latent virus- CLV.
- Family: Circoviridae, e.g., Nanovirus (Subterranean clover stunt virus; Banana bunchy top virus-BBTV).

TRANSMISSION, MULTIPLICATION, SYMPTOMS AND CONTROL OF PLANT VIRAL DISEASES

TRANSMISSION

1. Seed Transmission of Virus:

Transmission through the seeds of the host plant was earlier considered to play a minor part in the spread of virus diseases. Recently Bennett (1969) listed 53 viruses which are transmitted by seeds of about 124 plant species.

The seeds are important in the spread of a few viruses of legumes, wild cucumber, tomatoes, and curly top virus of beet sugar. In the latter case the seeds carry a high percentage of the virus. The virus, however, does not enter the embryo. It is carried in a portion of the seed of the diseased plants.

2. Transmission by Vegetative Propagation:

It is one of the chief methods of transmission of virus diseases especially of Potato, Rose, Sugarcane, Raspberry, Strawberry, Turnips, Bulb plants, fruit trees and many ornamentals.

The vegetative parts, the infected plants such as the tubers, bulbs, roots, offshoots, buds and scions which are used for propagation, will contain the virus present in the parent. The new plants raised by the above-mentioned vegetative methods are nearly always infected.

3. Transmission by Mechanical Means:

(i) By contact of infected and healthy leaves brought about by wind.

(ii) By rubbing the juice of the diseased plants over the surface of the leaves of healthy plants.

(iii) By grafting infected buds on to healthy plants.

(iv) Agricultural implements also play quite an important part. The knife used for cutting the seed pieces and the pruning shears will spread the disease.

(v) Some viruses spread below ground by contact between the roots of diseased and healthy plants.

(vi) Handling plants at planting time and in cultural operation will also help in the spread of viruses such as Sugar beet. Curly top virus and Cucumber mosaic virus.

4. Transmission by Cuscuta:

In many cases Dodder (Cuscuta) serves as a transmitting agent and an effective bridge between the infected host and the healthy plants by establishing intimate biological contact through its haustoria.

5. Soil Transmission:

Quite a number of viruses are transmitted through the soil. Common examples of soil borne viruses are Potato mosaic virus, Oat mosaic, Wheat mosaic, etc. In all these cases the disease is contracted from the soil.

6. Insect Transmission:

Some plant and animal viruses are spread and complete particles introduced into host cells by arthropod vectors and even by dog-bite as in rabies. Among the arthropods most important agents of spread of virus diseases are the insects.

The insect which carries the disease is called a vector. The insect vectors which play a major role in the dissemination of plant viruses are the Aphids, Leafhoppers, Flee beetles, Scale insects, thirps and White flies.

Most of the insect vectors are sucking insects. Aphids transmit more plant viruses than any other insects. Leafhoppers come next in the list. About three hundred plant virus diseases are known to have insert vectors.

7. Transmission by Fungi:

The first proof of the fungus as a vector of plant viruses was found by Gorgon in 1958. Fie found that the diseased lettuce was invariably infected by a soil chytrid, Olpidium. Later he discovered that the fungus acts as a reservoir and vector of the big vein virus.

The virus acquired by the fungus remains in the oospore. The latter germinates and produces the zoospores which function as infective agents and penetrate lettuce roots.

8. Some Soil Inhabiting Viruses have Nematode Vectors:

Animal viruses may gain access to the higher animals through the mouth and nose from dust or contaminated food. Besides infection from outside, virus may also be transmitted from cell to cell but the internal transmission need not be in the form of virus particles.

MULTIPLICATION

Viruses multiply only in living cells. The host cell must provide the energy and synthetic machinery and the low molecular-weight precursors for the synthesis of viral proteins and nucleic acids

The virus replication occurs in seven stages, namely;

1. Adsorption,
2. Entry,
3. Uncoating,
4. Transcription / mRNA production,
5. Synthesis of virus components,
6. Virion assembly and
7. Release (Liberation Stage).

Adsorption

It is the first step of viral replication. The virus attaches to the cell membrane of the host cell. It then injects its DNA or RNA into the host to initiate infection. In animal cells these viruses get into the cell through the process

of endocytosis which works through fusing of the virus and fusing of the viral envelope with the cell membrane of the animal cell and in plant cell it enters through the process of pinocytosis which works on pinching of the viruses.

Entry

The cell membrane of the host cell invaginates the virus particle, enclosing it in a pinocytotic vacuole. This protects the cell from antibodies like in the case of the HIV virus.

Uncoating[

Cell enzymes (from lysosomes) strip off the virus protein coat. This releases or renders accessible the virus nucleic acid or genome**.**

Transcription / mRNA production

For some RNA viruses, the infecting RNA produces messenger RNA (mRNA). This is translation of the genome into protein products. For others with negative stranded RNA and DNA, viruses are produced by transcription then translation.

The mRNA is used to instruct the host cell to make virus components. The virus takes advantage of the existing cell structures to replicate itself.

Synthesis of virus components

The following components are manufactured by the virus through the host's existing organelles:

- Viral protein synthesis: virus mRNA is translated on cell ribosomes into two types of virus protein.
- Structural: the proteins which make up the virus particle are manufactured and assembled.

