Viruses: Pathogens of Plants
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OBJECTIVES:
1. Understand the process of how plant viruses cause disease
2. Understand the types of plant-virus interactions
3. Understand the different types and mechanisms of virus movement
4. Understand the physiological and cytological responses to virus infections
How do plant viruses initiate an infection?

Plant viruses lack an active mechanism for cell entry.

Host receptors are not known for plant viruses.

If not acquired from a parent plant through fertilization or propagation, a virus must enter via a wound in the plant cell wall and must be at the site during injury.
Eukaryotic Cell - Animal

Prokaryotic Cell
Eukaryotic Cell
- Plant
Plant cell walls are thick compared with the sizes of the viruses infecting them (>10 μm versus <1 μm),

Plus, outer surfaces of plants have layers of waxes and pectin

No virus has evolved a mechanism to directly penetrate the plant cell wall
What types of interactions can occur once a virus is introduced into a plant?
1. Plant is **immune**, no or limited viral replication occurs, no cell-to-cell movement.

2. Virus replicates and moves throughout the plant without obvious symptoms evident – **latent (tolerance)**

3. Virus replicates and causes symptoms in new leaves but with time and further plant growth symptoms disappear - **plant recovers**

4. Virus replicates but the plant limits the virus movement by hypersensitive response - cell death (may not always be noticed; **necrosis, local lesions**)

5. Virus replicates and moves throughout the plant with **obvious symptoms**

Which of these possible responses do you think is the most common?
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Latent infections or Tolerance

- In wild plants, virus infection does not correlate with the presence of symptoms.
- Many of them have a persistent lifestyle and do not encode any proteins to aid in their dissemination.
- It is thought that these viruses may have been associated with their wild hosts for a long period on the viral evolutionary timescale.

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Plant Recovery:

- Plant shows symptoms in leaves after infection but the symptoms become milder with each new leaf and within a few weeks there are no symptoms and no detectable virus in the new leaves. (first studied in early 1960’s)

- The explanation for this was a mystery for many years

- Now known to be due to active plant responses to infection by viruses: post-transcriptional gene silencing (PTGS)
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These necrotic lesions are the result of mechanical inoculation on the leaf with TMV

*Tobacco mosaic virus (TMV)* in *N. glutinosa*
Virus replicates in the first infected cells, and the host produces a **hypersensitive response** at the site of inoculation (resulting in clusters of dead cells = local lesion).

In some plant/virus combinations, this is the end of any symptom production, virus spread, and virus reproduction.

**Local Lesions:**

- Only occur with some plant/virus combinations
- Some viruses never produce local lesions in any host
- Viruses can produce local lesions in some hosts but not others
- Lesions vary in size, color, time to appearance
- Local lesions can be the only symptom or can be followed by systemic infection
Local lesions vary in size, shape and color

*Chenopodium quinoa* inoculated with *Carnation mottle virus*
*C. quinoa inoculated with Parsnip yellow fleck virus*
*Nicotiana tabacum ‘Xanthi’ inoculated with Cherry leaf roll virus*
*Phaseolus vulgaris ‘Pinto 111’ inoculated with Bean pod mottle virus*
*Cucumis sativus inoculated with Clover yellow vein virus*
Uses of Local Lesions:

1. Number of lesions can be an indicator of the number of virus particles in the inoculum (similar to a plaque assay)

2. Biological indicator for identification of a virus

3. Rapid indicator of infectivity

4. Used to select and create host resistance in cultivated crops

Necrotic local lesions extending along the veins. Caused by a strain of *Bean common mosaic virus* in primary leaves of *Phaseolus vulgaris* ‘Monroe’, 13 days after mechanical inoculation.
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Systemic Infection:

- Virus enters a plant cell,
- Replicates in the cell,
- Moves to neighboring cells,
- Moves into the phloem, then to sinks (root and shoot apices), and then spreads to neighboring cells
- Virus replication in apical cells may cause developmental abnormalities
- Cellular abnormalities give rise to foliar symptoms

• Symptoms develop in leaves days/weeks after infection
• Symptoms develop in leaves distant from site of inoculation
When virus infects a plants – there are two kinds of movement:

**Cell to Cell**

**Systemic**
Generalized Virus Life Cycle

Entry/Penetration*

Genome uncoating

Translation

Rep protein(s)

Replication

Replicating Form

Transcription

mRNA

Translation

CP(s) MP(s)

vRNA/vDNA

Encapsidation = Virus Particles

Spread to neighboring cells

Spread to distant cells via vascular system

Translation

Transcription

mp(s) CP(s)

Transmission
Cell to Cell:
Relative sizes of some plant viruses compared with the size of plasmodesma

CTV, 2μm X 10 nm
TMV, 300 nm X 18 nm
PVY, 750 nm X 11 nm
LNYV, 220 nm X 80 nm
TSWV, 80 nm
CWTV, 70 nm
CaMV, 50 nm
CPMV, 28 nm

Encapsidated plant viruses are too large to move through plasmodesmata
Cell to Cell:
At least 3 mechanisms of cell-cell movement

1. Mechanism used by Tobamovirus (TMV, *Tobacco mosaic virus*)

2. Mechanism used by Comoviruses, Nepoviruses, Caulimoviruses, Tospoviruses

3. Mechanism used by Begomoviruses

In all cases viruses produce 1 or more proteins that allows cell-to-cell movement (Movement protein - MP)
Mechanism 1. Tobamovirus

- TMV MP binds and elongates single-stranded nucleic acids (RNA, DNA).