- Non – structural: not found in particle, mainly enzymes for virus genome replication.
- Viral nucleic acid synthesis (genome replication) new virus genome is synthesized, templates are either the parental genome or with single stranded nucleic acid genomes, newly formed complementary strands. By a virus called polymerate or replicate in some DNA viruses by a cell enzyme. This is done in rapidly dividing cells.

Virion assembly

A virion is simply an active or intact virus particle. In this stage, newly synthesized genome (nucleic acid), and proteins are assembled to form new virus particles.

This may take place in the cell's nucleus, cytoplasm, or at plasma membrane for most developed viruses.

Release (liberation stage)

The viruses, now being mature are released by either sudden rupture of the cell, or gradual extrusion (budding) of enveloped viruses through the cell membrane.

The new viruses may invade or attack other cells, or remain dormant in the cell. In the case of bacterial viruses, the release of progeny virions takes place by lysis of the infected bacterium. However, in the case of animal viruses, release usually occurs without cell lysis.

SYMPTOMS AND CONTROL OF PLANT VIRAL DISEASES

A **symptom** of plant disease is a visible effect of disease on the plant. Symptoms may include a detectable change in color, shape or function of the plant as it responds to the pathogen. Leaf wilting is a typical symptom of verticilium wilt, caused by the fungal plant pathogens Verticillium albo-atrum and V. dahliae.

Here are a few examples of common signs and symptoms of fungal, bacterial and viral plant diseases:

Fungal disease signs:

- Leaf rust (common leaf rust in corn)
- Stem rust (wheat stem rust)
- Sclerotinia (white mold)
- Powdery mildew

Fungal disease symptoms:

- Birds-eye spot on berries (anthracnose)
- Damping off of seedlings (phytophthora)
- Leaf spot (septoria brown spot)
- Chlorosis (yellowing of leaves)

Bacterial disease signs (difficult to observe, but can include):

- Bacterial ooze
- Water-soaked lesions
- Bacterial streaming in water from a cut stem

Bacterial disease symptoms:

- Leaf spot with yellow halo
- Fruit spot
- Canker
- Crown gall
- Sheperd's crook stem ends on woody plants

Viral disease symptoms:

- Mosaic leaf pattern
- Crinkled leaves
- Yellowed leaves
- Plant stunting

CONTROL OF PLANT VIRAL DISEASES

Plant Disease Control

It is very important to remember that a correct diagnosis is the most important step in the eventual control of a plant disease. Most diseases have a fairly well established control protocol. Most often, failure to control the disease happens because the problem was misdiagnosed in the first place.

This is a list of the most important general strategies for management of plant diseases:

- Crop Resistance - should be first line of defense whenever possible
- Cultural Methods
- Physical Methods
- Pesticides
- Regulation

These methods will be discussed further with examples.

Cultural methods for disease control refer to those growing methods that reduce pathogen levels or reduce the rate of disease development. These include:

- Sanitation
- Crop Rotation
- Host Eradication
- Improvement of Crop Environment

DNA CONTAINING-VIRUS - CAULIFLOWER MOSAIC VIRUS

- **Cauliflower mosaic virus** (**CaMV**) is a member of the genus Caulimovirus, one of the six genera in the family Caulimoviridae, which are pararetroviruses that infect plants.

- Cauliflower mosaic virus (CaMV) is the type species of the family Caulimoviridae.
- CaMV infects mostly plants of the family Brassicaceae (such as cauliflower and turnip) but some CaMV strains (D4 and W260) are also able to infect Solanaceae species
- Induces a variety of systemic symptoms such as mosaic, necrotic lesions on leaf surfaces, stunted growth, and deformation of the overall plant structure.
- The symptoms exhibited vary depending on the viral strain, host ecotype, and environmental conditions.
- CaMV is transmitted in a non-circulatory manner by aphid species such as Myzus persicae.
- Once introduced within a plant host cell, virions migrate to the nuclear envelope of the plant cell.

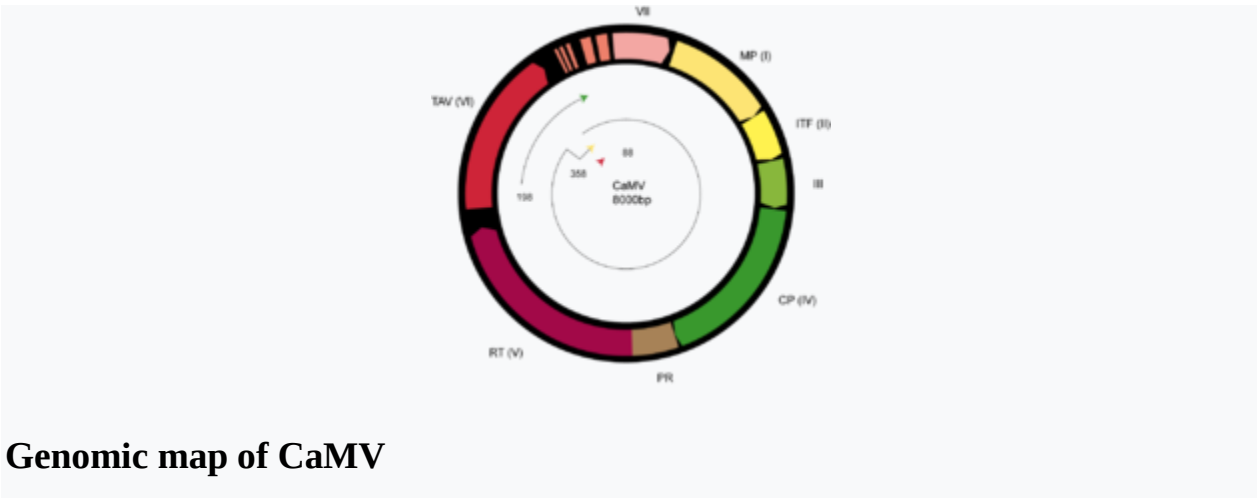
STRUCTURE

- CaMV contains a circular double-stranded DNA molecule of about 8.0 kilobases, interrupted by nicks that result from the actions of RNAse H during reverse transcription.
- These nicks come from the Met-tRNA, and two RNA primers used in reverse transcription.
- After entering the host cell, these single stranded "nicks" in the viral DNA are repaired, forming a supercoiled molecule that binds to histones.

- This DNA is transcribed into a full length, Terminally redundant, 35S RNA and a subgenomic 19S RNA.

GENOME

- The promoter of the 35S RNA is a very strong constitutive promoter responsible for the transcription of the whole CaMV genome.
- It is well known for its use in plant transformation.
- It causes high levels of gene expression in dicot plants.
- The differences in behavior are probably due to differences in quality and/or quantity of regulatory factors.
- Recent study has indicated that the CaMV 35S promoter is also functional in some animal cells, although the promoter elements used are different from those in plants.
- While this promoter had low activity compared to canonical animal promoters, levels of reporter products were significant.
- The promoter was named CaMV 35S promoter ("35S promoter") because the coefficient of sedimentation of the viral transcript, whose expression is naturally driven by this promoter, is 35S
- The 35S RNA is particularly complex, containing a highly structured 600 nucleotide long leader sequence with six to eight short open reading frames (ORFs).



Genomic map of CaMV

- ORF1 – Movement Protein (P03545)
- ORF2 – Aphid/Insect Transmission Factor (P03548)
- ORF3 – Virion-associated protein (VAP, P03551). Structural protein, DNA-binding capbilities
- ORF4 – Capsid Protein (CP, P03542)
- ORF5 – pro-pol (P03554): Protease, bifunctional Reverse Transcriptase and RNaseH
- ORF6 – Transactivator/viroplasmin (P03559): Inclusion Body Formation/Trafficking; Possibly more functions (See Below)
- ORF7/8 – Unknown (Appears to not be required for infection, Q83163, Q83164)
    - P6 has been shown to interact with a number of other CaMV proteins, such as P2 and P3
    - Investigating interactions between the two may help to elucidate the as yet unknown function of P7.
    - Another function of P6 involves modification of host NON-EXPRESSOR OF PATHOGENESIS RELATED 1 (NPR1) during the

course of infection. NPR1 is an important regulator of salicylic acid (SA) and jasmonic acid (JA)-dependent signaling, and is most closely associated with crosstalk between the two.

- Modification of NPR1 serves to inhibit plant cells' defensive responses by preventing SA-dependent signaling; modified NPR1 can properly traffic to the nucleus and bind the PR-1 promoter, but is unable to initiate transcription.
- Because active NPR1 is required for accumulation of SA, this leads to a further depletion of SA. Whereas regulation of SA-dependent signaling by P6-modified NPR1 is localized to the nucleus, regulation of JA-dependent signaling is cytoplasmic in nature and involves the COI1 pathway.
- In contrast to that of SA, JA-dependent signaling is increased in the presence of modified NPR1.

CaMV replicates by reverse transcription:

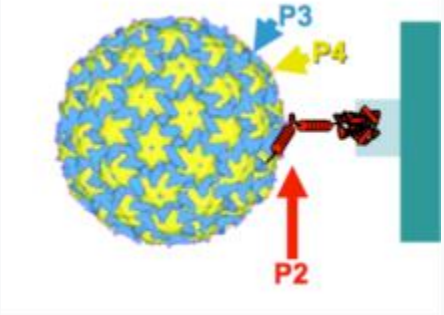
1. Viral particles enter a plant cell and are unencapsidated. At this stage the viral DNA consists of three fragments, one on the – strand (α) and two on the + strand (β and γ) which are imperfectly assembled into a circular genome with three gaps or discontinuities (D1, D2, and D3).
2. The viral DNA enters the nucleus where the discontinuities are filled in. At this point the viral DNA also associates with host histones, forming a minichromosome (not shown).
3. The host DNA-dependent RNA polymerase transcribes from the 35S promoter all the way around the viral genome, surpassing the 35S promoter.

(This creates two copies of the 35S promoter in the resulting RNA.) Transcription also initiates at the 19S promoter (not shown).