- TMV MP increases plasmodesmatal size exclusion limit from 0.7-kDa to app. 20-kDa.
TMV movement protein

- In TMV infected plants, and in 30k transgenic plants, the MP was localized to the plasmodesmata.

- The MP is phosphorylated by a cell-wall associated protein kinase. Regulation of plasmodesmatal transport by the MP is dependent on phosphorylation.

- The MP associates with cytoskeletal structures and with cortical ER.

- The coat protein is not required for cell-to-cell movement of TMV
2. Mechanism used by Comoviruses CPMV (Cowpea mosaic virus)

- CPMV MP does not bind nucleic acids
- The viral CP is essential for cell-to-cell movement
A short MP tubular structure forms in the plasmodesmata pore allowing CPMV virions to move from cell-to-cell.
2. Mechanism used by Nepoviruses TRSV (*Tobacco ringspot virus*)

- Tubular structures containing virus-like particles in *Tomato ringspot virus* (ToRSV)-infected *Nicotiana clevelandii* plant.
- Tubular structures are composed of TRSV movement protein
- Tubular structures will form in the absence of TRSV coat protein

3. Mechanism used by Begomoviruses

- Nuclear shuttle protein (NSP) and MP are both required for cell-to-cell movement.
- Both NSP and MP bind single-stranded nucleic acids.
- Coat protein is not required.
- Cell-to-cell movement often occurs in local lesions

- Cell-to-cell movement can be followed by systemic invasion
1) When a virus is inoculated into a leaf epidermal cell, the virus must move cell-to-cell if the infection is to spread.

2) In order for the virus to spread systemically, it must invade the mesophyll cells below the epidermis and pass through bundle sheath, parenchyma, and companion cells before finally entering the sieve element.

3) Once the virus has gained access to the phloem, it may be transported to other leaves (sinks) both up and down where the process of cell-to-cell movement may begin anew.

- Viral long distance movement is from source to sink leaves
- The process is very fast once it reaches the phloem
Effects of Viruses on Plants
Viruses are submicroscopic

But when they replicate in cells, their replication can leave evidence (inclusions) that are visible in the light and electron microscope:

“Inclusions” “Inclusion bodies”

**Inclusion Body:**

“An abnormal structure in a cell nucleus or cytoplasm having characteristic staining properties and associated especially with certain viral infections”
Ex. Potyvirus inclusions

Arrows point to pinwheel inclusions (top) and laminar aggregates (below)
Viral Inclusion Bodies Composed of:

- Virus particles
- Plus/or other proteins of the viral genome
- Plus/or modified host cell constituents

What are Inclusion Bodies?

- Sites of viral replication and/or assembly ("virus factories")
- Sites of accumulation of virus particles
- Sites of accumulation of excess viral protein(s)

The function of the inclusion body varies among virus families and sometimes even genera in the same family
Visualization of Viral Inclusions

Electron micrograph  Light micrograph

X body: Inclusion of *Tobacco mosaic virus* (TMV)
Effects of viruses on plant physiology (or how plant viruses reduce yields)

Viruses....

- Decrease the rate of **photosynthesis**
  (reduced amounts of chlorophyll, chloroplast ribosomes, and ribulose bisphosphate carboxylase)

- Increase the rate of **respiration**

- Increase the **activity of some enzymes**, including polyphenoloxidase

- Alter the levels or ratios of various **plant hormones**
  (auxins, gibberellin, abscissic acid, and ethylene)
  Epinasty and leaf abscission, may be due to increased levels of ethylene production
Effects of viruses on plant physiology or how plant viruses reduce yields

- Cause a **diversion of host metabolic materials**

- Reduce the availability of **fixed carbon**, which directly reduces photosynthesis, reduces the translocation of fixed carbon in the phloem.

- Impair the **movement of photo-assimilates** from the leaves and increase **starch accumulation**
How do viruses cause these changes??

Many symptoms are due to Post-transcriptional Gene Silencing (PTGS) –

- An ancient cellular process that plays an important role in management of cellular processes (elimination of mRNA)
- PTGS protects the cell against viruses and other nucleic acid-based threats
- Viral nucleic acid or mRNA of viral genes interferes with the translation of some of a cell's messenger RNA (mRNA) sequences
- So some genes that should be turned off (by normal PTGS) are not or are not turned off at the right time, this results in many of the typical virus symptoms
Do all virus infections result in a negative outcome?
**Camillia yellow mottle virus**  
(*Varicosavirus, no family assigned*)

**Abutilon mosaic virus in Abutilon spp.**  
(*Begomovirus, (Geminiviridae)*
Other Examples:

♦ Ex. 1: *Prune dwarf virus* causes a disease of cherry in which cherries are borne at the ends of the branches. This exposure results in larger and better colored fruit.

♦ Ex. 2: *Citrus exocortis viroid* and *Citrus dwarfing viroid* (aka *Citrus viroid* III) have been used to dwarf citrus, thereby allowing a predictable degree of tree size control and the use of higher planting densities.

♦ Ex. 3: Plants systemically infected by a mild strain of a particular virus may show resistance to a related more severe form of the virus. This phenomenon is known as cross-protection – This is used as a virus management technique, but it also can occur in wild plant species.

♦ Ex. 4: Viruses can turn on plant defense systems (SAR) and make the plant more resistant to other pathogens and to insects.

♦ Ex. 5: Viruses can improve the ability of wild plants to compete and survive