4. The viral RNAs pass into the host cytoplasm where they are transcribed.
5. The 3′ end of a $tRNA^{fMet}$ anneals to a site corresponding to discontinuity 1 (D1) near the 5′ end of the 35S RNA.
6. The $tRNA^{fMet}$ primes synthesis, by the viral reverse transcriptase (encoded by ORF V), of a new α strand.
7. RNase H removes the RNA from the DNA–RNA duplex, leaving behind the DNA.
8. This new DNA binds the 35S promoter at the 3′ end of the RNA template and synthesis of the α strand of DNA continues and RNase H continues to degrade RNA complexed to DNA.
9. Synthesis of the α strand completes. RNase H activity exposes purine-rich regions at the position of discontinuity 3 (D3), which primes the synthesis of the γ DNA strand.
10. RNase H activity exposes purine-rich regions at the position of discontinuity 2 (D2), which primes the synthesis of the β DNA strand. When the new γ strand of DNA reaches the 5′ end of the new α strand it switches to the 5′ end of the new α strand, recreating discontinuity 1 (D1). When the new γ strand of DNA reaches the 5′ end of the new β strand, it displaces the primer and some of the newly synthesized β strand, resulting in the recreation of discontinuity 2 (D2). When the new β strand of DNA reaches the 5′ end of the new γ strand, it displaces the primer and some of the newly synthesized γ strand, resulting in the recreation of discontinuity 3 (D3).

MOLECULAR MECHANISMS:

- The virus is acquired from an infected host during feeding by the aphid vector.
- To occur, a transmissible complex is composed of virions and protein P2 located in the vector's stylets.
- The P2 N-terminal domain recognizes a protein receptor located at the tip of the stylet and the P2 C-terminal domain binds to the P3-decorated virions.[15]



Transmissible complex of CaMV

- The mode of acquisition by the vector is controlled by the tissue and intracellular-specific localization of P2.
- This protein is only found in epidermis and parenchyma cells.
- viral protein P2 and P3 are first produced in numerous viral factories (electron-dense inclusion bodies), and are later exported and co-localize with microtubules, before concentrating in ELIB.
- CaMV specifically uses the microtubules to form the transmissible body and thus enable vector transmission.
- The complete molecular characterization and study of this virus was not carried further.

RNA CONTAINING VIRUS-TMV

Tobacco mosaic virus (TMV) was the first virus discovered. In 1889, Martinus Beijerinck, found that ‘tobacco mosaic disease’ was caused by a pathogen able to reproduce and multiply in the host cells of the plant. He called it ‘virus’ (from the Latin virus, meaning poison) to differentiate this form of disease from those caused by bacteria.

Tobacco yield losses

- TMV are currently estimated at only 1%, because resistant tobacco varieties are routinely grown.
- TMV can be a major problem because, unlike most other viruses, it does not die when the host plant dies and can withstand high temperatures.
- Thus, the virus can survive on implements, trellis wires, stakes, greenhouse benches, containers and contaminated clothing for many months.
- It can also survive in crop debris on the soil surface and infect a new crop planted on contaminated land.

- Tobacco products, particularly those containing air-cured tobacco, may carry TMV too.
- The virus cannot be transmitted in the smoke of burning tobacco, but smokers, especially those who roll their own cigarettes, could possibly carry the virus on their hands and transmit it to healthy plants.
- Sap-feeding insects such as aphids cannot transmit TMV. However, chewing insects such as grasshoppers and caterpillars do occasionally transmit the virus. They are not considered important vectors, however.

Transmission

- Tobacco mosaic virus is usually spread from plant to plant via 'mechanical' wounds caused by contaminated hands, clothing or tools such as pruning shears and hoes
- When plants are handled, the tiny leaf hairs and some outer cells are inevitably damaged and leak sap onto hands, tools and clothing.
- Seeds from infected plants can also carry the virus on their seed coats.
- The earlier the age at which the mother plant is infected, the more likely it is that the virus will contaminate the seed coat during seed harvesting.
- When the seed germinates, the virus may enter the seedling through small cuts caused by transplanting and handling, or during the germination/emergence process.
- Once inside the plant, the virus releases its genetic code (RNA). The plant mistakes this for its own RNA, and starts to produce viral proteins.

- The virus then spreads to neighbouring cells through microscopic channels in the cell walls (plasmodesmata), and eventually enters the translocation system of the plant (xylem and phloem). From here, it spreads to the entire plant.

Signs and symptoms

Symptoms first appear about 10 days after infection.

- The plants do not usually die, but growth can be seriously stunted. In the case of tomatoes, certain TMV strains can cause deformed fruit, non-uniform fruit colour and delay ripening.
- Specific symptoms depend on the host plant, age of the infected plant, environmental conditions, the virus strain and the genetic background of the host plant.
- However, common signs include mosaic-like patches (mottling) on the leaves, curling of leaves and the yellowing of plant tissues.

Managing the virus

No chemicals can cure a plant infected with a virus, and TMV is no exception. As mentioned before, however, resistant plant varieties are available.

TMV management involves using virus-free seedlings or plants and implementing strict hygiene procedures:

- Use new potting mix and new or thoroughly cleaned seedling trays when growing seedlings;

- If infected plants are discovered, either remove and destroy the plants and restrict access to the area, or always work in the affected area last and decontaminate yourself and your equipment afterwards;
- Remove all crop debris from the land, seedling production beds and benches in greenhouses;
- Place tools in a disinfectant solution for at least 10 minutes and rinse thoroughly with tap water;
- Disinfect door handles and other greenhouse structures that may have become contaminated by wiping thoroughly with recommended disinfectants;
- Thoroughly wash your hands with recommended disinfectants, such as carbolic soap, or a mixture of non-fat milk powder at 20% weight/vol, 10% bleach, and 70% ethanol, after handling tobacco products or TMV-infected plants. Make sure that the solutions are fresh, and replace regularly (it is recommended that the bleach solution be replaced every four hours).

POTY VIRUS

INTRODUCTION

Potyvirus is a genus of viruses in the family Potyviridae. Plants serve as natural hosts. There are currently 183 species in this genus including the type species Potato virus Y. The genus is named after the type virus (potato virus Y). Potyviruses account for ~30% of the currently known plant viruses. More than 200 species of aphids spread potyviruses and most are from the subfamily Aphidinae (genera Macrosiphum and Myzus).

VIRION

- The virion is non-enveloped with a flexuous and filamentous nucleocapsid, 680 to 900 nanometers (nm) long and is 11–20 nm in diameter.
- The nucleocapsid contains ~2000 copies of the capsid protein.
- The symmetry of the nucleocapsid is helical with a pitch of 3.4 nm.
- The genome is a linear positive sense ssRNA ranging in size from 9000–12000 bases/nucleotides. Most potyviruses have non-segmented genomes,
- In the species with a single genome, at the 5' end a protein is covalently linked (the Vg protein).
- It encodes a single open reading frame (ORF) expressed as a 350kDa polyprotein precursor.
- This is processed into seven smaller proteins: P1, helper component (HC), P3, cylindrical inclusion (CI), nuclear inclusion A (NIa), nuclear inclusion B (NIb), capsid protein (CP) and two small putative proteins known as 6K1 and 6K2.
- The P3 cistron also encodes a second protein
- Replication may occur in the cytoplasm, nuclei, chloroplasts, Golgi apparatus, cell vacuoles or more rarely in unusual sites.

- Potyviruses make proteinacous inclusions in infected plant cells.
- These may be crystals in either the cytoplasm or in the nucleus, as amorphous X-bodies, membranous bodies, viroplasms or pinwheels.
- The inclusions may or may not (depending on the species) contain virions.
- These inclusions can be seen in the light microscope in leaf strips of infected plant tissue stained with Orange-Green (protein stain) but not Azure A (nucleic acid stain).
- There are four different kinds of Potyvirus inclusions.
- Replication follows the positive stranded RNA virus replication model. Positive-stranded RNA virus transcription is the method of transcription.
- Translation takes place by -1 ribosomal frameshifting.
- The virus exits the host cell by tubule-guided viral movement.
- Plants serve as the natural host.
- The virus is transmitted via a vector (insects). Transmission routes are vector and mechanical.

TOMATO SPOTTED WILT VIRUS (TSWV)

INTRODUCTION

Tomato spotted wilt virus (TSWV) causes serious diseases of many economically important plants representing 35 plant families, including dicots and

monocots. This wide host range of ornamentals, vegetables, and field crops is unique among plant-infecting viruses.

Causal Agent

- TSWV is the only member of an RNA-containing virus group that has membrane-bound spherical particles 70-9Omm in diameter.
- Tomato spotted wilt, first described in Australia in 1919, was later identified as a virus disease.
- It is now common in temperate, subtropical, and tropical regions around the world.
- In recent years, TSWV has caused heavy crop losses in a wide variety of greenhouse-grown vegetable and ornamental plants across the United States and Canada.
- A lettuce-type strain is more commonly recovered from vegetables; an impatiens strain more readily infects ornamentals.
- There have been reports of TSWV infection in 174 plant species to date
- The virus is present only in the seed coat and not in the embryo.
- Seed transmission is thus not considered important for disease spread.
- Weed hosts serve as important virus reservoirs for TSWV and have been identified in Louisiana and Hawaii.

Vectors

- Tomato spotted wilt is one of only a few viruses transmitted by thrips and is by far the most important.
- Nine species are reported as vectors: Frankliniella occidentalis (western flower thrips); F schultzei, F fusca (tobacco thrips); Thrips tabaci (onion thrips); T setosus, T moultoni; F tenuicornis, Lithr~ps dorsalis, and Scirtothrips dorsalis.
- The first four are considered the most important vectors because of their wide distribution and the overlapping host ranges of these species and TSWV.
- The western flower thrips is the chief TSWV vector in greenhouse settings around the world, as well as in Hawaii's vegetable growing region.
- T tabaci is widely distributed in tropical, warm, and cool temperate areas around the world.
- Until recently, thrips problems in greenhouses were usually due to Frankliniella tritici (not a vector of TSWV), known simply as "flower thrips" or eastern flower thrips

Symptom Expression :

- Symptoms for tomato spotted wilt virus infection are ringspots (yellow or brown rings) or other line patterns, black streaks on petioles or stems, necrotic leaf spots, or tip dieback.

Control Spread of TSWV

- The primary greenhouse vector is the western flower thrips Resistance to specific organophosphates, carbamates, and synthetic pyrethroid insecticides is known in certain populations.
- Yellow sticky cards provide an easy way to detect the onset of an infestation
- These should be placed just above the crop canopy, at about one/1000 sq. ft., as well as near doors and vents to monitor the movement of thrips from the outside.
- Blue sticky cards catch more thrips, but since other insect pests (e.g., aphids, whiteflies, leafminers, fungus gnats) are not attracted to blue, yellow cards are preferred for general pest monitoring.
- The number of thrips/card should be recorded and graphed weekly to monitor population levels and aid in control decisions.
- Flowers can be checked for thrips by tapping a blossom over a sheet of paper, but it is more efficient to use sticky cards for detection and monitoring.
- Yellow or white flowers seem particularly attractive to thrips.
- The chief control using exclusion and sanitation.
- Inspect incoming plant material for TSWV symptoms and thrips infestations.
- Remove symptomatic plants from the greenhouse premises as soon as they are detected.
- Monitor the greenhouse continually for **thrips** infestations and treat accordingly.
- Eliminate weeds that may harbor the virus below benches and outside the greenhouse.

- In the field, immediately rogue any plants showing disease symptoms and check for thrips.
- Insecticide sprays may be necessary.

POTATO LEAFROLL VIRUS (PLRV)

INTRODUCTION

Potato leafroll virus (PLRV) causes a disease of potatoes worldwide and occurs in Western Australia.High levels of infection within a crop reduce returns as the virus greatly reduces tuber yield, size and marketability.

Infection

- Initial infection of potato crops by PLRV occurs when plants become infected by virus-carrying aphids during the growing season (primary infection).
- Infection also occurs when seed stocks containing infected tubers are planted, and infected potato plants grow from them (secondary infection).
- Aphids then spread the infection further.
- All tubers produced from an infected plant will carry the virus, so when they sprout the plant is already infected and can act as a virus source to neighbouring plants.

Symptoms

- Symptoms of primary infection are visible in the young leaves with upward rolling of the leaf margins.
- This occurs mainly in the part of the leaf near the base.

- The affected leaves are slightly pale and may show purpling or reddening.
- The leaves are often crunchy when touched.
- Symptoms of secondary infection are visible on all leaves, with upward rolling of lower leaves, while young leaves are often upright and pale.
- Lower leaves are stiff and crunchy when touched and often have purpled or reddened undersides (Figure 1). Infected plants are usually stunted
- Visible symptoms vary with the age at which the plant becomes infected, the variety, environmental conditions and crop vigour.
- Foliage symptoms are subtle or not expressed in infected plants.
- This is a serious issue as it can lead to virus infection being missed in visual inspections of seed potato or ware crops.
- This can then lead to crops having unacceptably high levels of PLRV.
- Potato leafroll virus, use only certified disease free seed tubers.
- Control volunteer potatoes and pluck out any plants that appear to be infected.
- The most popular potato varieties do not have any resistance to potato leafroll virus, but there are other cultivars that do not develop the necrosis on the actual tubers.
- Treatment for potato leafroll virus involves using chemical controls to eradicate aphids and reduce the spread of the disease.
- Apply insecticide from early to midseason.

RICE TUNGRO DISEASE (RTD)

INTRODUCTION

Rice tungro disease is caused by the combination of two viruses, which are transmitted by leafhoppers. It causes leaf discoloration, stunted growth, reduced tiller numbers and sterile or partly filled grains. Tungro infects cultivated rice, some wild rice relatives and other grassy weeds commonly found in rice paddies.

PATHOGEN

- Two morphologically unrelated viruses present in phloem cells.
- Rice tungro bacilliform virus (RTBV) bacilliform capsid, circular ds DNA genome and Rice tungro spherical virus (RTSV) isometric capsid ss RNA genome.

DISEASE CYCLE

- Transmission mainly by the leaf hopper vector Nephotettix virescens Males, females and nymphs of the insect can transmit the disease.
- Both the particles are transmitted semi-persistently, in the vector the particles are noncirculative and nonpropagative.
- Plants infected with RTSV alone may be symptomless or exhibit only mild stunting.
- RTBV enhances the symptoms caused by RTSV.
- RTSV can be acquired from the infected plant independently of RTBV, but acquisition of RTBV is dependent on RTSV which acts as a helper virus.

- Both the viruses thrive in rice and several weed hosts which serve as source of inoculum for the next.
- Ratoon from infected rice stubble serve as reservoirs of the virus.
- Disease incidence depends on rice cultivars, time of planting, time of infection and presence of vectors and favorable weather conditions

Management

- Field sanitation, removal of weed hosts of the virus and vectors.
- Grow disease tolerant cultivars like Pankhari203, BM66, BM68, Latisail, Ambemohar102, Kamod253, IR50 and Co45.
- Control the vectors in the nursery by application of Carbofuran 170 g/cent 10 days after sowing to control hoppers.
- Spray Phosphomidan 500 ml or Monocrotophos 1lit/ha (2 ml/litre) or Neem oil 3% or NSKE 5% to control the vector in the main field 15 and 30 days after transplanting.
- Set up light traps to monitor the vector population.

SUGARCANE MOSAIC VIRUS

INTRODUCTION

Sugarcane Mosaic Virus (SCMV) is a plant pathogenic virus of the family Potyviridae. The virus was first noticed in Puerto Rico in 1916 and spread rapidly throughout the southern United States in the early 1920s. SCMV is of great concern because of the high economic impact it has on sugarcane and maize.

Symptoms

The disease appears more prominently on the basal portion of the younger foliage as chlorotic or yellowish stripes alternate with normal green portion of the leaf.

- yellow stripes appear on the leaf sheath and stalks.
- Elongated necrotic lesions are produced on the stalks and stem splitting occurs.
- The necrotic lesions also develop on the internodes and the entire plant becomes stunted and chlorotic.

Pathogen

Sugarcane mosaic potyvirusis a flexous rod, 650-770nm long X 12-15nm with ss RNA genome.

Disease cycle

- The virus is mainly transmitted through infected canes used as seed.
- The virus also infects Zea maysand a number of other cereals
- which serve as potential sources of virus inoculum.
- The virus also spreads through viruliferous aphids viz., Melanaphis sacchari, Rhopalosiphum maidisin a non-persistant manner.
- The virus is also sap-transmissible.
- The incubation period varies from 7 to 20 days, depending upon the host variety and virus strain.
- The symptoms may be prominent or masked depending on the environmental conditions and variety.

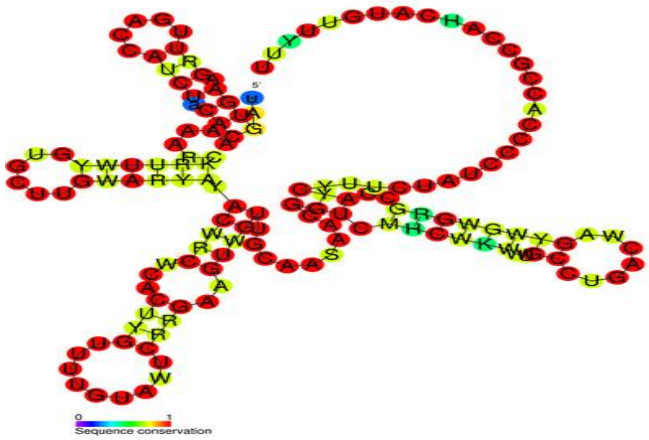
Management

- Roguing of infected plants and use of disease free planting material.

- Chemical sprays to manage the insect vector population in early crop stage.
- G row mosaic-resistant or, at least, tolerant varieties.
- Breeding mosaic-resistant varieties is needed.
- Saccharum spontaneumL. and S. barberi (Jesweit) carry resistance to mosaic and so varieties with this background must be preferred.
- Rogue out the diseased clumps periodically. Select setts from the healthy fields as the virus is sett-borne Aerated Steam Therapy (AST) at 56˚C for 3 hrs, for setts before planting is advised.

SUBVIRAL AGENTS

- Subviral agents are composed of three kinds: satellite viruses, viroids, and prions
- These transmissible agents are classified as subviral agents as they are less than a virus in some respects.
- Nonetheless, the viroid RNA is transmissible and causes pathogenic lesions in plants.



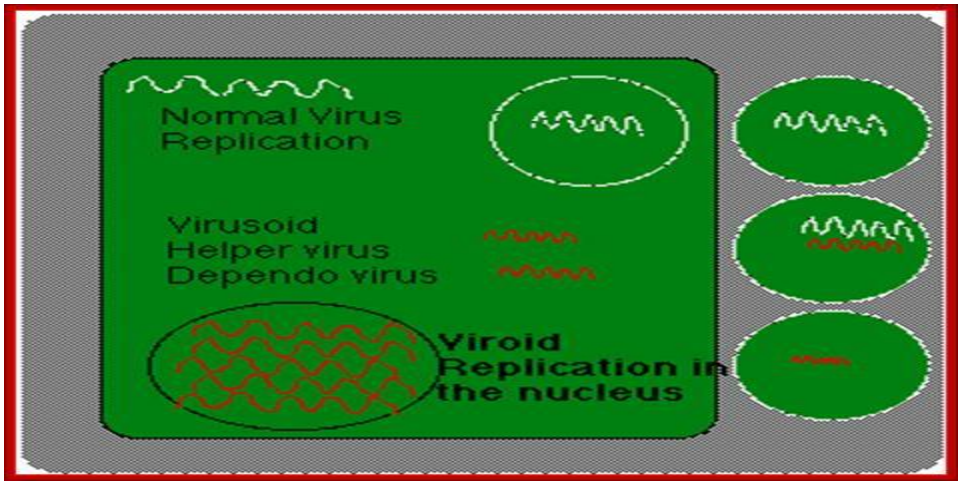
VIRUSOIDS

- Virusoids belong to a larger group of infectious agents called satellite RNAs, which are similar pathogenic RNAs found in animals.
- The plant virusoids, satellite RNAs may encode for proteins; however, like plant virusoids, satellite RNAs must coinfect with a helper virus to replicate.
- A second type of pathogenic RNA that can infect commercially important agricultural crops are the **virusoids**, which are subviral particles best described as non–self-replicating ssRNAs.
- RNA replication of **virusoids** is similar to that of viroids but, unlike viroids, virusoids require that the cell also be infected with a specific "helper" virus.
- There are currently only five described types of virusoids and their associated **helper viruses**.
- The helper viruses are all from the family of **Sobemoviruses**.
- An example of a helper virus is the subterranean clover mottle virus, which has an associated virusoid packaged inside the viral capsid.
- Once the helper virus enters the host cell, the virusoids are released and can be found free in plant cell cytoplasm, where they possess ribozyme activity.
- The helper virus undergoes typical viral replication independent of the activity of the virusoid.
- The virusoid genomes are small, only 220 to 388 nucleotides long.
- A virusoid genome does not code for any proteins, but instead serves only to replicate virusoid RNA. Virusoids are circular single-stranded RNA(s) dependent on viruses for replication and encapsidation.
- The genome of virusoids consist of several hundred (200–400) nucleotides and does not code for any proteins.

- ❖ Virusoids are essentially viroids that have been encapsulated by a helper virus coat protein.

NAMES OF FEW VIRUSOIDS:

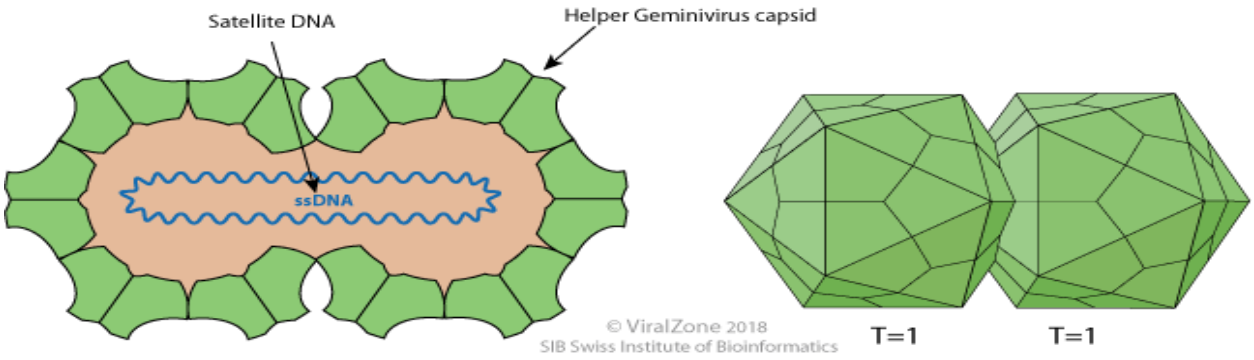
- Barley yellow dwarf virus: Helper virus-luteo virus.
- Tobacco ring spot virus: Helper virus-nepovirus.
- Subterranean clover mottle virus: Helper- sobemo virus.



SATELLITE VIRUSES

- ❖ Satellite viruses are particles that contain nucleic acid genomes encoding a structural protein that encapsidates the satellite genome.
- ❖ Satellite RNAs do not encode capsid protein, but are packaged by a protein encoded in the helper virus genome.
- ❖ Satellites are subviral agents that differ from viroids because they depend on the presence of a helper virus for their propagation.

- Satellite viruses are particles that contain nucleic acid genomes encoding a structural protein that encapsidates the satellite genome.
- Satellite RNAs do not encode capsid protein, but are packaged by a protein encoded in the helper virus genome.
- Satellite genomes may be single-stranded RNA or DNA or circular RNA, and are replicated by enzymes provided by the helper virus.
- The origin of satellites remains obscure, but they are not derived from the helper virus.
- Satellite viruses may infect plants, animals, or bacteria.
- An example of a satellite virus is satellite tobacco necrosis virus, which encodes a capsid protein that forms an icosahedral capsid that packages only the 1,260 nucleotide satellite RNA.
- The helper virus, tobacco necrosis virus, encodes an RNA polymerase that replicates its genome and that of the satellite.
- Satellite RNAs do not encode a capsid protein and therefore require helper virus proteins for both genome encapsidation and replication.
- Satellite RNA genomes range in length from 220-1500 nucleotides, and have been placed into one of three classes.
- Class 1 satellite RNAs are 800-1500 nucleotide linear molecules with a single open reading frame encoding at least one non-structural protein.
- Class 2 satellite RNAs are linear, less than 700 nucleotides long and do not encode protein.
- Class 3 satellite RNAs are 350-400 nucleotide long circles without an open reading frame.



- In plants, satellites and satellite viruses may attenuate or exacerbate disease caused by the helper virus.
- Examples of disease include necrosis and systemic chlorosis, or reduced chlorophyll production leading to leaves that are pale, yellow, or yellow-white.
- The symptoms induced by satellite RNAs are thought to be a consequence of silencing of host genes.
- For example, the Y-satellite RNA of cucumber mosaic virus causes systemic chlorosis in tobacco.
- This syndrome is caused by production of a small RNA from the Y-satellite RNA that has homology to a gene needed for chlorophyll biosynthesis.
- Production of this small RNA leads to degradation of the corresponding mRNA, causing the bright yellow leaves.